ON THE NATURE OF STOPPING A VOLUNTARY ACTION

by

JAMES TIMOTHY McGARRY

B.Sc.(Hons), Liverpool John Moores University, 1988

M.Sc., Bradford University, 1989

M.P.E. The University of British Columbia, 1993

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Department of Human Kinetics

The University of British Columbia
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ABSTRACT

The stopping of an earlier intended action is best explained in a race between a go process and a stop process (Logan & Cowan, 1984). The finish line, to which each process races, has been likened to a point of no return, specifically one that marks the onset of a final ballistic (unstoppable) process. Of note is the typical relation of reduced go probabilities and faster go latencies at shorter signal onset asynchronies (SOAs). (The SOA is the time interval between presentation of the go signal and presentation of the stop signal.) We report, in some cases, sub-maximal surface electromyograms (EMGs) at onset when trying to stop a maximal speeded action. These data indicate reduced synaptic drive to reach the motor pools as a result of earlier stopping effects and, as such, hold important implications for a theory of control. First, we interpret these data to suggest that the point of no return is phantom. Sub-maximal EMGs indicate a point in the control stream beyond which some EMG will be later observed but, importantly, they fail to mark the onset of a final ballistic process if, once breached, the same process remains subject to further effects of stopping. The alternative interpretation, however, that of a final ballistic process that receives sub-maximal input which results in sub-maximal output (i.e., EMG onset) cannot be ruled out from these data. We used the Hoffmann (H) reflex to probe further the mechanism of control for stopping a voluntary action. The H-reflex, an involuntary reflex that is taken as an index of spinal control, is relevant to the control of stopping because it is typically facilitated a short time before EMG onset. In other words, it provides a window of control within which a final ballistic process would otherwise be expected to locate. Thus, we interpret the effects of stopping on the H-reflex before EMG onset as strong evidence against a final ballistic process. Second, while the race model can explain the relation between the go probabilities, the go latencies and the SOAs, it fails to explain the sub-maximal EMG onsets that describe that same action in some cases. We submit a mechanism of excitatory-inhibitory interaction at all times up to the motor pool to explain both sets of empirical data. The viability of this theory is demonstrated using computer analyses.

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1 On the nature of stopping a voluntary action

1.1 Preface

The simple tenets of "self-organization, self-maintenance, (and) or self-repair" (Yates, 1987, p. 2, parentheses ours) that subserves an organism throughout its life cycle gives rise to many regularities. The principles of control that might govern such regularities, however, have long posed a fundamental question for biological science, from the analysis of cellular function through to the analysis of social behaviours. The general focus of this thesis centres somewhere between these two analyses on the control of human actions. That is, how the central nervous system, which might be thought of as a sensory-motor gateway to and from the outside world, is controlled from higher centres in the human brain in order to support purposeful action.

We take the neuron as the fundamental unit through which motor control is administered. The basic function of a neuron, of which there are many different types, is to communicate to, or message with, other neurons. These messages take one of two basic forms - excitation or inhibition. Skeletal muscle is thus activated through the neural action of supra-spinal and spinal centres that relay excitatory and inhibitory commands to the motor neurons (i.e., those nerve cells that attach to skeletal muscle). In this way, skeletal muscle contracts (i.e., develops tension) or relaxes (i.e., releases tension) depending on whether the relevant motor neurons receive excitatory or inhibitory inputs. Because skeletal muscle is activated only by those motor neurons that project to it, via what Sherrington (1947) called a "final common path" (see Gallistel, 1980), it follows that the control of skeletal muscle is effected through the control of its respective motor neurons, each of which is constantly being bombarded with excitatory and or inhibitory signals from higher centres (c.f., Lashley, 1951).

Since Descartes (see Gallistel, 1980), the brain has been thought to enact its will through the slavery of skeletal muscle, as specified in the above view. The later observations of extremely localized cortical mappings to motor function reinforced this view and led to an analogy for motor control of a puppet on a string (Turvey, 1990). Here, the brain was likened to the puppeteer and the puppet to the various muscles. Other analogies such as a telephone operator at a switchboard, or a

pianist at the piano, were also used to help explain how a "motor program" (c.f., a musical score) might *a priori* specify control of the to-be-produced action.

There are two key problems with the above account, first identified in largely inaccessible form by Bernstein (1935) and later advanced by Turvey (1977). First, the problem of degrees of freedom questions how a high dimensional motor problem might be reduced to a low dimensional motor solution. In other words, how might the vast array of inter-connecting neurons be managed effectively so as to produce orderly actions. Second, the problem of peripheral indeterminacy arises from the non-linear force-length relation of muscle. In effect, this means that any pattern of motor discharge (c.f., a motor program) yields varying patterns of force output as a function of (amongst other things) the joint angle. In other words, a 1:1 relation between motor discharge and force generation does not exist. Instead, a given pattern of motor discharge can yield many force patterns and a given force pattern can be generated from many patterns of motor discharge. How patterned outputs might be generated from non-patterned inputs remains a problem for motor control theory to this day.

Einstein, opposing the burgeoning theory of quantum mechanics at the time, once remarked to the effect that God was not playing at dice. The later advances of quantum theory, where "... quantum entities obey probabilities, not hard and fast certainties" (White & Gribbin, 1993, p. 221), and its subsequent acceptance determined that Einstein was wrong in this regard. Relatedly, in the context of motor control, stochastic properties at the level of the neuron have been proposed to account for the observed variance in reaction latencies, much like "... dice-throwing going on in the brain" (Barinaga, 1996, p. 344). In this thesis, we extend, using computer analysis, this analogy of dice-throwing to the processes of excitation and inhibition in order to explain the observed effects of stopping on an earlier intended action at various times.

Inhibition, the opposite process of excitation, plays a key role in the control of human actions. It prevents the selection of unwanted actions and allows for the rapid termination of no longer wanted actions. Stopping of actions is typically investigated using a second (stop) signal that is presented, on random trials, at a short variable time after a first (go) signal. The probability of action initiation

(i.e., a response to the go signal) and the latency of that initiation provide the primary dependent variables in such studies. In many studies, each has been found to reduce as the time interval between presentation of the go signal and presentation of the stop signal shortens. To date, this pattern of data is best accounted for by a race between a go (excitation) process and a stop (inhibition) process in which each process samples independently from its own latency distribution (Logan & Cowan, 1984). The series of studies reported herein seeks to investigate further the mechanism of control through which stopping an earlier intended voluntary action is effected, from measures of action amplitude, namely kinematics and electro-physiology (i.e., EMG), as well as from measures of probability and latency of action initiation.

In sum, the aim of this thesis is; (a) to describe the effect of stopping on a voluntary action from measures of action amplitude (EMG), probability and latency, (b) to explain these effects by way of a theory of control based on central nervous system physiology, and (c) to formalise and demonstrate the viability of this theory of control for explaining the empirical data using the technique of computer (modeling) analysis.

1.2 Stop-signal inhibition, neurological (neural) inhibition and reactive inhibition

Logan (1994) identified three forms of stopping that he opted to label as (a) stop-signal inhibition, (b) neurological (neural) inhibition, and (c) reactive inhibition. Stop-signal inhibition, the focus for the series of studies that constitute this thesis, was identified as a process of voluntary control that begins on presentation of the stop signal and ends on execution of the stop process. Its effect is to withhold or to withdraw the earlier intended action. While stop-signal inhibition is necessarily of neural origin, it is nonetheless regarded as being distinct and different from the properties of neural inhibition (Logan, 1994). We intend to show that this is not the case and, instead, that the empirical data from stop-signal inhibition can be explained from properties of excitatory-inhibitory interaction.

Excitation is the antithesis of inhibition. Thus, an excitatory input and an inhibitory input might be modeled as a positive signal and as a negative signal respectively, each serving to counter the effect of the other. In the above view, an excitatory neuron generates an excitatory input (pulse) that acts to excite its target neuron(s). The like view holds for an inhibitory neuron also. In fact, while

the target neuron is predisposed to excite or inhibit, it might well receive pulses of excitation and or inhibition depending on its physical structure, or wiring. The target neuron discharges if the net sum of the signals that converge on it in real time attain threshold for that neuron. Excitation pulses serve to drive the internal state of the excitatory neuron to threshold, whereas inhibition pulses serve to drive the internal state of the excitatory neuron away from threshold.

Just as the discharge of a neuron is a net result of the pulses that act on that neuron, so the discharge of a neural ensemble is a net result of the actions of the neurons within that ensemble. (The processes of excitation and inhibition are presumed to constitute a predominant ensemble of excitatory and inhibitory neurons respectively.) Excitatory-inhibitory ensembles interact with opposing effects yielding two consequences. First, the likelihood of attaining threshold in each assembly is decreased. Second, the latency to reach threshold in each assembly is increased. Interestingly, this latter effect is not only predicted from competing excitatory-inhibitory ensembles, but also from competing excitatory-excitatory ensembles as a result of what might be called reactive inhibition (Logan, 1994). Reactive inhibition arises as a byproduct of a competing alternative process that must be quashed (i.e., inhibited) before the selected movement can be generated. Examples of reactive inhibition include; response conflict (or competition) (e.g., Eriksen & Eriksen, 1974), dual task interference, or psychological refractory period (PRP), effects (e.g., Logan & Burkell, 1986; Pashler, 1994) and negative priming effects (e.g., Posner, Nissen & Ogden, 1978; Tipper, 1985) amongst others (Logan, 1994).

The common linkage between the various types of stopping listed above is that each acts to suppress ongoing activity. Stop-signal inhibition differs from neural inhibition and reactive inhibition in the mechanism through which this is thought to be achieved. In stop-signal inhibition, the inhibition (stop) process proceeds independently of the excitation (go) process (see later). In neural inhibition and reactive inhibition this is not the case. Instead, the stop process and the go process proceed inter-dependently, as each process shares, or competes for, a common pathway. In neural inhibition, this is observed in the algebraic sum of excitatory and inhibitory inputs that converge on a cell at any instant. In reactive inhibition, it is observed in the "... activation of a

pathway by an input item [that] produces facilitation (benefit) in the speed of processing subsequent items that share the same activated pathway ... but [that] produces widespread inhibition (costs) in the processing of items that use different pathways ..." (Posner et al., 1978, p. 138, square parentheses ours). We will briefly examine these effects of benefit and cost further.

Various studies have shown from latencies of reaction that the activity of a pathway facilitates or impedes processing. Eriksen and Eriksen (1974) showed that when a target letter (e.g., H) was flanked with compatible letters (H or K), similar letters (N, W and Z), dissimilar letters (G, J and Q) or incompatible letters (S or C), presented on either side of the target, then the latency of reaction to the target letter increased as a function of its flanking letters (i.e., noise). (The target letter, H, was presented on each trial from a set of paired alternatives in which each pair - H and K, or S and C - required a left key press or a right key press, respectively.) Specifically, compatible noise showed least increase, similar noise and dissimilar noise showed intermediate increase and incompatible noise showed most increase in latency as compared to a control (i.e., target only). In these types of tasks, it seems that the processing of each signal is actively inhibited until the target signal is identified from its distractors. The least inhibition occurred when the distractors constituted the least competing alternative (i.e., like distractors) and the most inhibition occurred when the distractors constituted the most competing alternative (i.e., unlike distractors) to the target letter.

The facilitation and the impairment of a pathway might be thought of in terms of positive priming and negative priming. Posner et al. (1978) reported a positive effect and a negative effect of priming as a result of correct and incorrect information (or cue) that was presented ahead of the to-be-presented signal (i.e., that signal that required an action). The signal cue comprised one of three signals (a right arrow, a left arrow or a plus sign) each presented with equal probability. The probability at which the right arrow, the left arrow and the plus sign indicated correctly the location of the to-be-presented signal was set at 0.8, 0.8 and 0.5 respectively. The experiment yielded valid, invalid or neutral trials depending on whether the signal cue contained correct ($p = 0.8$), incorrect ($p = 1.0 - 0.8 = 0.2$) or no information ($p = 0.5$) respectively. The reaction latencies showed benefits on valid trials and costs on invalid trials as compared to performance on neutral (control) trials.

Thus, information of the to-be-presented signal biases (primes) the information processes in favour of the expected signal, so yielding a cost-benefit trade-off that is reflected in task performance. Subsequent work, following Tipper (1985) (see below), suggests that the facilitation and the inhibition of pathways aids in the selection of a response, perhaps through the use of "relevancy labels" that participate in future processing activities (Buckolz, van Damme & O'Donnell, in press).

Tipper (1985) reported negative priming effects as a result of competing signals. In Tipper's (1985, Experiment I) task, a prime signal consisted of two figures, a selected (red) figure superimposed on an ignored (green) figure, and a probe signal that also consisted of two figures prepared in the same way. The prime signal preceded the probe signal in each trial. The experimental design was such that (a) the ignored prime was not related to the selected prime, nor was the selected probe related to the selected prime, (b) the ignored prime was the same as the selected probe (ignored repetition) or it was not related to the selected probe (control) and (c) the selected figures in the ignored repetition and the control trials were the same. The latency of the verbal response to the probe signal increased from control when the probe signal was the same as the ignored prime signal. These data were interpreted to suggest negative priming (i.e., inhibition) as a possible mechanism of selection (Tipper, 1985). In short, these studies (Eriksen & Eriksen, 1974; Tipper, 1985; Buckolz et al., in press) each showed that the presence of a signal distractor necessitates processing and that this processing interferes with the processing of the target signal as indexed in the latency of reaction.

Interference of a different kind is found in dual task studies. Here, the latency of reaction to a second signal is increased as an inverse function of the signal onset asynchrony (SOA) (i.e., the time interval between presentation of the first signal and presentation of the second signal). The reason for the increased latency in reaction to the second signal as a result of a first signal, known as the "psychological refractory period" (PRP) effect, is typically attributed to a bottleneck that is located somewhere on a single channel for which both processes compete (Welford, 1952). The first process to reach the bottleneck proceeds in uninterrupted fashion, whereas the second process to reach the bottleneck must wait until the bottleneck is vacated by the first process. Thus, shorter SOAs result

in longer latencies in the second process, as the second process must wait longer for the first process to clear the bottleneck.

The stopping process is not subject to the same bottleneck effects. Logan and Burkell (1986) used three (group) tasks - a dual task, a stop task and a change task - in which they analysed the latency of reaction to each signal. The dual task required a two choice (index finger, middle finger) right key press to the first (go) signal and a single left key press to a second (GO) signal. The stop task required the same two choice right key press to the go signal and the withdrawal of that key press to a second (stop) signal. The change task, a hybrid of the dual task and the stop task, required the same two choice right key press to the go signal, and the withdrawal of that key press as well as a left key press to a second (change) signal. In each task, the first signal was a visual signal (letter) and the second signal was an acoustic signal (tone). Interestingly, the latency of reaction to the second signal (i.e., left key press) in the change task was slowed only when the first action (i.e., right key press) was initiated. These data were interpreted in favour of late selection (Logan & Burkell, 1986) as well as an interruptible bottleneck (Pashler, 1994) consistent with the latter author's interpretation of earlier dual task interference studies. Pashler's explanation, at least, requires that the stopping process is not subject to the typical effects of interference from a bottleneck mechanism. We limit our focus on inhibition to this type of stopping process (i.e., stop-signal inhibition) which we shall hereafter refer to in the generic context of stopping.

1.3 On stopping a ballistic motor action

In physics, the term ballistic applies when energy is imparted to an object at onset, as in a bat striking a ball. The subsequent flight path of the ball is ballistic in the sense that the trajectory of the ball is specified at contact subject thereafter only to physical law. In the literature on motor control, the term ballistic is likewise used to describe that process of control whose "... neurological control signal, the envelope of motor neuronal impulses, is usually shaped or programmed in advance and not interrupted or changed once it has begun" (Stark, 1982, p. 565).[1] In this sense, a ballistic process is a process of control that is sufficiently brief for the efferent (central) signals that cause the action to be unaffected by the afferent (peripheral) signals that result from the sensory consequences of that

same action. Thus, a ballistic (open loop) process is distinct from a non-ballistic (closed loop) process in that the former, as defined, proceeds in the absence of feedback, whereas the latter is guided by the presence of feedback of some kind.

The epoch of a ballistic process is often taken to be the latency of reaction. This latency, which is faster for an acoustic signal than it is for a visual signal, tends to be in the order of about 200 ms, although short feedback loops of about 80 ms, as evidenced in visual corrections in anticipated tracking tasks, have been reported consistently (e.g., Flowers 1977, Megaw, 1972. See also Dewhurst, 1967, for fast compensatory changes to added load). These data fit with the general view of multi-layered control loops that are mediated by spinal and supra-spinal centres. Examples of control loops include latency reactions (~ 200 ms), long latency loops (~ 80 ms), as well as poly-synaptic, oligo-synaptic and mono-synaptic loops (~ 30 ms). In our view, the presence of multi-layered control loops serves to weaken the distinction between a ballistic process and a non-ballistic process.

Ballistic modes and non-ballistic modes of control need not be exclusive. Woodworth (1899), for example, long ago showed that the kinematics of aiming tasks are often typified by a ballistic process that is followed by an on-line, visually mediated, non-ballistic (i.e., continuous) process. The ballistic process serves to drive the limb to the vicinity of the target, while the non-ballistic process serves to guide the limb to its final destination. These findings are generally well accepted (e.g., Meyer, Abrams, Kornblum, Wright & Smith, 1988; Khan, Franks & Goodman, 1998), though for an alternative viewpoint, see Plamondon (1995a, 1995b).

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[1] This quote should not be taken as representative of Stark's (1982) position who, in fact, favours the counter position that limb movements are invariably not ballistic on the reasoning that muscles deliver active contractile forces in real time. Interestingly, Stark (1982, p. 565) notes that "Parenthetically, many psychologists, using elaborate paradigms, have shown just how difficult it is for the human brain to shortcut such a motor neuronal envelope once it has begun its control program. It is, however, possible to do so by means of elaborative predictive or massed trials."

In psychology, a ballistic process is defined not in relation to the absence of afferent signals (i.e., feedback), as typified in a stereotypical control burst from onset (Woodworth, 1899), but rather as a stage of involuntary control in the efferent stream before it takes effect at the level of the effectors (i.e., muscle). In other words, the input to a ballistic process in the control stream is preserved in its entirety as output from that same ballistic process, which is then either passed along as input to the next process or, if the ballistic process constitutes a final ballistic process then its output is observed in motor discharge. Thus, the final ballistic process reflects that point in the efferent stream that precedes the onset of action beyond which the unfolding control process can no longer be stopped (or changed). Importantly, it is the shielding of the control process from the presence of stopping effects that make for a final ballistic process. In contrast, the absence of stopping effects, as is the case when the time interval from presentation of the stop signal to the onset of the impending action is too brief for the stopping process to take effect, speaks neither for nor against a final ballistic process, as defined.[2]

1.4 On the presence and absence of a final ballistic process

In a social context, the risk of a faux pas from a "slip of the tongue" that betrays one's thoughts provides an example of the need to, on occasion, quickly prevent an action before it begins. Should the speaker be late to recognize the inappropriate comment, or should the social conditions change such that the intended comment is no longer appropriate, then the unfolding action must be stopped or changed in mid-stream. How stopping might be so effected provides the focus for this thesis.

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[2] If g = go latency, b = latency of the final ballistic process, s = stop latency and SOA = signal onset asynchrony, then the following conditions hold on any trial that a stop signal is presented. If $\{ SOA + s < g - b\}$ then the action is stopped and if $\{SOA + s > g - b\}$ then the action is initiated. Only if $\{(g - b < SOA + s < g)$ can evidence for $(b > 0)$ or against $(b = 0)$ a ballistic process be inferred. In contrast, the condition $\{SOA + s > g\}$ speaks neither for nor against a ballistic process since the time available in which to stop is less than the stopping latency $(g - SOA < s)$.

In a sports context, the athlete must initiate a cricket stroke on the basis of ball flight information in order to strike the ball at some future time. This type of action, where the execution of an intentional action is coincident with some future event, is easily accomplished by the skillful athlete if the future event is certain. It is less easily accomplished if the future event is uncertain. For instance, if the ball should take an unexpected swerve sufficiently late in its flight path, then the athlete might well commit to the stroke only to miss contact with the ball. (See McLeod [1987] for empirical data that support this example.) While the new information is presented before the anticipated future event unfolds, it is nonetheless presented too late for the athlete to stop, or change, the previously intended action. This type of situation has been explained by recourse to a point of no return, since, it seems that the unfolding control sequence of events that precede the action can no longer be stopped once a certain point has been reached (Bartlett, 1958). The point of no return is an important distinction in a psychological theory of control in that it signifies that point at which the process of control is thereafter committed to the onset of action. In other words, it marks the onset of a final ballistic (involuntary) process that is no longer subject to stopping or change (Osman, Kornblum & Meyer, 1986, 1990).

Some human skilled actions are highly complex. For instance, consider the keyboard skills of an expert typist. This type of skill might be thought of as a relatively continuous stream of discrete actions (i.e., key presses). In short, the expert typist must couple a high degree of accuracy with a high degree of efficiency which, in the work force, invariably translates as throughput, or typing speed. For highly skilled typists, each discrete action is initiated, produced and overseen within a time interval in the order of 150 ms - 200 ms (Rabbitt, 1978). Task complexity is further increased for the expert pianist, who must vary the inter-stroke intervals and strike force on the keys in accord with the musical score. The degree to which these actions can be stopped (changed) voluntarily as the sequence unfolds provides valuable information as to the mechanisms of control that oversee these actions.

The expert typist is frequently unable to stop typing on command, but rather continues to produce the next letter or so in the to-be-run-off sequence (e.g., Logan, 1982). This uninterrupted

sequence of discrete actions should not be taken as evidence for a point of no return, as defined, for, as Logan (1994, p. 205) comments "little is known about the mechanisms underlying the stop process, except that they take time to finish and when they finish, they stop responses". The short uninterrupted sequence of key presses that follow a stop signal might then simply occupy the time interval that is necessary to effect stopping (i.e., stop latency) and need not speak further in favour of a final ballistic process for reasons already advanced (see Footnote 2).

Involuntary errors provide another rich source of information as to the control mechanisms that underlie complex motor skills. Interestingly, involuntary errors are not generated unwittingly, rather the occurrence of a just-committed error can be quickly detected, and corrected post hoc, with a high degree of accomplishment (Rabbitt, 1966). In a later study, Rabbitt (1978) analysed the ability of expert typists using a manual typewriter to self-detect and self-report errors committed on-line. The typists were instructed to type copy, as usual, from a given text but, on detection of an error, to immediately stop typing, mark the error with an asterisk, identify the correct (intended) key stroke and to then continue with the typescript. The typists were able to detect and report their errors within zero, one or two key strokes following their occurrence, in spite of vision of the key strokes and hands being masked throughout. Of particular note is that errors were consistently produced with less force than non-errors, as indicated from the density impression of the key head on the paper, regardless of the frequency of key strokes that followed the error. In Rabbitt's words, the typists "... sometimes become aware (of the impending error) before they complete the keystroke implementing it. They may then attempt to 'pull back' incorrect keystrokes, producing fainter impression copy" (1978, p. 945, parentheses ours).

Thus, not only might the error be detected on-line before it occurs, its production might also be changed on-line as seen in the difference in key-stroke force between errors and non-errors. These data speak for a process of control that is subject to change at all times otherwise force modulation in the commission of an error should not occur. Thus, a point of no return that marks a final ballistic (involuntary) process is non-existent if, once breached, the to-be-committed error is subject to further attenuation before its commission. We will further investigate this argument shortly.

1.5 Stop-signal inhibition: The stop signal experimental paradigm

There are two ways in which the effect of stopping a motor action at various stages of its generation have been investigated. First, the ability to stop an intended action scheduled for some future event in time (c.f., the cricket example) has been investigated by the presentation of a stop signal, on random trials, at various times before the known future event. Second, the ability to stop an earlier intended action has been investigated by the presentation of a stop signal, on random trials, at various times following the presentation of a go signal. Since both experimental procedures pertain to the series of studies reported herein, we will detail each protocol in turn.

The first procedure investigates the ability to inhibit an earlier intended action that is scheduled to occur coincident with some future event (or signal). The signal is continuous, as in for example the passage of a sweep hand over a target (c.f., Slater-Hammel, 1960), which allows for the future event to be anticipated. On random trials, the future event is withdrawn, as in the example above by stopping the sweep hand at various times before it reaches the would-be-target. The probability of initiating an action increases as the time from stoppage of the sweep hand to the would-be target is shortened. Two features of this type of experimental task are of note. First, the go signal is absent and the instant that the go process begins is therefore unknown. Second, the instant that the stop process finishes is unknown even though its effect is sometimes observed. Thus, the latency of the go process as well as the latency of the stop process are unobservables in this type of task.

The second procedure investigates the ability to inhibit an earlier intended action as specified in the presentation of a go signal. On random trials, a stop signal is presented some time after the go signal as scheduled in the SOA. In this type of task, the probability of initiating an action increases as the SOA increases. Two features of this type of experimental task are of note. First, the latency of the go process is observed in the time from the go signal to the onset of the action. Second, the instant that the stop process finishes is unknown even though its effect is sometimes observed. Thus, the latency of the go process is an observable while the latency of the stop process is an unobservable in this type of task. We will consider the complex relation between the go process, the stop process, the SOA and the outcome probability further.

Lappin and Eriksen (1966) observed that constant probabilities of stopping an action at various SOAs require an equal change in the latency of the go process.[3] This led to the suggestion of a stopping deadline (Ollman, 1973) which might be thought of as "... a point of no return at the decision level ..." (Flowers, 1977, p. 174) that locates one stopping latency before the onset of action. If the stop signal is presented before the deadline then the action is stopped whereas if the stop signal is presented after the deadline then the action is produced. The probability of stopping is thus specified in the time relation between the go signal, the stop signal and the stopping deadline. Longer SOAs reduce the time interval from the stop signal to the stopping deadline and so reduce the likelihood of stopping.

Logan and Cowan (1984) offered a race between the go process and the stop process as a formal alternative to a stopping deadline. Here, each process begins to race on presentation of its own signal and the first process to cross the finish line specifies the outcome. If the go process wins the race then the action is initiated and if the stop process wins the race then the action is prevented. The race model, which in effect replaces the deadline with the finish line, well describes the latency relations between the go process, the stop process, the SOA (i.e., the starting handicap of each process) and the probability that each process will win the race (Logan & Cowan, 1984; Logan, Cowan & Davis, 1984; Osman et al., 1986). The race model is presented in further detail below.

1.6 On a race between the go process and the stop process

To date, the best account of how an unfolding action is stopped is provided in a race between the go process and the stop process (Logan & Cowan, 1984). Figure 1.1 details a go latency distribution and a stop process from a stop latency distribution whose variance is not shown for ease of presentation. The intersection of the go latency distribution by the stop process specifies the probabilities of initiating and of stopping an action on any trial that a stop signal is presented. Those go latencies that sample from the faster (left) side of the intersection win the race and specify the

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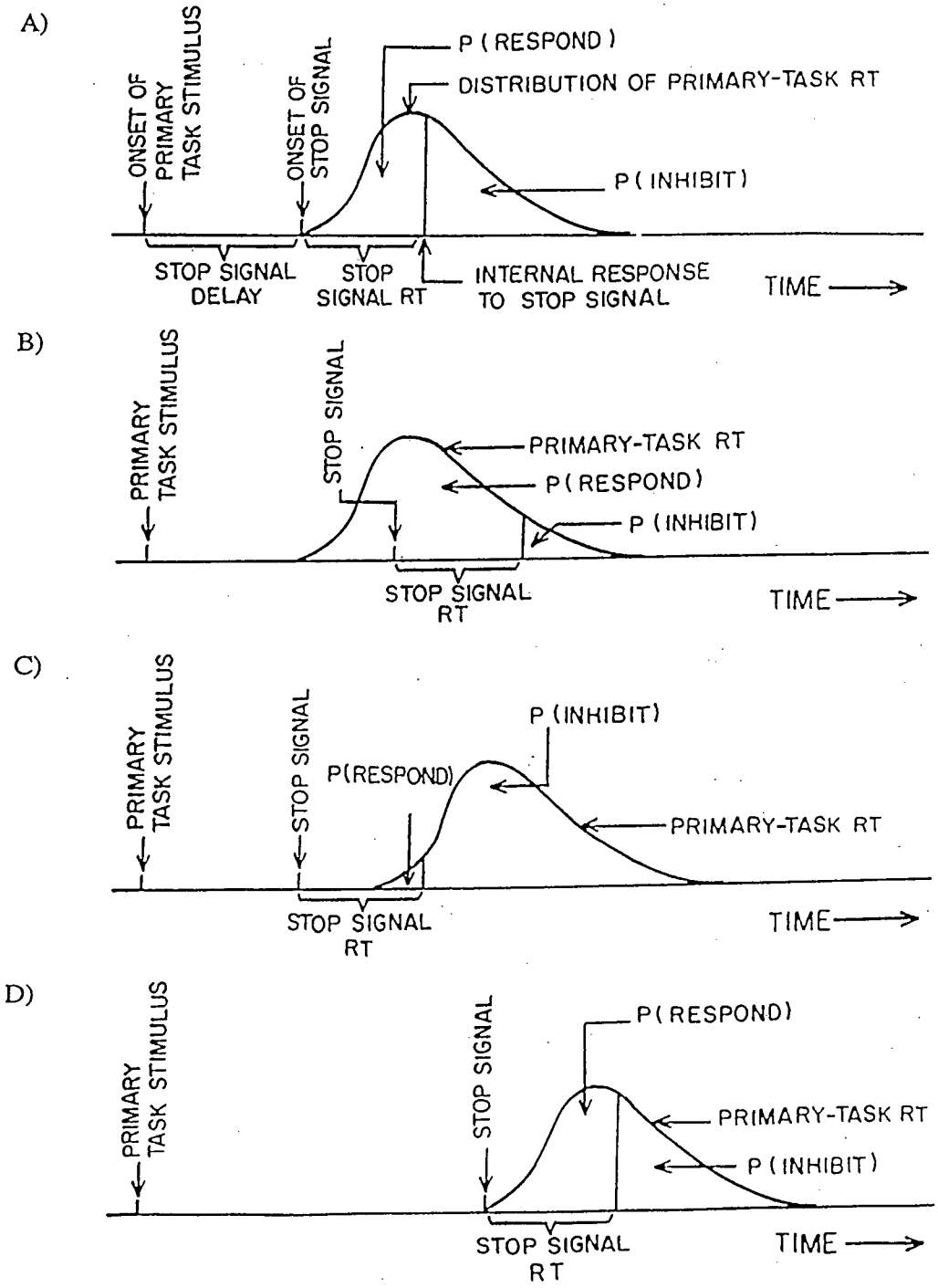
[3] The above relation would be maintained from an equal and opposite change in the latency of the stop process also.

Figure 1.1 Graphic representation of the assumptions and predictions of the horse-race model, indicating how the probability of inhibiting a response - P(inhibit) - and the probability of responding given the stop signal - P(respond) - depend on the distribution of primary-task reaction times, stop-signal reaction time, and stop-signal delay.
Note. From G.D. Logan and W.B. Cowan, 1984, Psychological Review, 91, p. 300.



probability of initiating an action (i.e., p[respond], Figure 1.1), and those go latencies that sample from the slower (right) side of the intersection lose the race and specify the probability of stopping that same action (i.e, p[inhibit], Figure 1.1). The probabilities that the go process wins the race, as well as their respective go latencies, decreases at shorter SOAs as the intersection of the go distribution by the stop process translates from right to left. The intersection of the underlying go distribution by the stopping process as detailed yields the following features.

First, those go latencies that are observed in the presence of a stop process are faster than those go latencies that are observed in the absence of a stop process. This is because only the faster proportion of the underlying go distribution, as specified in the faster (left) side of the stopping intersection, manages to win the race against the stop process. The proportion of go latencies that win the race increases as the SOA increases as the stopping intersection translates the go distribution from left to right (Figure 1.1, panels A and B). Relatedly, the go latencies that win the race increases as the SOA increases because increasingly slower portions of the go distribution are sampled from. Thus, slower go processes win the race with higher probabilities at longer SOAs and faster go processes win the race with lower probabilities at shorter SOAs (Figure 1.1, panels A and B).

Second, the outcome probability for any SOA changes if the go latency distribution changes. If the go latency distribution is simply extended on the time line then the probability of stopping is increased (see Figure 1.1, panels A and C). (Note. This assumes a constant stop latency as in Figure 1.1 or, in more general terms, that the stop latency does not increase to an equal or greater extent than the go latency distribution.)

Third, the outcome probability is constant providing that the stopping process intersects the go latency distribution at the same point, regardless of changes in the go latency distribution, the stop latency and the SOA (see Figure 1.1, panels A and D). In other words, the go latency, the stop latency and the SOA translate on a common time line (c.f., Lappin & Eriksen, 1966).

Fourth, the stop latency (which is an unobservable) can be estimated from the go latency distribution, the SOA and the stopping probability using integration. In Figure 1.1, this is the same as "... moving a vertical line (i.e., the stop latency intersection) across the (go) RT distribution until

the area to the left of the line equals the probability of responding given a (stop) signal, then reading off the value of the time axis, and then subtracting out the stop-signal delay (SOA)" (Logan & Cowan, 1984, p. 302, parentheses ours). These four features listed above hold from the empirical data in support of the race model (see Logan & Cowan, 1984; Logan et al., 1984).

The race model is an important development in a theory of control for two reasons. First, it speaks for independence (contextual and stochastic) between the go process and the stop process (see below). Second, if these conditions of independence hold, the race model provides a method through which the latency distribution of the stop process can be estimated (above). This allows for the comparison of stopping efficacies between participants, conditions and groups. It is beyond the scope of this review to detail the various methods as to how latencies of stopping might be obtained from the race model. Instead, the interested reader is referred to the primary (Logan & Cowan, 1984; Logan et al., 1984) and secondary (Logan, 1994; Band, 1997) sources.

1.7 On the independence of the go process and the stop process

The race model assumes two types of independence - contextual and stochastic - which we shall consider in turn. First, contextual independence assumes that the latency distribution of each process is unaffected by the presence or absence of the other process. That is, the go latency distribution observed in the absence of a stop signal is presumed to be the same as the go latency distribution that the stop process might race against on any trial. The same supposition holds for the stop latency distribution in the presence and absence of a go signal. Second, stochastic independence assumes that each process samples from its own distribution without effect from the other process. These two types of independence are complementary and, in general terms, might be taken to suggest that the go process and the stop process proceed without regard for, or effect on, the other process. This is a key observation that we will return to later.

The strongest evidence for contextual independence and stochastic independence lies in the proximity of the race model's predictions to the empirical data. Specifically, for given latency distributions for each process, shorter SOAs allow for sampling from the faster portion of the go latency distribution while longer SOAs extend the sampling to increasingly slower portions of that

same distribution (see Figure 1.1). The result is shorter go latencies that win the race with lower probabilities at shorter SOAs and longer go latencies that win the race with higher probabilities at longer SOAs (De Jong, Coles, Logan & Gratton, 1990; Logan & Cowan, 1984; Logan et al., 1984; Osman et al., 1986, 1990).

The assumption of independent processes provides for an estimate of the latency of the stopping process. For our purposes, it is sufficient to note that; (a) an estimate of stopping latencies can be obtained as a function of SOA, and (b) the estimates of stopping latencies increases at shorter SOAs (Logan & Cowan, 1984). This latter result is consistent with interference (PRP) effects (where the second process waits for the first process to vacate the bottleneck), although the stopping process is seemingly not subject to these effects (Logan & Burkell, 1986). The preferred explanation for the estimates of longer stop latencies at shorter SOAs lies in the same basis as that for faster go latencies at shorter SOAs. Stop latencies that win the race at longer SOAs are sampled from the faster portions of their underlying distribution, while stop latencies that win the race at shorter SOAs are sampled from the increasingly slower portions of that same distribution. The results from Monte Carlo simulations affirmed that the pattern of longer stop latencies at shorter SOAs, and vice versa, arises as a result of variance in the stopping process (De Jong et al., 1990). Subsequent analyses have shown that the amount of variance in the stopping process determines the increments and decrements of the stopping latencies as the SOAs shorten and lengthen (Band, 1997). The higher the stop variance, the higher the increments and decrements in the stop latencies.

One interesting, if often neglected, aspect of stopping is that the go latencies observed in the absence of a stop signal are slowed as contrasted to a control (i.e., the go latencies in reaction to a solitary go signal). It is not evident whether this slowing of go latencies in the stopping task violates contextual independence. On the one hand, if, as is the case, the go latencies increase (from control) as a result of the uncertainty of stopping, then the go latencies fail to meet the condition of contextual independence. On the other hand, if, as seems the case, the go latencies in the stopping task sample from the same underlying distribution regardless of the presence or absence of a stop process, then the go latencies satisfy the condition of contextual independence.[4] Taken together, we

reason that contextual independence holds insofar as it applies to the race model since the race model assumes the latter case and not the former case.

The increased go latency in the stopping task as contrasted to a control task might be explained from a speed-accuracy trade-off, for as Band (1997, p. 149) points out, the relevant literature "... teaches that the instruction to avoid errors obliges participants to slow down". We prefer a cost-benefit trade-off, much like the finding that the latency in a two-way (left, right) choice task is decreased or increased respectively as a result of the probability of correct or incorrect cueing of the to-be-presented signal (Posner et al., 1978). That said, the trade-offs speak to the same phenomenon. Increased latencies, however, cannot be explained from dual task interference effects that show that "... performance on a task can affect the performance on a competing task ..." (Band, 1997, p. 149) because these latencies are observed in the absence of the stopping process. Regardless, the point at issue is that the experimental protocol affects the behaviour of interest (c.f., the Heisenberg principle) and that the observed ability to stop a voluntary action might in fact be an artifact of the stopping task. In other words, a voluntary action might only be stopped if this contingency is scheduled for ahead of time, so resulting in increased go latencies. In this case, the entire sequence of control from signal presentation through to action initiation would then be regarded as constituting a final ballistic process.

1.8 On a point of no return in the control of thought and action

The finish line to which each process races has been likened to a point of no return. This point is an important rubicon in a race theory of control as it marks the onset of a final ballistic process. This reasoning is consistent with a discrete (stage) view of control that posits that information is processed within a stage before its passage as discrete quanta to the next stage in the series (e.g.,

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[4] It is the uncertainty of stopping, not the process of stopping, that results in the increased go latencies observed in the stopping task. For related findings in regard to the effects of a second (go) signal on the latencies of reaction to a first (go) signal under conditions of certainty and uncertainty, see Herman (1969).

Donders 1969, Sternberg, 1969). The final ballistic process (i.e., the last stage in the series to precede motor output) is a natural extension of this type of processing. The alternative is a continuous process whose control stream is constantly subject to change. It is more problematic for a point of no return, and thus a final ballistic process, to fit into this type of control process.

Osman et al. (1986, 1990) varied factors experimentally that they believed to discriminate between the voluntary (control) and involuntary (ballistic) processes.[5] These authors reasoned that, if Sternberg's (1969) additive factors logic holds, then, for any given SOA; (a) factors that lengthen only the control process will increase the probability of stopping, and (b) factors that lengthen only the ballistic process will not affect the probability of stopping. These predictions follow because if only the control process is extended then longer time is available for stopping, whereas if only the ballistic process is extended then the time available for stopping remains unchanged. The subsequent results were interpreted in support of a "... point of no return (that) occurs very late in the information-processing system" (Osman et al., 1990, p. 183, parentheses ours).

The continuity of human actions affords the possibility of incomplete actions when stopping an action at various times. Incomplete actions are an important finding in regard to the control of stopping. For example, the task performance in a hand (left, right) squeeze task to a target letter flanked with compatible noise (i.e., same letter as target) or incompatible noise (i.e., same letter as the other target) yielded evidence that the incorrectly selected action could be inhibited at any time up to its designated criterion of 25 % maximum force (Coles, Gratton, Bashore, Eriksen and Donchin, 1985; Eriksen, Coles, Morris and O'Hara, 1985). These observances that an unfolding action might be

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[5] The ballistic process refers to the final ballistic process (i.e., b, Footnote 2) and the control process refers to those processes that precede this final ballistic process (i.e., g - b, Footnote 2). In principle, the control process might constitute a multitude of separate control processes and ballistic processes, but the control process as listed is nonetheless subject to stopping (or change) before its entry to the final ballistic process.

stopped "... in very late stages of response processing ... cast(s) considerable doubt on the notion that such stages are ballistic" (De Jong et al., 1990, p. 165, parentheses ours). On this reasoning, De Jong et al. (1990) repeated the Coles' studies (above) using the stop signal task and showed that the earlier intended action can be inhibited (interrupted) at any time before its completion at 25% maximum force. These data were interpreted as evidence yielding "... no ballistic process immediately prior to the response ..." (De Jong et al., 1990, p. 178).

Whether or not the claims of De Jong et al. (1990) stand against the claims of Osman et al. (1986, 1990) depends on how the onset of an action is defined. If, on the one hand, the criterion for the onset of the action (hand squeeze) is taken as the onset of force (i.e., > 0 % maximum), then the reported stopping effects occur after the onset of the action and before its completion. These data then speak neither for nor against a point of no return before the onset of the action (c.f., Osman et al., 1986, 1990), but they speak against a point of no return after the onset of the action and before its completion. If, on the other hand, the criterion for the onset of the action (hand squeeze) is taken as the onset of the designated force criterion (i.e., > 25 % maximum), then the reported stopping effects occur before the onset of the action. These data then speak against a point of no return before the onset of the action (c.f., Osman et al., 1986, 1990). Thus, while the findings of De Jong et al. (1990) and of Osman et al. (1990) might or might not contradict each other, as outlined above, a unitary position regarding a point of no return cannot be reached from these data. The probity of a point of no return in the control of voluntary action thus remains an outstanding issue in the extant stopping literature to date.

1.9 Scope of the present research

The series of studies presented herein seeks to investigate the nature of stopping an earlier intended maximal speeded action. Incomplete movements are an important finding in this regard. Various chronometric, kinematic and electro-physiological (i.e., EMG) measures will be used to describe the effects of stopping on a voluntary action at various times. The principal aim of these studies is to synthesize the race theory for the control of thought and action with some basic neuro-physiological principles on which motor control is necessarily predicated.

1.10 Statement of ethics

Each experiment herein abides by the ethical guidelines of the University of British Columbia.

2 Experiment I

2.1. On a point of no return in the control of thought and action revisited

Two issues stem from the preceding monologue. First, the typical finding that the latencies of the go process are slowed by the uncertainty of stopping gives pause to the view that a maximal speeded action can in fact be stopped.[6] Second, the unresolved issue with regard to a point of no return, and thus a final ballistic process might be further informed from the observances of incomplete actions. We will consider briefly each issue in turn.

First, the race model presumes; (a) contextual independence, in that the latencies of each process remain unaffected by the presence or absence of the other process, and (b) stochastic independence, in that the sampling from each distribution is random. If the race model is to be used as a theory of control to explain action then the go process and the stop process must abide by the conditions listed above. The assumptions of contextual independence and stochastic independence would suggest that the go mechanism and the stopping mechanism function independently of each other, a term that we use hereafter to indicate that each process proceeds in its usual way regardless of the presence or absence of the other process. Evidence for functional independence was observed in the stop-signal task in the abrupt interruption in the lateralized readiness potentials (LRPs), surface EMGs and force records (De Jong et al., 1990), evidence that these authors interpreted in support of the race model (p. 178).

We noted earlier that the go latencies are slowed as a result of uncertainty in the stopping task. In a dual task study in which a random second signal required a reversal of the first action, Henry and Harrison (1961, Table 1) reported that, on average, go latencies to the first signal increased by 22 ms, as compared to control latencies, even though waiting for the stop-reversal signal was punished

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[6] The observed latencies of reaction to solitary signals are of like order to the estimated stopping latencies of about 200 ms (Logan et al., 1984). To stop a maximal speeded action after the go signal under these time constraints would therefore be very difficult.

with a mild electric shock. Of like order, Slater-Hammel (1960) reported a 26 ms constant error (delay) in a coincident-timing task on those trials that a stop signal was not presented. These data attest that the stopping (or reversal) task changes the ability somewhat to perform the action that is to be stopped by extending its go latencies, so providing for the eventuality of stopping should it arise on any trial. Thus, the entire go process might be considered ballistic if it cannot be stopped without *a priori* slowing (see Footnote 6). This experiment sought to offset slowing in the go process by using a monetary pay-off schedule in order to try to address this question.

Second, studies that have investigated the race model have tended to focus on the behavioural properties of the action. This has typically led to the classification of an action as discrete. This procedure tends to overlook those actions initiated in the presence of a stop signal that differ in form (i.e., incomplete actions) from those actions initiated in the absence of a stop signal. In fact, incomplete actions offer important additional information in the analysis of stopping control (see for example Coles et al., 1985; De Jong et al., 1990; Eriksen et al., 1985). In this study, we analyse the effects of stopping on a maximal speeded action (elbow extension) at various times from surface EMG data for both complete and incomplete actions.

2.2 Method

2.2.1 Participants

Twelve (6 males, 6 females) right hand dominant participants ranging in age from 19 years through 47 years were recruited in this study. Testing occurred in a single session. Each participant received financial remuneration on completion of the session (see later).

2.2.2 Apparatus

The right forearm of the participant was strapped prone to an arm manipulandum with the elbow joint centred above the axis of rotation in the transverse plane. Surface EMG data were recorded using silver/silver chloride electrodes positioned on the muscle bellies of the triceps brachii (lateral head) and the biceps brachii (long head). The EMG signal was amplified (1-100 K range) using a multi-channel EMG system (model 544, Therapeutics Unlimited Inc.) and sampled at 1000 Hz using a 12 bit analogue-digital converter. Kinematic data were recorded from; (a) an optical

encoder that sampled angular displacement at 1000 Hz using a custom made computer interface card (Dynapar E20-2500-130), and (b) a Kistler accelerometer (type 8638B50, 50 G), positioned 42 cm from the centre of rotation, whose angular acceleration signal was filtered with an active low pass filter (Krone-Hite 3750) at 50 Hz before being sampled at 1000 Hz.

2.2.3 Procedure

Extending and flexing the right elbow in the transverse plane caused a response cursor presented on an oscilloscope screen to move in the right and left horizontal direction respectively. The participant oriented his (her) arm to a home position by aligning vertically the response cursor to a signal cursor presented in a fixed location at the left of the oscilloscope screen. The signal cursor was matched to a location of -22½ degrees. For reference, full elbow extension would be reached at +90 degrees.

Once the signal cursor and the response cursor were aligned, the participant initiated the start of each trial with a verbal acknowledgment of readiness. The warning signal (a brief offset and onset of both the signal cursor and the response cursor) marked the beginning of each trial and preceded the go signal (the offset of the signal cursor) by a variable foreperiod (1000 ms - 3500 ms). The stop signal was a tone sounded for 100 ms at 1000 Hz. On-line kinematic feedback (i.e., displacement, velocity and acceleration) of the movement was presented by way of the response cursor displayed on the oscilloscope screen in real time.

The go signal instructed the participant to extend forcefully his (her) elbow as fast as possible from the home location of -22½ degrees to a general target amplitude of at least 32½ degrees. The target location (i.e., +10 degrees) was presented as a vertical line overlaid on the oscilloscope screen. The participant was informed that the target location represented a general minimum extension requirement only and that the response cursor should cross the vertical line on each trial. We accepted trials that undershot the target on occasion but we rejected trials that fell systematically short of the target location. Off-line augmented feedback in relation to task performance (see later) was presented after each trial using a computer monitor positioned alongside the oscilloscope screen.

2.2.4 Protocol

2.2.4.1 Experimental tasks

Three experimental tasks - go-only (control), inhibit and forced - were given to each participant. The structure of the experimental protocol determined that the go-only task must precede the forced task because the pay-off schedule awarded in the latter was based on latency performance in the former (see below). The order of the go-only task and the inhibit task was not varied because no order dependency of tasks was expected.

Typically, latencies of reaction are reduced with practice at the task and this finding applies in the stopping task also (see for example Jodo & Inoue, 1990). It remains unclear however what, if any, effect that practice might have on the probabilities of stopping as a function of SOA. If go latencies are reduced more than stop latencies then the probability of stopping would be decreased for a given SOA. If, however, stop latencies are reduced more than go latencies then the probability of stopping would be increased for a given SOA. Lastly, if go latencies and stop latencies are reduced by the same amount then no effect on the probability of stopping for a given SOA would be observed. We did not investigate the stopping data for effects of practice since, while important in its own right, it is not fundamental to our question at large, namely the mechanism through which stopping an earlier intended voluntary action is effected.

The go-only task (40 trials) required the participant to react to the go signal and to extend the right elbow as fast as possible. The inhibit task (60 trials) required the participant to react to the go signal and to extend the right elbow as fast as possible and, also, to try to stop that action should a stop signal be presented. The participant was informed that the first instruction outranked the second instruction in its importance. In the forced task (60 trials), the participant was informed that a monetary pay-off schedule related to participant performance was designed to reinforce these same task (inhibit) instructions as well as their precedence. Stop signals were presented with .333 probability. SOAs were presented on stop signal trials at -100 ms, -50 ms, 0 ms, 50 ms and 100 ms with equal probability. Catch trials consisting of neither a go signal nor a stop signal were presented

with .100 probability in each task to discourage anticipation. The stop signals, SOAs and catch trials were counterbalanced over trials.

2.2.4.2 Pay-off schedule

The go latency was assigned as "fast", "intermediate" or "slow" on each trial in the inhibit task and the forced task. These labels were assigned individually on the basis of each participant's performance in the go-only task in an effort to offset the slowing of go latencies that typically accompany the stop signal task. Specifically, using the mean ($\overline{X}_{go\text{-}only}$) and the standard deviation ($SD_{go\text{-}only}$) and a two-tailed t-test at a level of significance of $p = .05$, we found that $\overline{X}_{forced}$ must lie within .446 $SD_{go\text{-}only}$s of $\overline{X}_{go\text{-}only}$ for $\overline{X}_{forced}$ and $\overline{X}_{go\text{-}only}$ to be non-significant ($p > .05$). (We presumed *a priori* that $SD_{go\text{-}only}$ was equal to SD_{forced}.) Thus, go latencies in the inhibit task and the forced task were classified as fast, intermediate or slow if they fell below, within or above the bounds of $\overline{X}_{go\text{-}only}$ and .466 $SD_{go\text{-}only}$ respectively (i.e, fast < $\overline{X}_{go\text{-}only}$ - .466 $SD_{go\text{-}only}$, $\overline{X}_{go\text{-}only}$ - .466 $SD_{go\text{-}only}$ > intermediate > $\overline{X}_{go\text{-}only}$ + .466 $SD_{go\text{-}only}$ and slow > $\overline{X}_{go\text{-}only}$ + .466 $SD_{go\text{-}only}$).

Those trials that yielded fast go latencies in the absence of a stop signal or no elbow extensions (or flexions) in the presence of a stop signal were assigned as "correct" responses. Likewise, slow go latencies in the absence of a stop signal or elbow extensions (or flexions) initiated in the presence of a stop signal were assigned as "incorrect" responses. Intermediate go latencies in the absence of a stop signal were assigned as neither correct responses nor as incorrect responses.

In the forced task only, a $0.50 reward for a correct response and a -$0.25 penalty for an incorrect response was added on each trial to the participant's balance. In addition, a $0.50 bonus was added on each trial (in the absence of a stop signal) that the updated $\overline{X}_{forced}$ fell below a known target latency (i.e., $\overline{X}_{go\text{-}only}$ + .466 $SD_{go\text{-}only}$). The $\overline{X}_{forced}$ was updated on each trial that a go signal was presented in the absence of a stop signal. The participant was paid his (her) final balance at the end of the testing session to a $5 minimum. Knowledge of the $5 minimum payout was withheld from the participant until the testing session was completed.

2.2.5 Data Collection

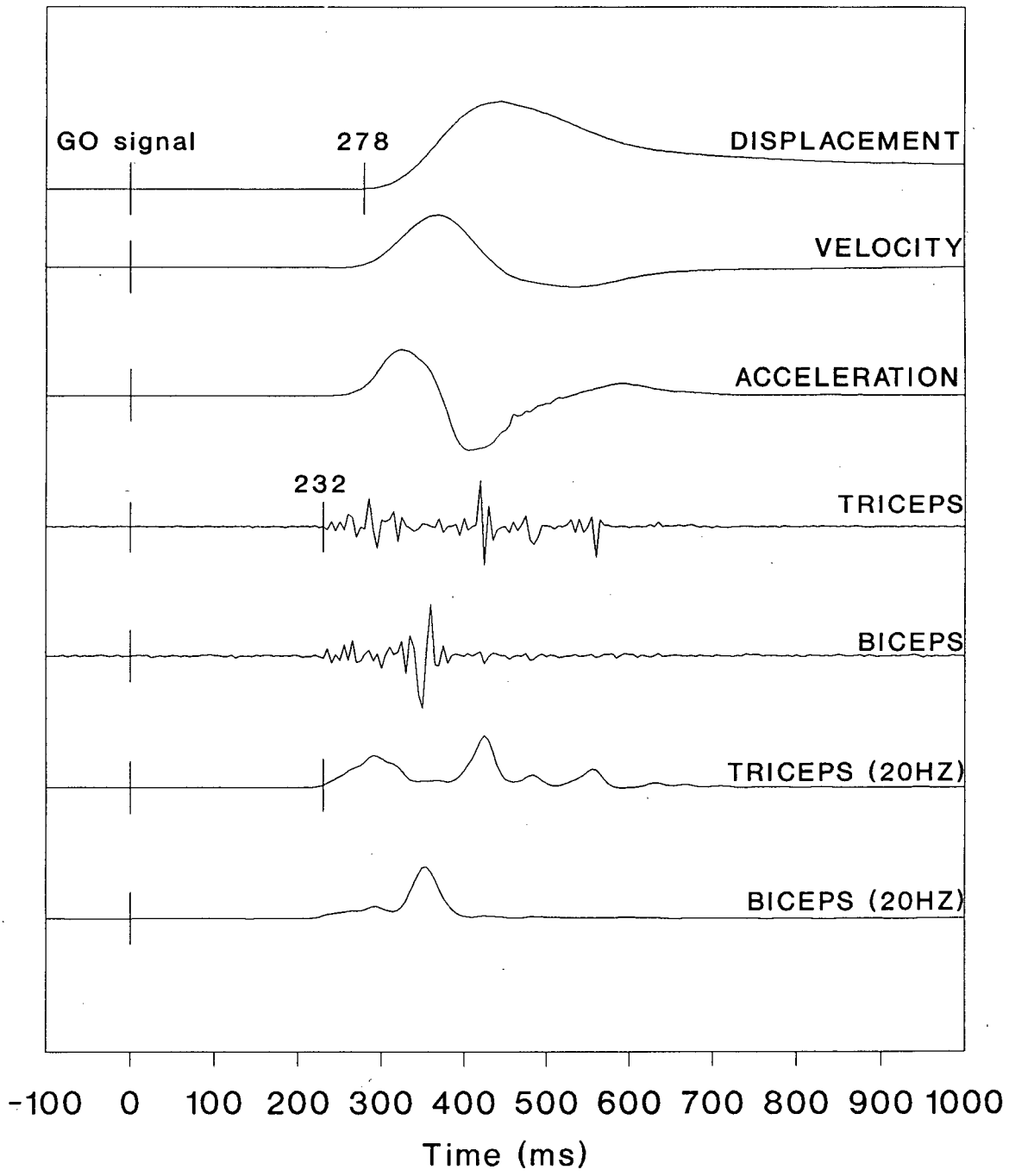
Kinematic data and EMG data were collected for each trial (see Figure 2.1). On each trial, the experimenter identified the onset of displacement (i.e., the latency of reaction) from on-line inspection of the data and, in the process, so determined exclusively whether the response was "moved" or "stopped". The go latency, the type of go latency (fast, intermediate, slow) and the updated $\overline{X}$ for that task was presented as feedback on each trial in each task condition that a stop signal was not presented. (The type of go latency was not presented in the go-only task.) The type of response (moved, stopped) was also provided on each trial in each task condition. In the forced task, the associate reward (or penalty) and bonus, if any, for that trial, as well as the updated monetary balance, were also presented as feedback on each trial. Go latencies were re-analysed post hoc for each trial following completion of the study.

2.2.6 Data Analysis

Figure 2.1 details an example of the kinematic (displacement, velocity and acceleration) data, the untreated EMG (triceps and biceps) data and the rectified-filtered (20 Hz) EMG (triceps and biceps) data associated with a maximal speeded elbow extension for Participant 5. The linear envelopes of the EMG data (latter two traces, Figure 2.1) were obtained by filtering the rectified data at 20 Hz. The latencies of reaction (RT) and triceps EMG onset (TEMG) were recorded post hoc by the experimenter by identifying the onset of extension and the onset of the untreated triceps EMG respectively (see Figure 2.1, RT = 278 ms, TEMG = 232 ms).

The onset of an elbow extension was defined as the onset of displacement (extension) from baseline in which the optical encoder first twice registered a positive change within successive 20 ms time windows. TEMG markers were placed at the beginning of triceps EMG activity as observed over and above the baseline signal. TEMG constitutes the pre-motor component of the action and it is often taken as an index of the latency of the processing overheads that are responsible for generating an action. The time interval between TEMG and RT (i.e., motor time, MT) constitutes the motor component of the action and it is often taken as an index of the electro-mechanical delay from

Figure 2.1 Kinematic (displacement, velocity and acceleration) data and EMG (triceps untreated, biceps untreated, triceps rectified-filtered and biceps rectified-filtered) data for a maximal speeded elbow extension observed in the go-only task for Participant 5. The rectified EMG data are filtered at 20 Hz.



the onset of activity in the muscle effectors to the onset of its observed effect (e.g., Anson, 1989).

We obtained the $\overline{X}$ and the SDs for RT, TEMG, MT, peak velocity and time to peak velocity for each participant in each task condition, excluding those trials in which a stop signal was presented. Individual trials on which any dependent variable fell three SDs outside its $\overline{X}$ were discarded in their entirety as untypical trials. This analysis was repeated in stepwise fashion for each dependent variable in turn until the analytic condition was met. We discarded 12.5% trials in 2 cases, 10% trials in 3 cases, 7.5% trials in 5 cases, 5% trials in 3 cases, 2.5% trials in 10 cases and 0% trials in 13 cases. Catch trials, used to detect anticipation, were not analysed in this study.

2.3 Results and Discussion

2.3.1 Differential slowing effects of the countermanding tasks on reaction time

We analysed the TEMG, MT and RT data using a one way (task) repeated measures ANOVA and followed up significant F-ratios with Tukey post-hoc analysis (Stevens, 1990, p. 199). The TEMG data were significantly different across tasks, $F(2,22) = 38.6$, $p < .001$, $HSD = 24.3$, $p < .01$, as a result of incremental slowing in the forced task and the inhibit task (see Table 2.1, $N = 12$). The RT data showed the same effect, $F(2,22) = 35.9$, $p < .001$, $HSD = 26.1$, $p < .01$, which is only to be expected given the substantial proportion of shared variance between the TEMG and the RT variables. Relatedly, as expected, the MT data were insensitive to task condition, $F(2,22) = 1.4$, $p > .05$, and, in effect, reflect a constant as a result of physical delay.

Further analysis indicated individual differences between participants. In particular, participants were either able ($N = 4$) or unable ($N = 8$) to prevent the slowing of go latency in the forced task.[7]

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[7] We assign the terms "able" and "unable" based on performance in the stopping task in arbitrary fashion and as such these terms might be interchanged respectively with "willing" and "unwilling" instead. For example, Buckolz, Hall and Alain (1982) reported that participants who were unable (unwilling) to prevent slowing of the go latencies in the stopping task (see above) were in fact able (willing) to do as a result of changed experimental instruction.

Table 2.1. Descriptive statistics for the time to triceps EMG onset (TEMG), motor time (MT) and the latency of reaction (RT) by task (go-only, inhibit, forced) across participants (able, unable).

			Task					
			Go-only		Inhibit		Forced	
Group	Variable	N	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD
	TEMG	12	199.8	25.7	265.1	38.5	238.7	37.7
	MT		55.9	6.9	58.2	7.4	57.5	6.9
	RT		255.8	22.7	323.3	35.4	296.2	35.6
Able	TEMG	4	205.4	23.5	259.3	43.2	211.1	31.3
	MT		57.7	6.8	60.7	7.4	56.8	5.6
	RT		263.1	25.0	320.0	42.6	267.9	34.2
Unable	TEMG	8	197.1	27.8	268.1	38.7	252.5	34.1
	MT		55.1	7.3	56.9	7.6	57.9	7.9
	RT		252.1	22.3	325.0	34.4	310.4	28.4

Note. Units of measurement are milliseconds. N = number of participants. $\overline{X}$ = mean. SD = standard deviation.

We therefore subjected the data to a two way (group by task) ANOVA with repeated measures on the second factor (see Table 2.1). This variance analysis revealed significant interaction effects for both TEMG, $F(2,20) = 8.6$, $p < .01$, and RT, $F(2,20) = 9.0$, $p < .01$, an indication that, for whatever reason, different strategies were likely used by the participants in an attempt to cope with the experimental demands. That a monetary incentive was insufficient for some participants to prevent the slowing of the go latencies is perhaps not too surprising if one recalls that even a mild electric shock failed to prevent interference from a possible second signal (Henry & Harrison, 1961). For the time being, exactly how the uncertainty of stopping (as opposed to the process of stopping) affects the go process remains an open question.

Two follow up one way (task) repeated measures ANOVA - one for each group (able and unable) - revealed significant differences across tasks for TEMG, (able) $F(2,6) = 15.8$, $p < .01$, (unable) $F(2,14) = 58.9$, $p < .01$, and RT, (able) $F(2,6) = 16.0$, $p < .01$, (unable) $F(2,14) = 55.7$, $p < .01$, but no significant difference for MT, (able) $F(2,6) = 2.6$, $p > .05$, (unable) $F(2,14) = 1.3$, $p > .05$. Tukey post hoc analysis showed that the differences in TEMG ($HSD = 47.1$, $p < .01$) and RT ($HSD = 49.9$, $p < .01$) for the able group were the result of slowing in the inhibit task only (see Table 2.1, $N = 4$). In contrast, the same differences in TEMG ($HSD = 23.8$, $p < .01$) and RT ($HSD = 25.2$, $p < .01$) for the unable group were the result of indiscriminate slowing in both the inhibit task and the forced task (see Table 2.1, $N = 8$).

2.3.2 The order of motor unit recruitment is specified in Henneman's (1957) size principle

In general, motor units (the smallest functional units of contractile muscle) are recruited according to size as a result of the intrinsic properties of their motor neurons (Henneman, 1957). Small motor neurons that attach to slow twitch fibres discharge early and large motor neurons that attach to fast twitch fibres discharge late as a result of lower levels and higher levels of excitatory input to the motor pool. (The motor pool houses the collective of motor neurons that project to a particular skeletal muscle.) Small motor neurons and large motor neurons increase their discharge frequencies to their maximum rates with increased excitation, the result being that small motor neurons reach their rate limits (i.e., maximum discharge frequencies) early while large motor neurons are only just beginning to be recruited. Ramp increases in excitatory input to the motor

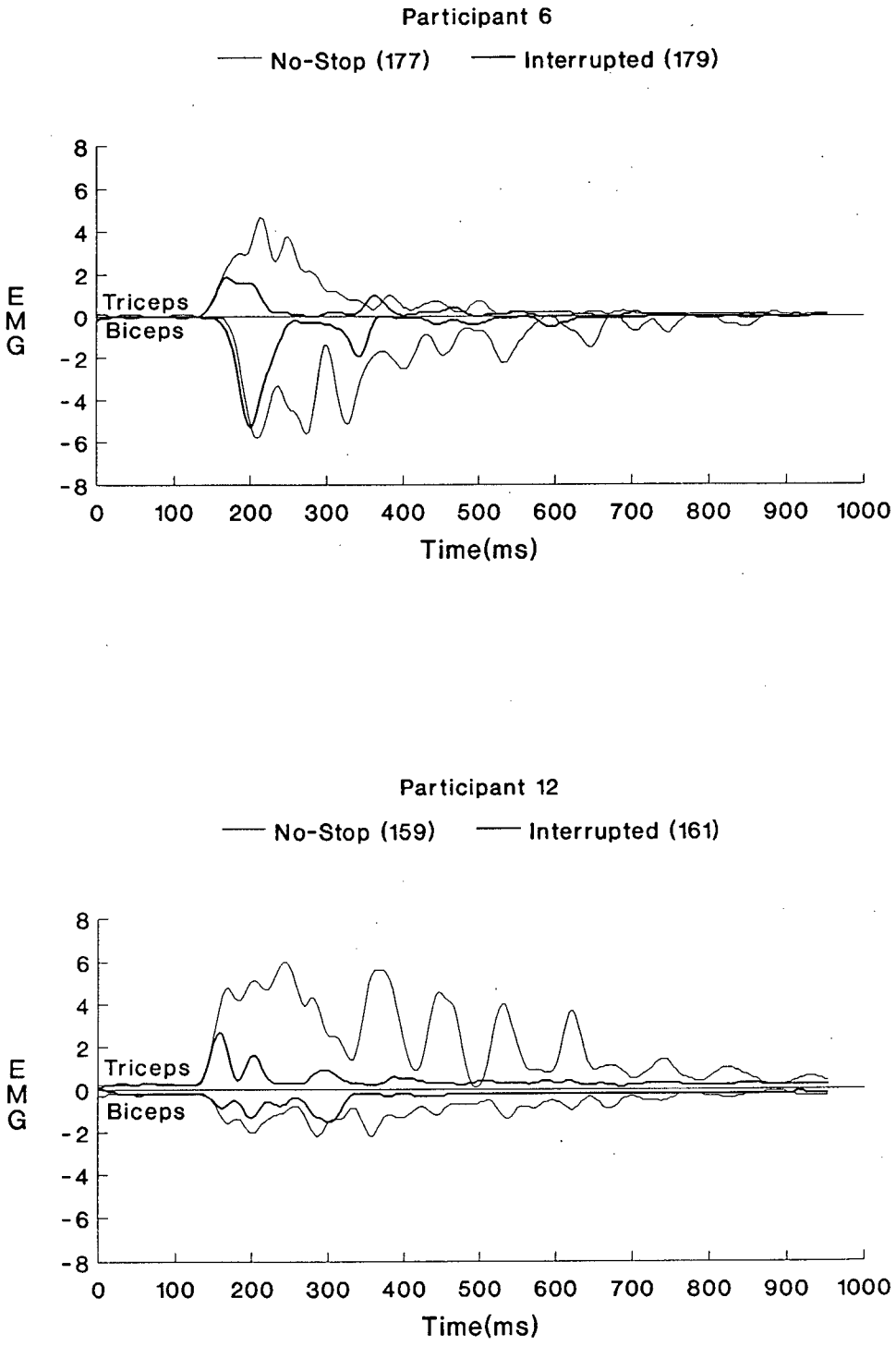
pool recruits more motor units of increasing size whose individual contribution of force output to the total force output is in accord with Weber's fraction law (Ghez, 1991). Relatedly, the drop-out of already active motor units in the motor pool as a result of varying types of inhibitory influence occurs in the reverse order in which they were recruited (Henneman & Mendell, 1981; Miles & Turker, 1986). That motor neurons discharge as a function of their intrinsic properties (i.e., size) allows for simplicity in the control of force as the level of force is specified directly in the level of synaptic drive to reach the motor pool.

The control of action can be reduced to the control of motor neurons that deliver force to skeletal muscle. Each motor discharge generates action potentials in the muscle fibres of its corresponding motor unit. These motor unit action potentials that result from motor discharge cause skeletal muscle to contract and a short time thereafter to generate a given force output (above). The size, duration and sign of these action potentials as recorded by the surface electrodes are dependent on various factors including the size of the motor neuron, the distance of the muscle fibre action potentials from the surface electrodes, and their direction of travel in relation to the surface electrodes. In brief, surface EMG records are the temporal-spatial sum of each motor unit action potential within the vicinity of the detecting electrode. (For a reconstruction of surface EMG from the individual motor unit action potentials, see Section 4.3.5 Generation of EMG from the pattern of motor neuron discharge.) We use surface EMG records in this study as the principal measure with which to analyse and to infer to the principles of control that regulate the initiation and stopping of a voluntary action.

2.3.3 Interrupted responses and partial responses when initiating and stopping a speeded elbow extension

Figure 2.2 shows the rectified-filtered (20 Hz) EMG for the triceps and biceps (inverted) from a maximal speeded elbow extension for Participant 6 (upper panel) and Participant 12 (lower panel). Each panel of EMG data details an example from two near adjacent trials, as indicated from their trial numbers (located in parentheses). The trial pairs are presented together in the same panel after synchronising their triceps EMG onsets (i.e., TEMGs). In one trial (no-stop) a stop signal was not

Figure 2.2 EMG (rectified-filtered 20 Hz) triceps and biceps (inverted) profiles showing an interrupted (late) response as contrasted to a no-stop response from the same participant. (Parentheses indicate the trial number for that response.) Units of EMG are arbitrary.



presented and in the other trial (interrupted) a stop signal was presented. Thus, any differences in the EMG data between the two trials can be attributed to the effect of the stopping process.

The abrupt tail off in EMG of both the triceps and the biceps in the interrupted data as contrasted with the no-stop data shows that the stopping process forcefully stops the action after its typical onset and before its completion (c.f., De Jong et al, 1990). The EMG traces follow the same ramp-like time course before its cut-off point (i.e., the point of effect of the stopping process), which suggests like patterns of recruitment and rate discharge of motor units before any evidenced effect of the stop process on the go process. In this example, the stopping process arrives at its point of action, presumed to be the motor pool, shortly after the go process, whereupon it proceeds to swiftly end any further motor discharge. We hereafter refer to this type of response as an interrupted response by virtue of its observed effect on the EMG traces.

Figure 2.3 details two additional interrupted responses from Participant 2 (upper panel) and Participant 10 (lower panel). Each of these interrupted responses are cut-off early in their EMG trace after onset, as contrasted to their respective no-stop responses. This is presumably a result of the stop process reaching the motor pool within a shorter time interval of the go process than that for a late interrupted response (c.f., Figure 2.2). In Figure 2.4, sub-maximal EMG data are observed at onset for Participant 6 (upper panel) and for Participant 12 (lower panel). These data possess marked characteristics as typified in their reduced EMG onsets, as contrasted to the interrupted responses from the same participants (c.f., Figure 2.2). Importantly, this observation mandates an alternative explanation to that rendered for the interrupted responses, namely that the marked reduction in EMG onset is a result of the stop process managing to suppress, yet failing to prevent, the go process from reaching the motor pool. These sub-maximal EMG data likely result from the recruitment of fewer motor neurons and, in all likelihood, a reduced frequency of discharge. We hereafter refer to this type of response as a partial response, once again by virtue of its effect on the EMG traces.

We rule out signal noise as the source of the partial response on three counts. First, an analysis of the data revealed that the sub-maximal EMG data above baseline was associated (90.9%) with a

Figure 2.3 EMG (rectified-filtered 20 Hz) triceps and biceps (inverted) profiles showing an interrupted (early) response as contrasted to a no-stop response from the same participant. (Parentheses indicate the trial number for that response.) Units of EMG are arbitrary.

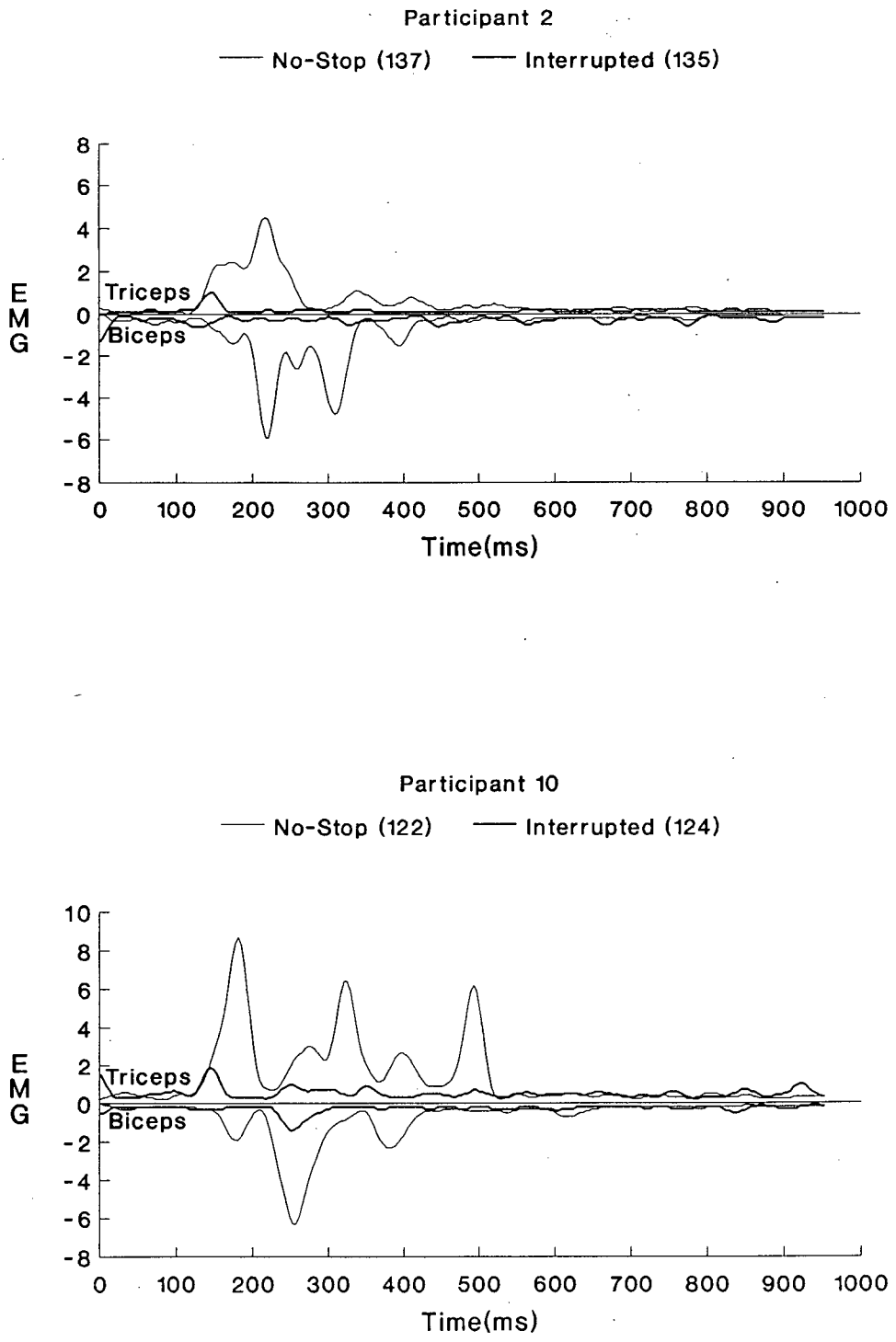
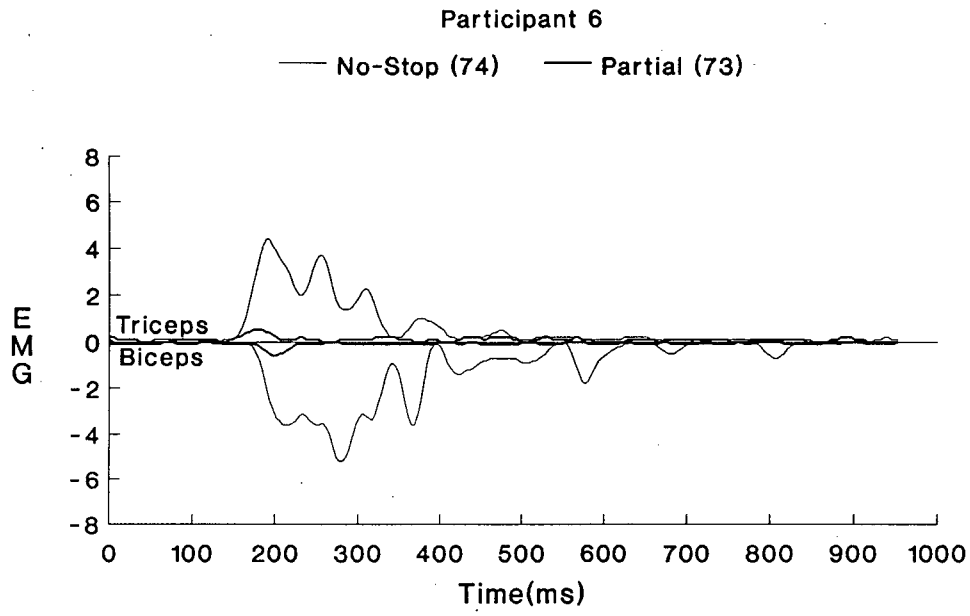
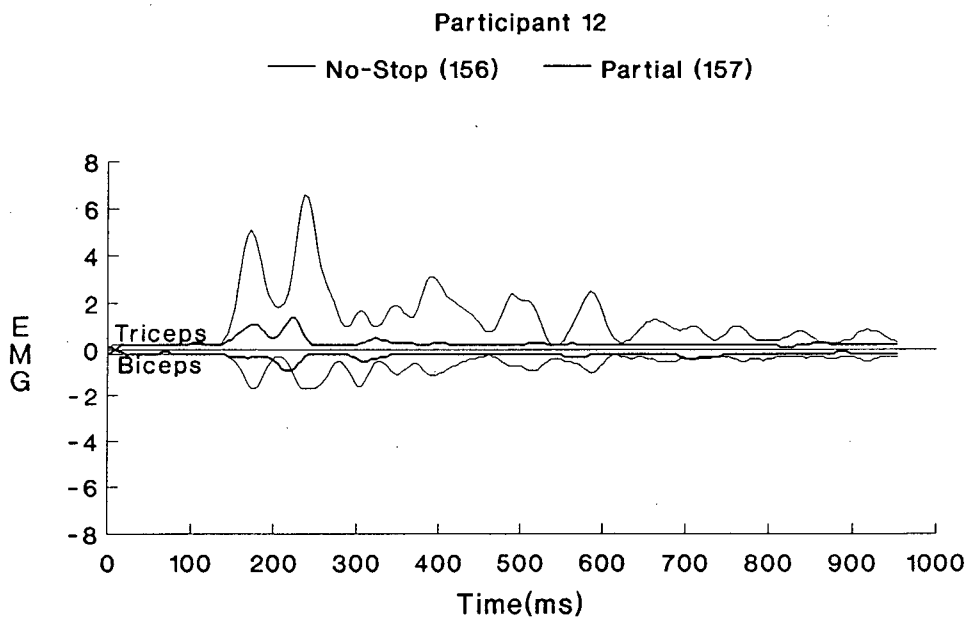


Figure 2.4 EMG (rectified-filtered 20 Hz) triceps and biceps (inverted) profiles showing a partial response as contrasted to a no-stop response from the same participant. (Parentheses indicate the trial number for that response.) Units of EMG are arbitrary.





consequent change in extension. Importantly, displacement was recorded in this study by an optical encoder that is less subject to noise than the EMG signal. Second, in some cases, systematic (pulse like) signal noise was observed above baseline with no consequent change in extension. Thus, the sub-maximal EMG data that typifies a partial response leads to a resultant change in extension while the observed noise in the EMG signal does not. Third, the observance of a partial response extends across participants, SOA and task condition (see Table 2.2), a finding that lends further testimony to its distinction.

The race model might account for the partial responses by simply considering them as early interrupted responses, or, alternatively, as a failure of the stop process to prevent completely the go process from reaching the motor pool. We suggest that the partial responses, as typified in sub-maximal EMG onsets, might result from leakage of the go process as a result of a non-instantaneous stopping process. Since it would necessarily require some time for the stopping process to quieten the activity of a fully fledged go process, some activity of the go process might well escape stopping and leak out as sub-maximal EMG onsets. If this interpretation is correct, then the stopping process must arrive at the motor pool some time before the go process, otherwise reduced EMG onsets would not be observed. Thus, not only must the point of no return be late in the processing of an action, it must also precede the arrival of the go process at the motor pool by the time needed to quieten successfully a fully prepared go process. So far, this conclusion supports Osman et al. (1986, 1990) who reported a final ballistic process to occur very late in the information processing system.

2.3.4 Evidence for a phantom point of no return in the preparation of a speeded elbow extension

We propose the following mechanism as detailed in Figure 2.5 to account for the interrupted responses and the partial responses. Here, the go latency is varied and the stop latency and the SOA are held constant for ease of presentation only. The latency relations of the go process and the stop process with reference to the finish line specify the various types of responses reported in this study.

We suggest that the finish line for the race between the go process and the stop process is the motor pool, its excitation as indexed in EMG leading to some type of action. Full reponses are

Table 2.2. Frequency of interrupted responses and partial responses by participant, the time between presentation of the go signal and presentation of the stop signal (SOA) and task.

Interrupted	Task										
	Inhibit SOA(ms)					Forced SOA(ms)					
Participant	-100	-50	0	50	100	-100	-50	0	50	100	Total
2					2					[a]2	4
3		1	1		2			1	1		6
4			1		1						2
5				1	1						2
6										1	1
7	1			2	1	3	3		1		11
8	3	1							1	1	6
10		1						3	2	1	7
11				1	2				1		4
12	1			2			1	2	1		7
Total	5	3	2	6	9	3	4	6	7	5	50

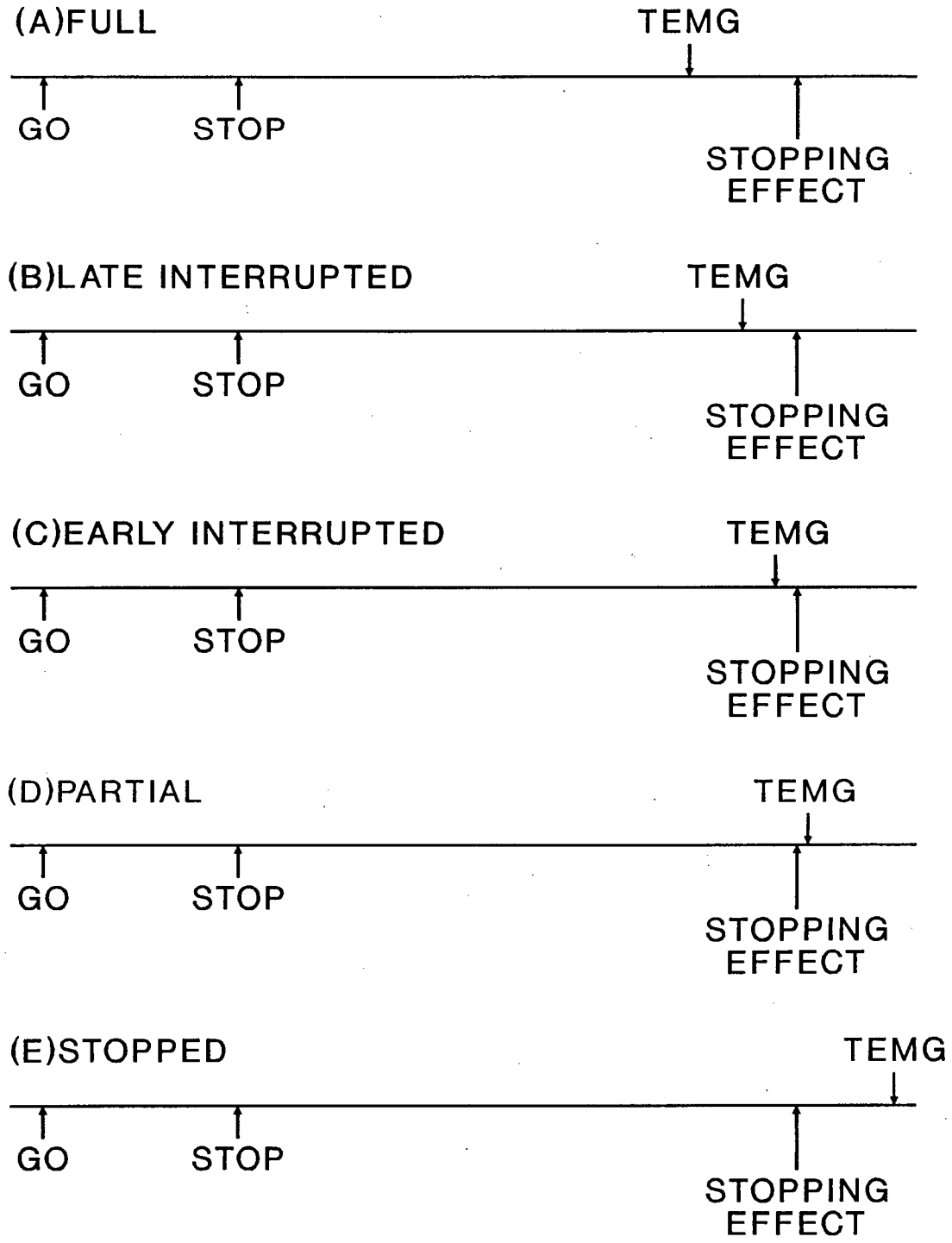
Partial	Task										
	Inhibit SOA(ms)					Forced SOA(ms)					
Participant	-100	-50	0	50	100	-100	-50	0	50	100	Total
2											-
3					[b]1					1	2
4		1		1						1	3
5	2									[a]1	3
6					2						2
7		[a]1	[b]2	1	[b]1		1				6
8									1	[b]2	3
10			[a]1	[b]1							2
11				3	1			2			6
12	1	1			[b]1	2		[b]1			6
Total	3	3	3	6	6	2	1	3	1	5	33

Note. [a]No change in displacement observed. [b]No criterion change in displacement observed. Able = Participants 1, 7, 8 and 12. Unable = Participants 2, 3, 4, 5, 6, 9, 10 and 11. Participant 1 was excluded from EMG analysis because of error in EMG data scaling which compromised the signal. Participant 9 was excluded from EMG analysis because of excessive signal noise.

observed when the go process wins the race by a sufficient margin such that the stopping process has no effect at the motor pool (Figure 2.5A). Interrupted responses are observed when the go process wins the race by a lesser margin than that for full reponses such that the stopping process takes effects at the motor pool some time shortly after the usual arrival of the go process (Figure 2.5B and 2.5C). Whether the action is interrupted late (Figure 2.5B) or early (Figure 2.5C) after its onset is simply a function of the time interval between the arrival of the go process and the stop process at the motor neuron pool. The action is interrupted earlier as the go-stop winning margin is reduced. Interrupted responses are marked in each case by an abrupt cut-off in the EMG trace from its usual time course (see Figures 2.2 and 2.3). The partial response is markedly different from an interrupted response. Here, the stop process converges on the motor pool a little before the go process (Figure 2.5D) but it fails to prevent completely the later arrival of the thus weakened go process. The result is partial EMG activity that is graded on account of the number of motor units recruited and their discharge frequencies (see Figure 2.4). Stopped responses are observed when the stop process wins the race by a sufficient margin such that the go process is suppressed before it can reach the motor pool (Figure 2.5E).

The difference between the various types of response lies in the timing with which the go process and the stop process act on the motor pool. This, in turn, specifies the recruitment and rate discharge of the motor neurons that results in EMG onset. For interrupted responses, the stopping process arrives at the motor pool some time after the go process (see Figure 2.5B and 2.5C) and so acts to prevent further recruitment and rate discharge, as well as stopping the effects of the already active motor neurons. For partial responses, the stopping process arrives at the motor pool some time before the go process (see Figure 2.5D) and, in so doing, acts to weaken the go process before its subsequent arrival. The result is reduced synaptic input to reach the motor pool as evidenced in sub-maximal EMG onsets. It follows that the weakened input to reach the motor pool remains subject to further effects of stopping as the stop process continues to suppress the go process completely. Thus, the stop process can inhibit the go process at all times before EMG onset to

Figure 2.5 Proposed latency relations detailing (A) a full response, (B) a late interrupted response, (C) an early interrupted response, (D) a partial response, and (E) a stopped response as a function of variance in go latency. The time from presentation of the go signal and presentation of the stop signal (SOA) is constant. Zero (no) variance in the stop process is assumed for ease of presentation. GO = go signal. STOP = stop signal. TEMG = time to EMG agonist onset. STOPPING EFFECT = observed effect of the stop process.



varying effect (c.f., a stopped response, a partial response), as well as at all times after EMG onset also to varying effect (c.f., an interrupted response, a full response).

If this mechanism is correct, then we reason that the stopping process continues to act on the go process before motor discharge even after the point of no return has been surpassed. In this case the final ballistic process is not inviolate to further effects of stopping. Thus, we submit that the point of no return is phantom. It marks that point in the control of a voluntary action beyond which the to-be-produced action cannot be prevented, but, at the same time it fails to mark a final ballistic process if the process of control is accessible to further effects of stopping. In this light, the point of no return is of no theoretical significance to the control of voluntary action.

Osman (personal communication) suggested, for our consideration, an alternative mechanism to the one proposed above. If the preparation of a response is delineated to discrete processing stages, as is the case from an information processing view, then it might be that the stop process acts on each stage in punctate fashion with differing degrees of effect, possibly as a result of the openness of each stage to and, perhaps, by the effort invested in, stopping. For instance, a less effective (effortful) stopping response would allow that stage to pass on degraded output to the next stage and so on until the degraded output of the last stage results in a partial response. We have no way of distinguishing between the two mechanisms from the data reported in this study. That motor neurons receive both excitatory projections and inhibitory projections from inter-neurons (Enoka, 1994, p. 136) supports our suggestion of an open control mechanism that competes right up to the point of motor discharge. Notwithstanding, while a partial response lends weight to the argument against a final ballistic process that might precede muscle activity, a final ballistic process cannot be satisfactorily ruled out on this basis.

2.3.5 Some further reflections on the observance of partial responses

The partial responses and the interrupted responses reported in this study show that the stopping process affects the go process both before and after its execution. This is only consistent with reasoned expectation since it is plainly evident that an action can be withheld, if it is caught early enough, or withdrawn as it is unfolding. De Jong et al. (1990) reported no evidence for a braking

effect on the go process from measures of latency, LRP, surface EMG and force, a result that they interpreted in support of the race model (i.e., independent processes). Sub-maximal EMG records in this study detail a weakened go process to arrive at the motor pool as a result of stopping and provide good evidence for De Jong et al.'s (1990) absent braking effects. Importantly, these data have a bearing on the race model as we will see later.

For now, the observance of a braking effect might be squared with De Jong et al.'s (1990, p. 179) assertion that "... even if central inhibition processes do not succeed in preventing central motor outflow, the overt response can be inhibited or interrupted by preventing the transmission of such outflow to peripheral motor structures". For example, partial responses might result from early central motor outflow (or leakage) before their later inhibition (i.e., interruption) at the motor pool. Logan (personal communication) suggested an alternative consideration in that the central stopping mechanism might prevent an action by re-programming it to approximately zero magnitude (in effect reducing its gain). The residual activity would then be allowed to trickle down and manifest as a partial response even though the action, for the most part, would be stopped successfully.

The experimental criterion for what constitutes an action is an important consideration here because participants might, in some cases, consider a partial response as a successfully withheld action. We have no data in this regard since the displacement data, used on-line by the experimenter in the first instance to mark the go latencies and so identify the type of response on each trial (i.e., moved, stopped), were re-analysed after their collection. Nevertheless, partial responses have been identified in each of the later studies presented here, in which participants were explicitly informed beforehand that any change in displacement as recorded in the optical encoder would constitute an action (i.e., an unsuccessfully stopped response) and be enforced as such by the experimenter (McGarry & Franks, in review-a, in review-b). This finding was also repeated in a study in which partial responses were identified solely from the presence of EMGs (McGarry, Inglis & Franks, in review-c). In short, partial responses, as well as interrupted responses, constitute part of the full repertoire of graded responses that are observed as an expected result of sealing the excitatory drive to the motor pool at various times in order to effect stopping of an earlier intended action.

2.4 Summary

This study reports various types of response that were identified from surface EMG records when trying to stop an earlier intended action. For the first time, partial responses as reflected in sub-maximal EMG data at onset were, in some cases, observed and taken to suggest that the stopping process results in weakened synaptic drive to reach the motor pool. These unique observations have important implications on the control of stopping as it pertains to a point of no return (i.e., a final ballistic process). In particular, sub-maximal EMG onsets were taken to suggest that the point of no return is phantom, given that the effects of stopping are seemingly evidenced right up to the point of motor discharge.

3 Experiment II

3.1 Sub-maximal EMG data from onset and their bearing on the control of thought and action

We (McGarry & Franks, 1997) have taken the sub-maximal EMG data observed at onset when trying to stop an otherwise maximal speeded elbow extension to suggest that the point of no return is phantom. It was reasoned that these data indicate a point beyond which some motor activity will be observed at a later time but, importantly, that this point fails to mark a final ballistic process if that process is subject to further effects of stopping. That a final ballistic process might receive sub-maximal input that results in sub-maximal output, however, provides for an alternative mechanism by which the sub-maximal EMG onsets might be produced. Thus, while a final ballistic process is weakened by the observance of sub-maximal EMG data, it cannot be satisfactorily ruled out at present.

In our judgement, the sub-maximal EMG data not only speak against a final ballistic process, but they also speak against the race model's account of stopping in an important way. Recall from the race model that the winner of the race specifies the outcome. If the go process wins the race then an action is initiated (i.e., a go response is observed) and if the stop process wins the race then the earlier intended action is withheld (i.e., a stopped response is observed). We (McGarry & Franks, 1997) identified three types of go response observed in the presence of a stop signal; full responses, interrupted responses and partial responses. We suggested that the margin of victory between each process crossing the finish line can explain each of these types of go response. Thus, a large go-stop margin yields full responses and a small go-stop margin yields interrupted responses, the margin of victory further specifying whether the responses are interrupted early or late in the time course of their usual production. Of note is that the go-stop winning margin cannot explain the partial responses because interrupted responses and or full responses would be predicted on these occasions. Instead, we suggested that the stop-go margin explains the partial responses and the stopped responses just as the go-stop margin explains the interrupted responses and the full responses. Thus, a small stop-go margin yields partial responses and a large stop-go margin yields stopped responses. These latency relations are detailed in Figure 2.5.

The race model specifies that the outcome probability is the result of a race between the go process and the stop process. Thus, partial responses might be likened to the go process winning the race but crossing the finish line at much reduced velocities. The race model fails to explain this observance. It follows that a theory of control for stopping a voluntary action must extend beyond the race model if the EMG data that describe the effects of stopping on an action are to be explained.

To reiterate, while the race model well explains the relation between the go latencies, the stop latencies, the SOA and the outcome probabilities, the relations as expressed in EMG onsets that describe the same stopping effects on a voluntary action require further explanation. The aim of this study is to suggest a mechanism of control that is consistent with physiology that can explain both sets of data. Specifically, we posit that the control process is the result of excitatory-inhibitory interaction at the level of individual neurons whose net effect is observed at the motor pool. The key objection to this position would be the expectation of longer go latencies in the presence of a stopping process in contrast to the factual observance of shorter go latencies (c.f., Figure 1.1). We present a computer analysis that demonstrates that this supposition need not hold.

3.2 Method

The same experimental task, apparatus, procedure and protocol and associate measures as in Experiment I were used with the following exceptions.

3.2.1 Participants

Eight (5 males, 3 females) right hand dominant participants ranging in age from approximately 20 years through approximately 35 years were recruited in this study. One participant partook in Experiment I. Testing occurred in a single session. Each participant received financial remuneration on completion of the session (see later).

3.2.2 Apparatus

The apparatus was adjusted so as to reduce the possibility of unwanted co-contraction in the agonist-antagonist muscles that might result from gripping a vertical handle at the end of the manipulandum. This was achieved by placing the right hand of the participant prone on a custom-

designed hand rest with the middle finger isolated between two upright dowels. The right hand was then strapped to the hand rest so as to restrict finger and thumb movement.

3.2.3 Procedure

The same experimental procedure as that for Experiment I was used in this study.

3.2.4 Protocol

3.2.4.1 Experimental tasks

Two experimental tasks - the go-only task (40 trials) and the forced task (120 trials) - were given to each participant. Stop signals and catch trials were once again presented at probabilities of .333 and .100 respectively. SOAs of -50 ms, 0 ms, 50 ms and 100 ms were used in the forced task with equal probabilities.

3.2.4.2 Pay-off schedule

In the go-only task, a \$0.10 reward was provided for every millisecond that the participant's $\overline{X}_{\text{go-only}}$ ($n = 40$) fell below 300 ms. This pay-off was undertaken in order to provide an incentive to react as fast as possible. In the forced task, a \$0.25 reward for a correct response and a -\$0.25 penalty for an incorrect response, as well as a possible \$0.25 bonus, was added on each trial to the participant's balance. The bonus was awarded on each trial that the updated $\overline{X}_{\text{forced}}$ fell below a target latency equal to that for a slow go latency (for further detail see section 2.2.4.2 Pay-off schedule). The participant was paid his (her) final balance at the end of the testing session to a \$5 minimum. Each participant was unaware of the \$5 minimum payment at the time.

3.3 Results and Discussion

3.3.1 Data analysis

The time to the onset of triceps EMG and the onset of biceps EMG were analysed post hoc by the experimenter for each trial, so yielding measures of latency for triceps EMG (TEMG) and biceps EMG (BEMG). TEMG reflected the latency from the onset of the go signal to the leading edge of triceps EMG activity above baseline. BEMG reflected the latency from the onset of the go signal to that burst of biceps activity interpreted to provide the primary braking force rather than the leading

edge of biceps activity per se. The latency between the onset of the triceps and the onset of the biceps (BTD) was obtained for each trial from the time interval between TEMG and BEMG.

Each trial was analysed post hoc using a computer algorithm to obtain its extension displacement, that is the difference in displacement between the minima (preceding the maxima) and the maxima. Those responses, in the presence of a stop signal, that yielded no reliable EMG characteristics and extension displacement were classified as stopped responses. The extension displacement for each no-stop response was then used to segregate the full responses from the non-full responses (i.e., interrupted responses and partial responses) for each participant as follows.

Those go responses in the presence of a stop signal whose extension displacement was equal to or greater than one $SD_{no\text{-}stop}$ below $\overline{X}_{no\text{-}stop}$ were classified as full responses (i.e., $trial_{full} \geq \overline{X}_{no\text{-}stop} - SD_{no\text{-}stop}$). (The no-stop responses are those go responses observed in the stopping task in the absence of a stop signal.) Thus, full responses are indistinct in their extension displacements from the no-stop responses since these extension displacements would be expected to sample from the no-stop distribution with .84 probability. Those go responses in the presence of a stop signal whose extension displacement was equal or less than two and a third $SD_{no\text{-}stop}$s below $\overline{X}_{no\text{-}stop}$ were classified as non-full responses (i.e., $trial_{non\text{-}full} \leq \overline{X}_{no\text{-}stop} - 2.33\ SD_{no\text{-}stop}$). Non-full responses are distinct in their extension displacement from the no-stop responses since these extension displacements would be expected to sample from the no-stop distribution with .01 probability.

The non-full responses were then classified further as either interrupted responses or partial responses from a visual analysis of their untreated triceps EMGs and rectified-filtered (20 Hz) triceps EMGs. In essence, any non-full responses that were not classified as partial responses were classified as interrupted responses by exclusion. The mean extension displacements of the various responses, collapsed across participants, affirm the validity of these procedures as reported in the following parentheses. ($N_{no\text{-}stop} = 8$, $\overline{X}_{no\text{-}stop} = 45.18$, $SD_{no\text{-}stop} = 13.51$; $N_{full} = 8$, $\overline{X}_{full} = 44.56$, $SD_{full} = 13.28$; $N_{interrupted} = 7$, $\overline{X}_{interrupted} = 17.47$, $SD_{interrupted} = 6.68$; $N_{partial} = 6$, $\overline{X}_{partial} = 1.47$, $SD_{partial} = 1.20$. N = number of participants. $\overline{X}$ = mean. SD = standard deviation. Units are degrees.)

3.3.2 On the use of EMG onsets as an index of synaptic drive to reach the motor pool

We used EMG onsets to delineate partial responses from interrupted responses. Surface EMG records constitute a non-faithful interference pattern of the spatial-temporal sum of the motor discharges, not least because of the on-going change in displacement of the underlying muscle fibres from the detecting electrodes as a result of muscle shortening or lengthening. In fact, we use EMG onsets as an index of early synaptic drive to reach the motor pools without regard for a change in muscle length because we analyse the EMG data in the short time window t before any extension occurs ($t \leq 30$ ms). (See Section 4.2.7 Quantitative measures of the rise in EMG from onset [Q_{10}, Q_{20} and Q_{30}], for further detail). For example, the latency from the onset of motor discharge (i.e., EMG) to the onset of displacement (extension) is typically in the order of 30 ms - 50 ms which is outside of the window that we used to segregate visually the partial responses from the interrupted responses in this study.

3.3.3 Intra-rater and inter-rater reliability analyses of the latencies of triceps EMG onset (TEMG) and biceps EMG onset (BEMG)

Intra-rater and inter-rater reliability analyses of the TEMG and the BEMG markers was performed post hoc on those trials (n = 138) that comprised the full responses and the interrupted responses as observed across participants (see Table 3.1). These trials were re-analysed blind by the same observer and by an independent observer at a later time. Pearson product moment correlations (r) showed the data analyses to be reliable (r_{af} =.99, r_{ai} =.99) and objective (r_{bf} =.98, r_{bi} =.97). a, b, f and i denote intra-rater, inter-rater, full responses and interrupted responses respectively.

3.3.4 Latency of triceps EMG onset (TEMG) as a function of the time interval from presentation of the go signal and the stop signal (SOA)

The race model specifies that faster go latencies are observed with lower probabilities at shorter SOAs. We analysed TEMG latencies as a function of SOA but excluded those trials observed at SOA -50 ms on account that the participants sometimes treated incorrectly the stop signal as the go signal and reacted accordingly. (This is analogous to a sprinter waiting for the starter's gun and reacting in

Table 3.1. Descriptive and statistical analyses for the latencies to triceps EMG onset (TEMG) and biceps EMG onset (BEMG) (i.e., BTD).

		Response Type							
		No-Stop		Full		Interrupted		ANOVA	
	N	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD	F	p
BTD	7	105.4	23.7	102.5	25.8	80.1	24.6	14.568	.001

		Response Type			
		No-Stop	Full	Interrupted	Partial
BTD	No-Stop	-	2.9	25.4*	
	Full		-	22.4*	
	Interrupted			-	
	Partial				-

Note. Units of measurement are milliseconds. * $p < .01$. N = number of participants. $\overline{X}$ = mean. SD = standard deviation.

error to another signal such as a muscle twitch from a competitor.) This situation, is further confounded by latencies being faster for acoustic stimuli (c.f., the stop signal) than for visual stimuli (c.f., the go signal). Excluding SOA -50 ms, the typical data pattern of shorter go latencies at shorter SOAs as described in the race model was found to hold. The TEMG data, collapsed across participants, are reported in the following parentheses. ($N = 8$. $\overline{X}_{No\text{-}stop} = 197.2$ ms, $SD_{No\text{-}stop} = 27.8$ ms; $\overline{X}_{SOA\ 0\ ms} = 131.5$ ms, $SD_{SOA\ 0\ ms} = 29.4$ ms; $\overline{X}_{SOA\ 50\ ms} = 159.8$ ms, $SD_{SOA\ 50\ ms} = 25.4$ ms; $\overline{X}_{SOA\ 100\ ms} = 186.3$ ms, $SD_{SOA\ 100\ ms} = 23.9$ ms.)

3.3.5 Exclusion of partial responses from statistical analysis

The data were subjected to statistical analysis following the segregation of response types. Unfortunately, only a few partial responses were observed as in our earlier study (McGarry & Franks, 1997) and their infrequent occurrences across participants precluded their inclusion in the statistical analysis (see below). The few observances bear witness to the experimental problem of inducing unwitting partial responses when trying to stop a maximal speeded action. That said, the infrequency with which partial responses were observed, given the experimental protocol, in no way speaks against the authenticity of their observation.

3.3.6 Effect of stopping on the timing of neural sequences

BTD was analysed by response type for statistical differences using a one-way (response type) repeated measures ANOVA (see Table 3.1). Participant 5 was excluded from the analysis because of the absence of interrupted responses. Since BTD is the time interval between TEMG and BEMG, BTD was analysed using the data from each SOA, including SOA -50 ms. This is because faster TEMGs in the SOA -50 ms that arise from an incorrect reaction to the stop signal are not expected to compromise the effects of stopping on the timing of the neural sequences as indexed in BTD.

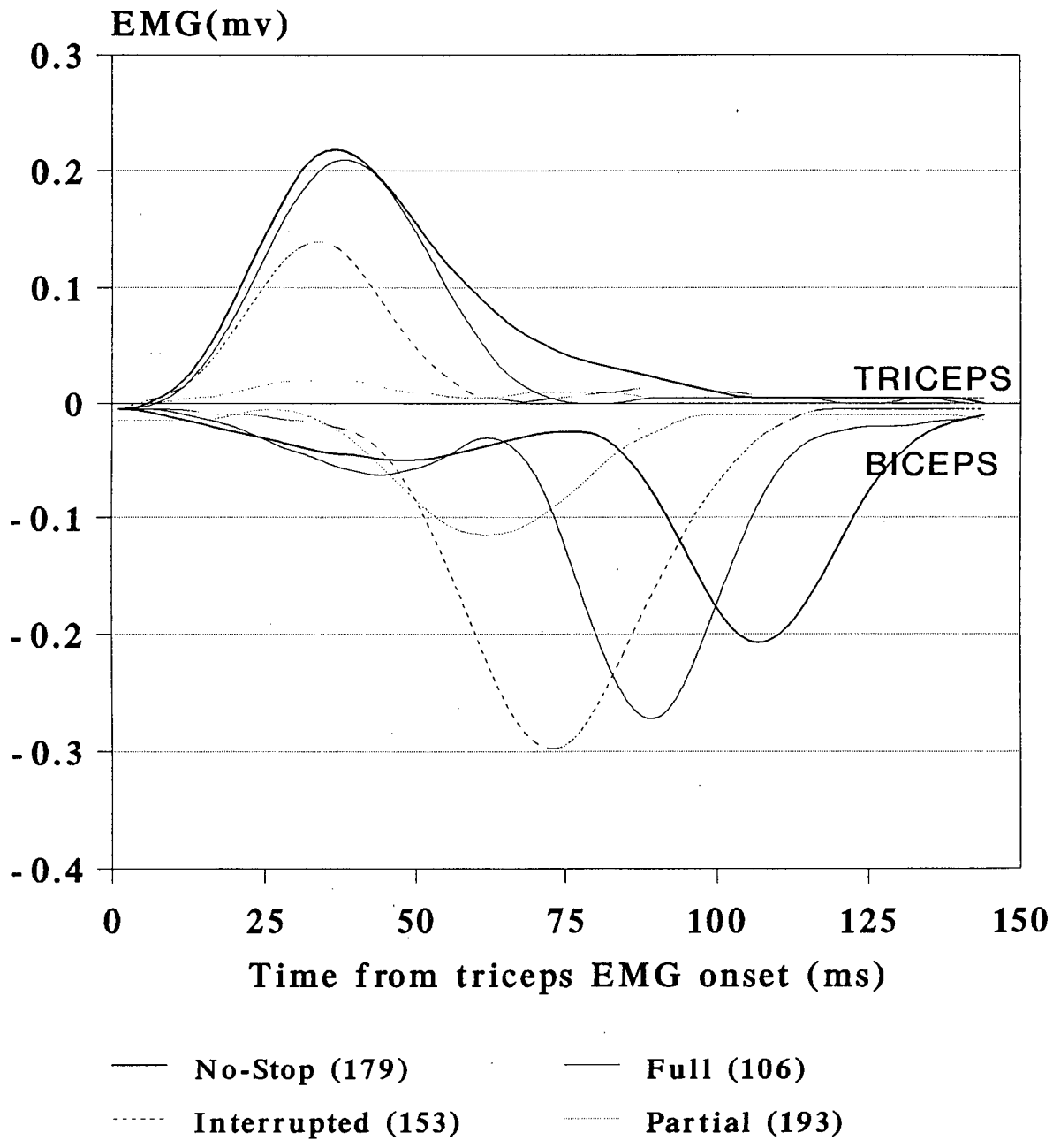
Table 3.1 details a statistical difference between response types for BTD, $F(2,12) = 14.568$, $p = .001$. Post hoc analysis using the studentized range test (Howell, 1997) revealed that the difference exists between the no-stop responses and the interrupted responses ($p < .01$) and, also, between the full responses and the interrupted responses ($p < .01$). No difference was found between the no-stop

responses and the full responses ($p > .05$). That the interrupted responses would be expected to sample from the slower portion of the underlying no-stop distribution than would the full responses (see Figure 2.5) might explain why a difference in BTD exists between the interrupted responses and the full responses. Longer TEMG latencies for the interrupted responses than for the full responses might shorten BTD if the BEMG latencies are not lengthened to the same extent as the TEMG latencies. In fact, the prediction that the interrupted responses have longer TEMG latencies than the full responses is not supported from a two-tailed paired t-test, $t(6) = .698$, $p = .511$. These results affirm that the shorter BTD latencies for the interrupted responses are not an artifact of longer TEMG latencies for those same responses.

That the biceps are advanced in time in relation to the triceps for the interrupted responses as opposed to the full responses suggests that the stopping process not only acts to suppress neural excitation, as evidenced in the reduced EMG onsets that typify the partial responses, but that it also acts to change the timing of the neural sequencing of the agonist-antagonist pair in order to counter unwanted agonist activity. That the biceps might be advanced in time is not surprising, given that the extension displacement is at least one and a third $SD_{no\text{-}stop}$s of a response less for an interrupted response than it is for a full response. (This is known from the procedure, described earlier, that used extension displacement to segregate full responses from non-full responses.) We interpret the advance in BEMG in relation to the TEMG (i.e., BTD), in the order of 20 ms (Table 3.1), to constitute a meaningful change in the timing of the neural sequences to the agonist-antagonist muscles.

Figure 3.1 details an example of the rectified-filtered (20 Hz) EMG data synchronised in time to the onset of triceps EMG (i.e., TEMG) for a no-stop, full, interrupted and partial response from Participant 3. The time line begins at TEMG (i.e., triceps EMG onset) and the first triceps-biceps(inverted) EMG burst only is shown for ease of comparison. The following pattern of data is noted. First, the EMG rise from onset, and the time line of its continuance, delineates each type of response as first identified by McGarry and Franks (1997). Second, the onset of antagonist (biceps) braking activity, as indicated in the steep rise in EMG, is advanced in time in relation to the agonist (triceps) from the no-stop response through to the interrupted response. (Recall that a post hoc

Figure 3.1 Example of a no-stop, full, interrupted and partial response from Participant 3. Linear EMG envelopes for triceps and biceps (inverted) are full wave rectified and filtered at 20 Hz. Each response is time-synchronised to the onset of triceps EMG (TEMG). (TEMG thus assumes a zero reference value by convention.) The first EMG burst of triceps and biceps (inverted) are shown only for ease of comparison.



statistical analysis of BTD across participants showed that the no-stop responses were not significantly different from the full responses, but that the full responses were significantly different from the interrupted responses. See Table 3.1). Third, the data from Figure 3.1 raises the possibility that not only is the neural sequencing of the antagonist (biceps) advanced in time in relation to the agonist (triceps), but that the magnitude of the antagonist might likewise be facilitated in relation to the agonist as a result of stopping. This is indicated in the increasing rectified-filtered (20 Hz) peak EMGs for the antagonist (biceps) in relation to the agonist (triceps), once again from the no-stop response through to the interrupted response. Indeed, in this particular example (Figure 3.1) the partial response, uncharacteristically, almost resulted in flexion rather than extension at the onset of displacement (not shown) as a result of the different contributions of the agonist-antagonist pair.

Increased antagonist (biceps) activity as a result of stopping, as indicated above, is confirmed in an independent study by Kudo and Ohtsuki (1998). These authors reported, also from an analysis of stopping an elbow extension at various times, a significant increase in antagonist (biceps) EMG activity as a counter result of unwanted agonist (triceps) EMG. Following Georgopolous et al.'s (1981) reasoning for a continuous process of control from the observation of graded kinematics in double-step tasks, Kudo and Ohtsuki (1998, p. 28) concluded that "... the execution of rapid movement is not an unrevisable ballistic process but a continuous and real time process that can be modified at any time". (See also Becker & Jurgens [1979] and Henis & Flash [1995] for further consideration as to how the data from double-step studies might be explained from the vector sum of two discrete processes.) This is consistent with our earlier inference from sub-maximal EMG data of an on-line process of control that is open to stopping at all times (McGarry & Franks, 1996, 1997).

In sum, the results from this study indicate that the effect of stopping extends beyond the reduction of neural drive to change the timing (Table 3.1, Figure 3.1), as well as possibly the magnitude (Figure 3.1, Kudo & Ohtsuki, 1998), of the relative synaptic contributions to the agonist and antagonist motor pools. These data are suggestive of a fast and flexible system of control that changes as the sequence of events unfolds to meet new intentions.

3.3.7 Further observations on partial responses

Figure 3.2 (upper panel) and Figure 3.3 (upper panel) each detail an example of an isolated EMG spike train that was observed when stopping a maximal speeded action from Participant 8 and Participant 1, respectively. Figure 3.2 (lower panel) and Figure 3.3 (lower panel) detail the same spike train as that in their respective upper panels with the relevant part of their time axis stretched. (The lower panels are stretched from a 1500 ms range to a 200 ms range for Figure 3.2 and a 300 ms range for Figure 3.3.) We rule out signal noise as the source of these EMG spikes on three counts. First, the spikes were observed above baseline noise. Second, there was an accompanying elbow extension displacement (not shown). Third, in both examples the observations occurred in the presence of a stop signal, while no like isolated spikes were observed in the absence of a stop signal. These spikes undoubtedly constitute a sub-maximal, indeed minimal, motor action and suggest single motor unit discharge within the detection vicinity of the surface electrode. (The surface electrode represents a sub-population only of the motor activity within an individual muscle.) These data provide good support for our earlier inference that the stopping process acts on the go process right up to single motor discharge (McGarry & Franks, 1997).

The inter-spike intervals of 73 ms and 73 ms (Figure 3.2) yielded a firing rate of about 14 Hz. Interestingly, Le Bozec and Maton (1987) reported an average contraction time of 68 ms ± 9 ms for the lateral head of the triceps brachii, with minimal firing rates between 8 Hz and 11 Hz and maximal firing rates between 14 Hz and 16 Hz.[8] Given the similar order of discharge frequency reported in this experiment, we interpret the inter-spike intervals of 73 ms as being within the expected limits for single motor unit discharges that might occur in the lateral head of the triceps brachii. This result, albeit from a limited view of the muscle, is consistent with the position that the control of a maximal speeded action can be reduced to single motor discharge as a consequence of

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[8] In Le Bozec and Maton's (1987) study, participants were required to maintain a constant isometric torque. The level of isometric torque was varied experimentally up to 30 % of maximal voluntary isometric contraction.

Figure 3.2 Example (rescaled) of likely single motor neuron discharge from the triceps EMG from Participant 8. The triceps EMG spike trains are presented on the usual time line (upper panel) and an extended time line (lower panel) when stopping a maximal speeded elbow extension. The inter-spike intervals between the triceps EMG spikes are 73 ms and 73 ms respectively.

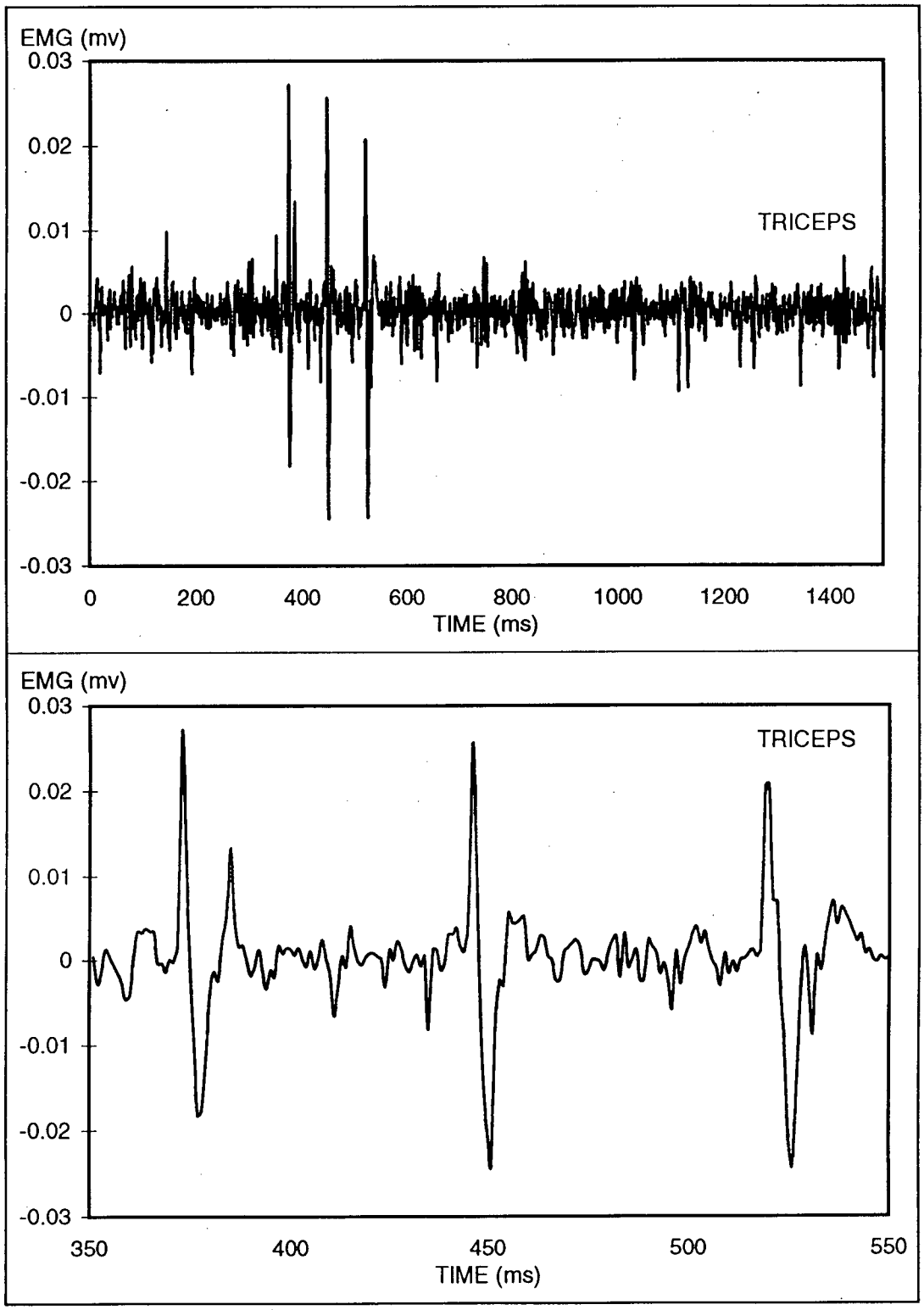
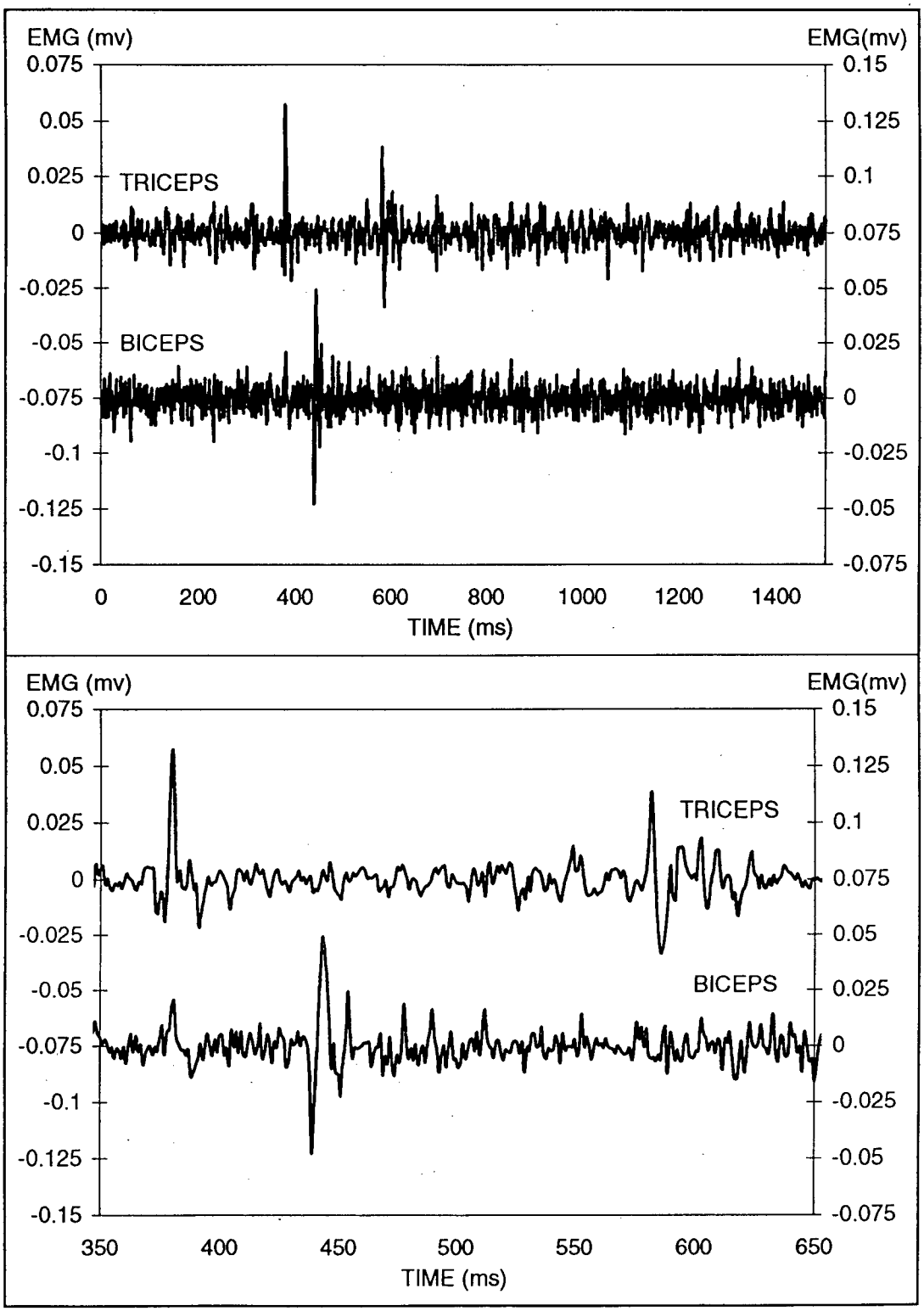


Figure 3.3 Example (rescaled) of likely single motor neuron discharge from the triceps EMG (left y-axis) and the biceps EMG (right y-axis) from Participant 1. The triceps EMG spikes and the biceps EMG spikes are presented on the usual time line (upper panel) and on an extended time line (lower panel) when stopping a maximal speeded elbow extension. The inter-spike intervals between the first triceps spike and the only biceps spike and between the only biceps spike and the second triceps spike are 65 ms and 143 ms respectively.



stopping. This finding affirms our earlier inference that partial responses are a discrete classification of a continuum of response reductions from zero (i.e., full) through maximum (i.e., stopped).

Figure 3.3 shows that the stopping process can also act in like fashion on both the agonist (triceps) and antagonist (biceps) motor pools. This is observed in the reduced neural gain in the tri-phasic EMGs that are responsible for the motor action, in contrast to the relative action of the stopping process on these same motor pools as reported in Figure 3.1. In this trial (Figure 3.3), the neural activity was abated for the most part while the sequencing order of the neural command to the respective motor pools was preserved. Thus, it seems that the lead excitation (see later) that converges on the respective agonist-antagonist motor pools escaped the stopping process while further excitation of those same pools was prevented.

3.3.8 Excitatory-inhibitory interaction versus a race between excitatory (go) and inhibitory (stop) processes

Partial responses, as delineated from EMG onset, indicate much reduced synaptic drive to reach the motor pools. We submit an account of excitatory-inhibitory interaction in an effort to explain these results. We proceed in accord with the physical principles that govern neural behaviour, as outlined in brief by Anderson (1988, p. xv) in his introduction to generic connectionist models:

> "There are very many neurons, or nerve cells, in the human brain, at least ten billion. Each neuron receives inputs from other cells, integrates the inputs and generates an output, which it then sends to other neurons or, in some cases, to effector organs such as muscles or glands. Neurons receive inputs from other neurons by way of specialized structures called synapses and send outputs to other neurons by way of output lines called axons. A single neuron can receive on the order of hundreds or thousands of input lines and may send its output to a similar number of other neurons. A neuron is a complex electrochemical device that contains a continuous internal potential called a membrane potential, and, when the membrane potential exceeds a threshold, the neuron can propagate an all-or-none action potential for long distances down its axon to other neurons. Synapses come in a number of different forms, but two basic varieties

are of particular note: excitatory synapses, which make it more likely that the neuron receiving them will fire action potentials, and inhibitory synapses, which make the neuron receiving them less likely to fire action potentials."

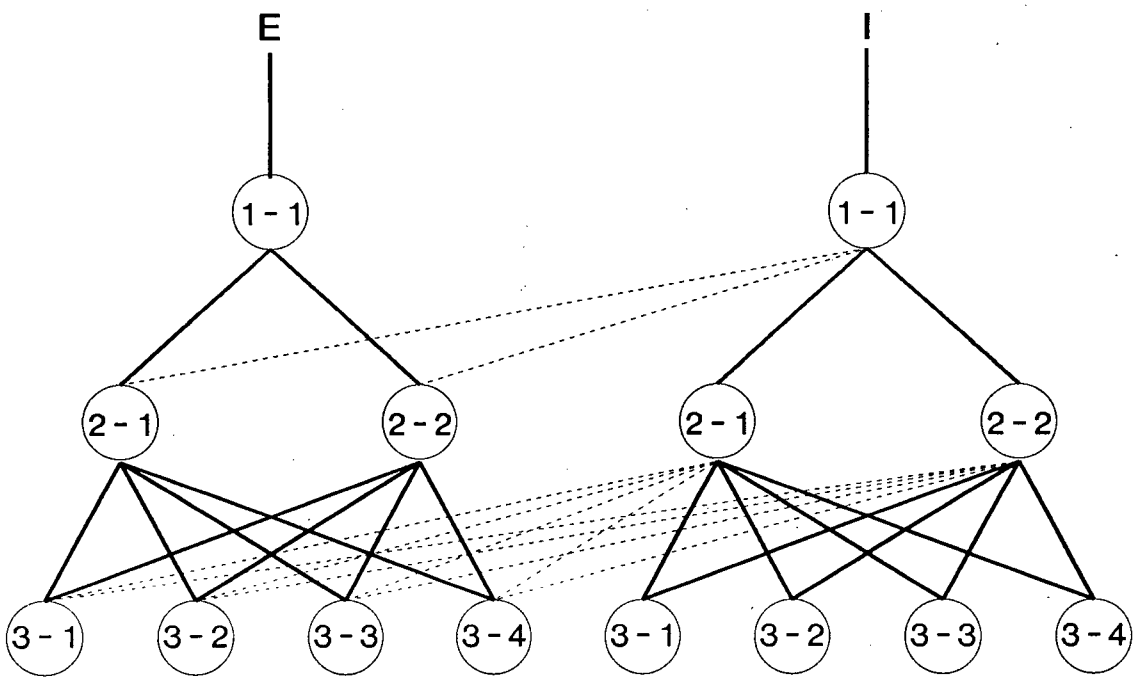
The discrete transition in random time between two output states (e.g., 0, 1) is reported to yield a good approximation to neural properties (Hopfield, 1984) Thus, we posit control to be effected through an array of neurons that propagate in stochastic fashion before their convergence on the motor pool. Figure 3.4 details a binary hierarchy ($\underline{b} = 2$) of only three levels, or generations ($\underline{g} = 3$), for ease of presentation. The hierarchy constitutes two families whose first generation members are sibling related. The excitation (E) family reflects the go process and the inhibition (I) family reflects the stopping process. Solid lines reflect excitatory connections within a family and dashed lines reflect inhibitory connections that project in uni-lateral fashion only from the I-family to the E-family.

Each neuron (or cell), hereafter referenced by membership to its family ($\underline{f}$), generation ($\underline{g}$) and sibling order ($\underline{s}$), is limited, for simplicity, to a single synapse (input) and to a single axon (output) that connects each parent to each child (see Figure 3.4). Hence, a parent can excite only its children and, likewise, a parent can only be excited by its own parents. In addition, an uncle (or aunt) in the I-family can inhibit only its nephews (or nieces) in the E-family. Each cell is considered initially to be in a neutral, or vacant, state (0) until it is either excited (1) by one of its parents or inhibited (-1) by one of its uncles (or aunts) in random time.

Each family excites randomly in linear fashion from parent to child. In fact, the I-family performs double duty since, as parents, they excite their children and, as uncles (or aunts), they inhibit their nephews (or nieces). This is achieved by duplicating the I-excitation pulse (1) that is sent to each child ($I : \underline{x} - \underline{y}$) in random time as an I-inhibition pulse (-1) to each like nephew (or niece) ($E : \underline{x} - \underline{y}$) in the same random time. Thus, the I-family exerts increasing inhibitory influence on the E-family as the process of inhibition propagates the hierarchy.

The hierarchy proceeds according to the following rule: (a) a child in a neutral state can be excited by any of its parents or inhibited by any of its uncles (or aunts); a child in an excited state can

Figure 3.4 Hypothesized hierarchical structure of control for producing and stopping a speeded voluntary action. The structure contains only three levels for ease of presentation. Excitation spreads in the excitation (E) and inhibition (I) families by way of excitatory connections (solid lines). Inhibition from the I-family is exerted on to the E-family by way of uni-lateral inhibitory projections (dashed lines). Increased excitation in the I-family thus exerts increased inhibitory influence on the E-family at each generation. The processes of excitation and inhibition propagate the structure in random fashion. The excitation-inhibition that acts on the motor neurons (last generation, E-family) determines the motor discharge pattern and thus the various types of EMG onsets observed in this study. Note. From T. McGarry and I.M. Franks (in review-a).



be inhibited by any of its uncles (or aunts); and (c) a child in an inhibited state can not be excited by any of its parents. This rule allows for the stopping of the excitation process at all times. This is achieved when all the next to-be-excited neurons within a generation are inhibited.

Excitation pulses and inhibition pulses were generated in random time using the pseudo-random number generator of a personal computer. The algorithm draws from a linear distribution such that any real number from 0 through 1 is generated on any occasion. We scaled the random real number to reflect a different pulse distribution for the E-family and the I-family. Specifically, we used lower bounds of 30 and 25 and ranges of 50 and 40 to generate random integers from 30 through 80 for the excitation pulses (i.e., time-to-excite) and 25 through 65 for the inhibition pulses (i.e., time-to-inhibit) respectively.

The behaviour of the model (Figure 3.4) might best be understood as follows. The go signal (E) sends an E-excitation pulse (1) to the first parent (E : 1 - 1). On its receipt, the parent discharges an E-excitation pulse in random time (see above) to each child in the next generation (E : 2 - 1, E : 2 - 2). Likewise, on its receipt, each child (E : 2 - 1, E : 2 - 2), itself a parent, discharges an E-excitation pulse in random time to each child in the next generation (E : 3 - 1 through E : 3 - 4) and so on. Thus, the process of E-excitation self propagates in stochastic fashion as it traverses the hierarchy before arriving at the motor pool (i.e., the last generation, E : g - 1 through E : g - s). The first motor discharge necessarily constitutes some type of go response. If a green light is imaged to trigger in the body of each cell to receive (or discharge) an E-excitation pulse, then the random checkered sequence of green lights in the E-family reflects the activity of the go process. The latency from the go signal to the first motor discharge (Go-EMG, see later) marks the EMG onset (or TEMG).

The process of inhibition (i.e., I-excitation and I-inhibition, see Figure 3.4) is patterned in like manner. The stop signal (I) sends an I-excitation pulse (1) to the first parent (I : 1 - 1). On its receipt, the parent discharges in random time (see above) to the next generation; (a) an I-excitation (1) pulse to each child (I : 2 - 1, I : 2 - 2), and (b) an I-inhibition pulse (-1) to each nephew (or niece) (E : 2 - 1, E : 2 - 2). Likewise, on the receipt of an I-excitation pulse, each child (I : 2 - 1, I : 2 - 2), itself a parent as well as an uncle (or aunt), discharges in random time to the next generation; (a) an I-excitation (1)

pulse to each child (I : 3 - 1 through I : 3 - 4), and (b) an I-inhibition pulse (-1) to each nephew (or niece) (E : 3 - 1 through E : 3 - 4) and so on. If a red light is imaged to trigger in the body of each cell to receive (or discharge) an I-excitation pulse, then the random checkered sequence of red lights in the I-family reflects the activity of the stop process. The same sequence of I-excitation in the I-family is observed as I-inhibition in the E-family. The latency from the stop signal to the first motor discharge (Stop-EMG, see later) marks the time that the stopping process begins to take effect at the motor pool (i.e., last generation, I : g - 1 through I : g - s, or, alternatively, E : g - 1 through E : g - s).

If a go signal is presented a brief time before a stop signal, as specified in the SOA, then green lights followed shortly thereafter by red lights each begin to propagate the E-family in random time. The rule of operation might now be re-stated; a neuron in the E-family lights green by virtue of its parents and red by virtue of its uncles (aunts) and, importantly, the red state is terminal. In neurophysiology, this is not the case and the inhibitory state is transient not terminal since an inhibited neuron can be later excited. Notwithstanding, it is the sequencing of green lights at the motor pool (E : g - 1 through E : g - s) that determine the distinct types of responses reported in this study that result from stopping an earlier intended action at various times.

For simplicity, we delineate the various types of response from the motor discharge histories as follows: full, if the first motor neuron to discharge is excited and all of the other motor neurons in the motor pool are subsequently excited; interrupted, if the first motor neuron to discharge is excited and some of the other motor neurons in the motor pool are inhibited; partial, if the first motor neuron to discharge is inhibited and some of the other motor neurons in the motor pool are excited; and stopped, if the first motor neuron to discharge is inhibited and all of the other motor neurons in the motor pool are subsequently inhibited. (Note. For the partial responses, the stopping process wins the race to the motor pool but fails to prevent some excitatory discharge a short time thereafter in one or more of the remaining vacant motor neurons in the motor pool. c.f., Figure 2.5.)

The results from the model are detailed in Table 3.2. The simulation consisted of r runs (r = 1000) each beginning with a different starting seed in the computer generated pseudo-random number

Table 3.2. The probability of responding as a function of the time between presentation of the go signal and the stop signal (SOA) for the neural architecture (Figure 3.4, 9 levels) and the race model (Logan & Cowan, 1984).

			SOA							
	GO	STOP	10	20	30	40	50	60	70	80
NO-STOP										
n	1000		26	87	234	416	637	805	915	983
$\overline{X}_{\text{Go-EMG}}$	271.66		252.31	255.59	258.97	261.97	264.91	267.54	269.51	271.14
$SD_{\text{Go-EMG}}$	13.78		6.38	6.75	8.25	9.20	10.17	11.29	12.30	13.40
$p\{\overline{X}_{\text{Go-EMG}}\}$	1.00		0.58	0.63	0.69	0.79	0.88	0.93	0.95	0.98
$p\{SD_{\text{Go-EMG}}\}$	0.00		0.42	0.41	0.41	0.36	0.29	0.22	0.19	0.12
FULL										
n			10	32	111	249	477	682	825	938
$\overline{X}_{\text{Go-EMG}}$			249.70	253.09	256.28	259.21	262.45	265.44	267.82	270.08
$SD_{\text{Go-EMG}}$			4.81	5.48	7.06	8.09	9.01	10.37	11.51	12.68
$p\{\overline{X}_{\text{Go-EMG}}\}$			1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
$p\{SD_{\text{Go-EMG}}\}$			-	-	-	-	-	-	-	-
INTERRUPTED										
n			7	31	64	94	104	82	55	31
$\overline{X}_{\text{Go-EMG}}$			253.57	256.39	259.89	264.78	271.25	279.00	284.96	291.77
$SD_{\text{Go-EMG}}$			5.41	7.50	8.09	8.58	10.46	8.90	8.30	6.99
$\overline{X}_{\text{Stop-EMG}}$			254.86	258.77	262.36	267.60	273.92	281.67	287.58	294.65
$SD_{\text{Stop-EMG}}$			5.30	7.05	8.13	8.29	10.22	9.19	7.96	7.24
$p\{\overline{X}_{\text{Go-EMG}}\}$			0.64	0.66	0.73	0.79	0.73	0.76	0.77	0.81
$p\{SD_{\text{Go-EMG}}\}$			0.18	0.31	0.26	0.24	0.25	0.26	0.25	0.24
PARTIAL										
n			9	24	59	73	56	41	35	14
$\overline{X}_{\text{Go-EMG}}$			254.22	257.88	263.02	267.74	274.07	279.59	285.17	296.86
$SD_{\text{Go-EMG}}$			8.07	6.46	8.73	9.82	8.67	8.84	6.16	7.37
$\overline{X}_{\text{Stop-EMG}}$			252.67	256.13	261.34	266.22	272.63	278.41	284.11	295.57
$SD_{\text{Stop-EMG}}$			8.40	6.58	8.59	9.84	8.55	8.78	5.97	7.23
$p\{\overline{X}_{\text{Go-EMG}}\}$			0.06	0.10	0.07	0.08	0.08	0.11	0.09	0.10
$p\{SD_{\text{Go-EMG}}\}$			0.06	0.13	0.11	0.09	0.10	0.12	0.10	0.13
STOP										
n		1000	974	913	766	584	363	195	85	17
$\overline{X}_{\text{Stop-EMG}}$		224.50	224.33	223.46	221.80	219.75	217.64	215.96	214.39	209.29
$SD_{\text{Stop-EMG}}$		10.95	10.54	9.83	9.40	9.02	8.38	8.06	7.32	5.21
$p\{\overline{X}_{\text{Go-EMG}}\}$		-	-	-	-	-	-	-	-	-
$p\{SD_{\text{Go-EMG}}\}$		-	-	-	-	-	-	-	-	-
p{interact}			.026	.087	.234	.416	.637	.805	.915	.983
p{race model}			.004	.024	.106	.302	.582	.824	.951	.991

stream. Each process was allowed to traverse the hierarchy ($\underline{b}$ = 2, $\underline{g}$ = 9) in the absence of the other process in order to determine the underlying latency distributions of the go process and the stop process. The resultant distributions ($\underline{n}$ = 1000) were $\overline{X}_{\text{Go-EMG}}$ = 271.66, $SD_{\text{Go-EMG}}$ = 13.78, $\overline{X}_{\text{Stop-EMG}}$ = 224.50 and $SD_{\text{Stop-EMG}}$ = 10.95. (Units of time are arbitrary.) These distributions allowed for the outcome probabilities for given SOAs to be predicted using the race model as provided in the following example for SOA 40. We assumed zero (no) variance in the stopping process in the calculation for reasons of simplicity.

The stop mean ($\overline{X}_{\text{Stop-EMG}}$ = 224.50) intersects the go distribution at 264.50 at SOA 40 (i.e., SOA plus $\overline{X}_{\text{Stop-EMG}}$). This intersection is 7.16 or .52 $SD_{\text{Go-EMG}}$ s negative of the $\overline{X}_{\text{Go-EMG}}$ (271.66). If the go distribution is presumed normal, then the probability from the z-table that the go latencies will win the race at SOA 40 is .302 (see p{race model}, Table 3.2). In contrast, the probability of motor discharge from the model is .416 (see p{interact}, Table 3.2). This value was obtained from 416 go responses being produced from 1000 runs at SOA 40. The 416 go responses comprised 249 full responses, 94 interrupted responses and 73 partial responses as identified from their motor discharge patterns. The probabilities of a go response as a function of SOA, as predicted from the race model and as observed from the computer model, were analysed for differences using a paired t-test following Fisher's z transformation. The results yielded no significant difference between each model's predictions, $\underline{t}(7)$ = .442, $\underline{p}$ = .672.

Importantly, the same latency relations that the race model predicts, that of faster go latencies at reduced probabilities for shorter SOAs, are produced by the computer model also (see Table 3.2). Faster go latencies at shorter SOAs were observed for each type of go response, that is, for the full, interrupted and partial responses. The reverse pattern for the stopped response was also observed, namely that of shorter stop latencies for longer SOAs. This is because, as for the go process at shorter SOAs, the stop process must sample from the faster portion of its distribution at longer SOAs if it is to win the race (c.f., Section 1.7 On the independence of the go process and the stop process.) Recall that the stop process must not only win the race, as defined by the first motor

neuron to light red instead of green, but that it must proceed thereafter to inhibit all the available motor neurons before their excitation from the go process (c.f. Figure 2.5D and E). This is because the go process might still be active after losing the race to the motor pool, even though much of its neural impetus will have been reduced from earlier stopping effects.

The means ($p\{\overline{X}_{Go\text{-}EMG}\}$) and standard deviations ($p\{SD_{Go\text{-}EMG}\}$) of the proportions of motor neurons to discharge as a function of; (a) response type, and (b) SOA, are also presented in Table 3.2. We take the lower proportions of motor discharges for partial responses than for interrupted responses and full responses to be suggestive of the graded EMG onsets that are reported in this study. This present measure ignores the rate at which these motor neurons are recruited. Nevertheless, an extended model (see later) that reconstructed the EMGs from the motor discharge histories affirmed that the same patterns of motor discharge histories that delineate the response types reported here yielded the graded EMG onsets reported earlier (McGarry & Franks, 1997). (For further detail, see Section 4.3.5 Generation of EMG from the pattern of motor discharge.)

The go latencies for the full, interrupted and partial responses increased at longer SOAs as would be expected from the race model (see Table 3.2). In addition, the predicted ordering of full responses being faster than interrupted responses which, in turn, are predicted as being faster than partial responses (see Figure 2.5) was observed for any given SOA (see Table 3.2). The reverse prediction holds also in that the stop latencies were faster for the partial responses than for the interrupted responses for any SOA. (The stop latencies were absent for the full responses because they lost the race to the motor pool.) This result is consistent with the latency relations that would be expressed if variance in the go process rather than the stop process was assumed to be zero (not shown).

The expected relation of shorter stop latencies at longer SOAs were seemingly not observed for the interrupted responses or for the partial responses (see Table 3.2). The reason for this is that the stop latencies reported in Table 3.2 are taken with reference to the go signal and not with reference to the stop signal. If the SOA is subtracted from the stop latencies detailed in Table 3.2, then it can be deduced that the expected pattern of faster stop latencies at longer SOAs holds for both

interrupted responses and partial responses alike. If, as is the case, the SOA is not subtracted from the stop latencies in Table 3.2 then, in keeping with the time relations detailed in Figure 2.5, it can be observed that; (a) interrupted responses are observed when the go latencies are a little shorter than the sum of the SOA plus the stop latencies, and (b) partial responses are observed when the go latencies are a little longer than the sum of the SOA plus the stop latencies.

In sum, the hierarchical architecture provides for an increasing run-away effect as the neurons traverse the structure. This type of activity lends the appearance of independent stochastic latencies for the go process and the stop process as per the race model, yet, at the same time, it affords for the observance of graded EMG onsets by reason of the inhibitory influence that the stop process exerts on the go process at all levels in the hierarchy. These data demonstrate categorically that the latency relations that are expressed in the race model can also be generated from an account of excitatory-inhibitory interaction.

3.3.9 Excitatory-inhibitory interaction continued

The order and rate of discharge of the motor neurons affects the kinematics of the action. For example, increased recruitment of motor neurons and, later, increased firing rates, leads to increased force of muscle contraction. Small (slow) motor neurons discharge earlier and at a lower frequency than large (fast) motor neurons because of lower synaptic thresholds (Henneman, 1957; Henneman, Somjen & Carpenter, 1965a, b; Feiereisen, Duchateau & Hainart, 1997). These properties of motor neurons that discharge as a result of the level of synaptic drive to reach the motor pools determine the type of graded EMG onsets reported in this study. Steep EMG onsets result presumably from the recruitment and discharge of the motor neurons in the pool at maximal rates. If the stop process acts on the motor pool a short time thereafter, then any further recruitment of the larger motor neurons is prevented and those motor neurons that are already active are rapidly inhibited (in the reverse order of their recruitment), thus yielding interrupted responses at various times. If reduced synaptic drive acts on the motor pool, as a result of earlier effects of stopping on the go process, then only the small (slow) motor neurons are recruited at EMG onset. The result is shallow EMG onsets that typify the partial responses. In principle, this account explains how a maximal speeded action

might be reduced to a solitary motor unit twitch contraction from a single (small) motor neuron discharge as a result of stopping. This reasoning receives strong support from some of the EMG data reported in this study (Figures 3.2 and 3.3).

The single EMG spike trains were observed over and above the baseline signal. The presence of single motor spikes weakens the case further for a point of no return that might locate upstream of these discharges on two counts. First, we (McGarry & Franks, 1997) already noted that motor neurons receive inhibitory projections in support of a stopping mechanism that acts directly on the motor pool. Thus, a final ballistic process would not be expected to locate before motor discharge if the motor pool is subject to direct inhibitory influence as is the case. Second, we assert that to argue for a point of no return upstream of motor discharge would lead to a position of infinite regress. If, in Figure 3.4, motor discharge at level g is preceded by a ballistic process at level g-1 , then what of a ballistic process at level g-2 that precedes neural discharge at level g-1 and so on? We reason, in the noted absence of satisfactory evidence that would suggest otherwise (though see Osman et al., 1986), that the point of no return in all likelihood locates at the motor pools. In this context, a point of no return is of no theoretical consequence in the control of a voluntary action (McGarry & Franks, 1997).

The observances of single motor discharges when stopping a maximal speeded action is consistent with the position that the observed EMG data are the net result of an unfolding process of control that manifests at the motor pools. We have suggested that the stream of inhibition competes against the stream of excitation at all times up to motor discharge in order to explain the complement of responses - full, interrupted, partial and stopped - reported in this study. The viability of this explanation is supported from a computer analysis of excitatory-inhibitory interaction that yielded the graded EMG onsets that describe these response types (above), as well as maintaining the relations between latencies, SOAs and outcome probabilities that the race model describes.

3.4 Summary

This study reports on the various types of actions - full, interrupted, partial and stopped - observed when stopping a maximal speeded action at various times. These types of action are

discrete classifications of a continuum of reductions from full responses through to stopped responses, and can be explained from the time relation of the go process to the stopping process at the finish line. Single EMG spikes were reported in this study and provided good evidence for response reductions right up to single motor unit discharge. These data argue strongly that the finish line to which each process races is the motor pool, so speaking against a point of no return, or a final ballistic process, in the control of a voluntary action. In addition, the findings of advanced timing in the onset of the antagonists (biceps) in relation to the onset of the agonists (triceps) for the interrupted responses as opposed to the full responses, as well as the possibility of increased antagonist activity in relation to agonist activity, indicate a fluid on-line mechanism of control as the action unfolds. Finally, this study shows by way of computer analyses that an account of excitatory-inhibitory interaction at the level of the neuron can best account for the triceps EMG onsets, while, at the same time, retaining the relations between the go latencies, stop latencies, SOAs and outcome probabilities as described previously in the race model.

4 Experiment III

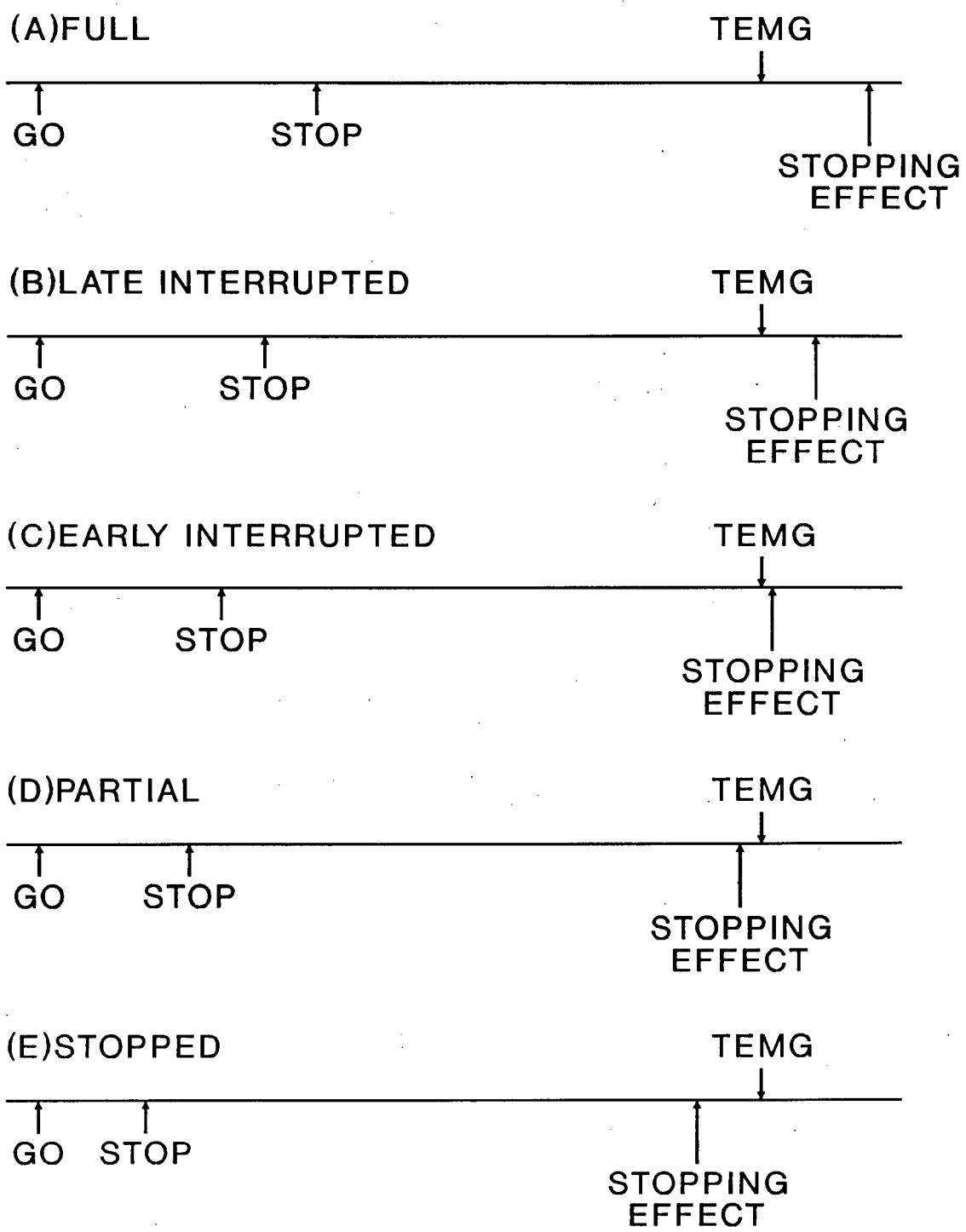
4.1 On the control of stopping an earlier intended voluntary action

4.1.1 On graded EMG onsets

We have reasoned earlier that the various distinctions of EMG onsets are of theoretical import to a race theory for the control of action. Quantitative measures of EMG onset can be obtained on the assumption that the rate of rise in EMG is described in a power law relation, in which case the integrated area under the rectified-filtered EMG envelope for the first $\underline{t}$ ms ($Q_{\underline{t}}$) can be used to quantify this rate of rise from onset (Gottlieb, Corcos & Agarwal, 1989a). (In fact the integrated area is taken from the untreated EMG data and is therefore independent of the chosen post sampling filter.) Hence we use $Q_{\underline{t}}$ as an index of the EMG onset, where $\underline{t}$ is any positive integer that is less than the time to the first peak of the EMG envelope (see later). The higher the $Q_{\underline{t}}$ (for any constant $\underline{t}$), the higher the power law relation and the steeper the EMG onset. Thus, the first aim of this study is to affirm statistically the distinction of the various response types - no-stop, full, interrupted, partial and stopped - as delineated from EMG onsets (McGarry & Franks, 1997). The hypothesis is that no-stop responses, full responses, and interrupted responses will not differ in their rise in EMG onset, while partial responses will have a reduced rise in EMG onset as compared with each of the other types of response.

We have advocated elsewhere (see Section 2.3.4 Evidence for a phantom point of no return in the preparation of a speeded elbow extension for further detail) that the various types of responses observed in the presence of a stopping process are specified in the relation of the go latencies and the stopping latencies in regard to the finish line, that is by how far the go process or the stop process wins the race (see Figure 2.5). Figure 2.5 and Figure 4.1 detail the same latency relations at the finish line in different ways. In Figure 2.5 these relations are expressed by varying the go latencies and keeping the SOAs and the stopping latencies constant, while in Figure 4.1 these same relations are expressed by varying the SOAs and keeping the go latencies and the stopping latencies constant. These relations (see Figure 4.1) yielded; (a) a full response, (b) a late interrupted response, (c) an early interrupted response, (d) a partial response, and (e) a stopped response, each on account of the

Figure 4.1 Proposed temporal relations detailing (A) a full response, (B) a late interrupted response, (C) an early interrupted response, (D) a partial response and (E) a stopped response as a function of the time between presentation of the go signal and the stop signal (SOA). Zero (no) variance in the go process and the stop process is assumed for ease of presentation. GO = go signal. STOP = stop signal. TEMG = time to EMG agonist onset. STOPPING EFFECT = effect of the stop process.



size of the go-stop (Figure 4.1A, B and C) or stop-go (Figure 4.1D and E) margins at the finish line. In practice, the go latencies, the stopping latencies and the SOAs would each vary from trial to trial although their relations at the finish line, as detailed in Figure 2.5 and in Figure 4.1, would still be produced so yielding the various types of responses reported earlier.

The go-stop and the stop-go margins expressed above are in relation to the finish line and are therefore independent of where the finish line might locate in the stream of control. In other words, the presence or absence of a point of no return has no bearing on the above relations. If these hypothesized latency relations are correct, then it follows from Figure 4.1 that more full responses than interrupted responses and, likewise, more interrupted responses than partial responses will gather at the high end of the SOA spectrum. Of course, the reverse hypothesis holds at the low end of the SOA spectrum. These predictions are in line with the race model and, as such, are not expected to differ fundamentally when variance in each process is allowed for (c.f., Logan & Cowan, 1984; De Jong et al., 1990). Thus, the second aim of this study is to affirm statistically that the graded EMG onsets lie on a temporal continuum in the hypothesized SOA direction.

4.1.2 On measures of latency and amplitude

That the race model well describes the latency relations between the go process, the stop process, the SOA and the outcome probabilities is well documented (De Jong et al., 1990; Logan & Cowan, 1984; Logan et al., 1984; Osman et al., 1986, 1990). To recap, shorter SOAs yield decreased probabilities of an action at shorter go latencies and longer SOAs yield increased probabilities of an action at longer go latencies. These results are observed because the stopping latencies intersect the go latency distributions at various times as a function of the SOA (see Figure 1.1).

If the amplitude of an action rather than the latency of an action is the preferred measure then the race model is troubled by the observance of graded EMG onsets. This is the case if the control of stopping an earlier intended action is to be explained satisfactorily. The problem herein is that the graded EMG onsets are analogous to the go process winning the race but crossing the finish line at much reduced velocities. One might think that the stopping process retards the progress of the go process as each process races to the finish line, but this account is unsatisfactory as it stands in that

it, presumably, would predict an increase in go latencies in the presence of the stopping process. This is not the case as observed from the empirical data.

We (McGarry & Franks, in review-a) demonstrated using computer analyses that excitatory-inhibitory interaction at all times up to the motor pool can explain unitarily the patterns of empirical data on stopping, namely the outcome probabilities of the latency relations as a function of SOA and their respective graded EMG onsets. We extend this computer model in this study in order to develop further a theory for the control of action. Thus, the third aim of this study is to augment the race model by analyzing how the winning margins of continuous processes might explain EMG onsets of varying amplitudes while, at the same time, maintaining the data relations that the race model expresses. In so doing, we seek, by way of a connectionist architecture, to provide a synthesis between the results observed thus far from cognitive science and some of the principles from neuro-science.

In summary, the purpose of this study is three fold: First, to quantify and establish the hypothesized differences in the classifications of the various response types from EMG onsets; second, to establish the hypothesized distribution of the response types (full, interrupted and partial) as a function of SOA; and third, to develop further a computer model that seeks to re-produce the graded EMG onsets while, at the same time, retaining, for the most part, the latency relations as described in the empirical data.

4.2 Method

The same experimental task, apparatus, procedure and protocol and associate measures as in Experiment II were used with the following exceptions.

4.2.1 Participants

Twenty (12 males, 8 females) right hand dominant participants ranging in age from approximately 20 years through 40 years were recruited in this study. One participant partook in Experiment I, one participant partook in Experiment II and one participant partook in Experiments I and II. Testing occurred in a single session. Each participant received $20 remuneration on completion of the session.

4.2.2 Apparatus

The same apparatus as that from Experiment II was used in this study.

4.2.3 Procedure

Two experimental (task) conditions - the go-only task and the inhibit task - were administered to each participant. Stop signals were presented with .333 probability. Variable SOAs ranging from -105 ms through 150 ms in 15 ms steps were used in accord with a computer algorithm (see later). Catch trials consisting of neither a go signal nor a stop signal were presented with .100 probability in each task to discourage anticipation. The stop signals and catch trials were counterbalanced over trials.

4.2.4 Protocol

The latency of each trial was identified on-line by the experimenter from an observed change in displacement. The trial was marked as "BAD" and rejected on-line by the experimenter if; (a) its latency of reaction exceeded 400 ms in the absence of a stop signal, or (b) the participant failed to react to a go signal in the absence of a stop signal. Trials yielding long latencies (i.e., above 400 ms) were rejected in an effort to offset the slowing that typically accompanies the stopping task. In spite of this procedure, latency slowing was still observed, as indicated from a later analysis of TEMGs using a one-tail paired t-test between those trials observed in the go-only task and those trials observed in the inhibit task in the absence of a stop signal (i.e., no-stop). The result, $t(19) = 4.374$, $p = < .001$ ($N_{go\text{-}only} = 20$, $\overline{X}_{go\text{-}only} = 207.68$, $SD_{go\text{-}only} = 24.09$; $N_{no\text{-}stop} = 20$, $\overline{X}_{no\text{-}stop} = 235.57$, $SD_{no\text{-}stop} = 27.51$), affirmed that the stopping task is a stubborn distractor in the generation of an action (c.f., Henry & Harrison, 1961; McGarry & Franks, 1997). Every other trial was marked as "GOOD" and accepted on-line by the experimenter.

10 GOOD trials in the go-only task and 100 GOOD trials in which a stop signal was presented in the inhibit task were collected. A few practise trials preceded each task for purposes of familiarity. The number of GOOD trials, the go latency and the response status (i.e., correct, incorrect) were presented after each trial as feedback.

The infrequencies with which a partial response have been observed in the earlier studies (McGarry & Franks, 1997, in review-a) is a presumed consequence of the brief window within these types of actions can be viewed. Thus, it is difficult to evoke these unwitting yet important responses from the experimental protocol. In order to maximize the frequencies of partial responses observed in this study, the SOA was changed systematically on the basis of task performance in an effort to probe the response around 0.500 probability. This algorithm was expected to optimize the likelihood of observing partial responses since the participant would, in many cases, be on the verge of initiating and of stopping that same action.

The SOA on the first trial in which a stop signal was presented was set at -30 ms, after which the SOA was updated according to the probability of stopping. Whether the participant moved or stopped in the presence of a stop signal was determined from the presence or absence of the latency mark respectively, as identified on-line by the experimenter on the basis of a change in displacement. The updated probability of stopping on trial n was used in order to determine the SOA for the next stop trial on the basis of its earlier probability on trial n - 1. (n is the number of trials that a stop signal is presented.) If the updated probability of stopping on trial n was further from .500 than its probability on trial n - 1, then the SOA was incremented or decremented by 15 ms depending on the direction of change in the updated probability otherwise the SOA was left unchanged. The SOA algorithm used the following nested if statement within -105 ms and 150 ms limits:

if p_n - .500 > 0 then

  if p_n - .500 > p_{n-1} - .500 then

    if SOA < 150 ms then SOA = SOA + 15 ms

else

if p_n - .500 < 0 then

  if p_n - .500 < p_{n-1} - .500 then

    if SOA > -105 ms then SOA = SOA - 15 ms

where p = probability of stopping and n = number of stop signal trials presented. For example, if p_{n-1} = .400 (e.g., 8 from 20) and p_n = .381 (i.e., 8 from 21) then the SOA is decremented 15 ms (providing

that the SOA is not -105 ms) for the next trial in which a stop signal is to be presented. If, however, $\underline{p}_{\underline{n}}$ = .429 (i.e., 9 from 21) then the SOA is left unchanged. Post hoc analysis of the probability of stopping showed that this algorithm was generally successful ($\underline{n}$ = 20, $\underline{p}_{\underline{x}}$ = 0.513, $\underline{p}_{\underline{sd}}$ = 0.075).

4.2.5 Data analysis

The data were re-analysed post event. Triceps EMG were marked by the experimenter for all trials except those in which no action occurred (i.e., stopped). The onset of triceps EMG was taken as the leading edge of EMG above baseline. This process was undertaken with extreme care, given its importance as it relates to measures of EMG onset.

The extension displacement data and, later, the untreated triceps EMG data and the rectified-filtered (20 Hz) triceps EMG data were used to classify the response types as full, interrupted, partial or stopped as reported earlier (see Section 3.3.1 Data Analysis for further detail) to which we add the following comments.

4.2.6 Classification of the various response types in the presence of a stop signal

We wish to verify our earlier distinction that sub-maximal EMG data that typify partial responses are not of the same form as those that typify (early) interrupted responses. We achieve this aim by categorizing the data into various response types - no-stop, full, interrupted and partial - and then analyzing these response types for statistical difference. These classifications are discrete categorisations of a continuum from maximal EMG through to zero EMG. They are necessary in order to verify partial responses as being distinct from interrupted responses that, in some cases, might be stopped very early in their execution.

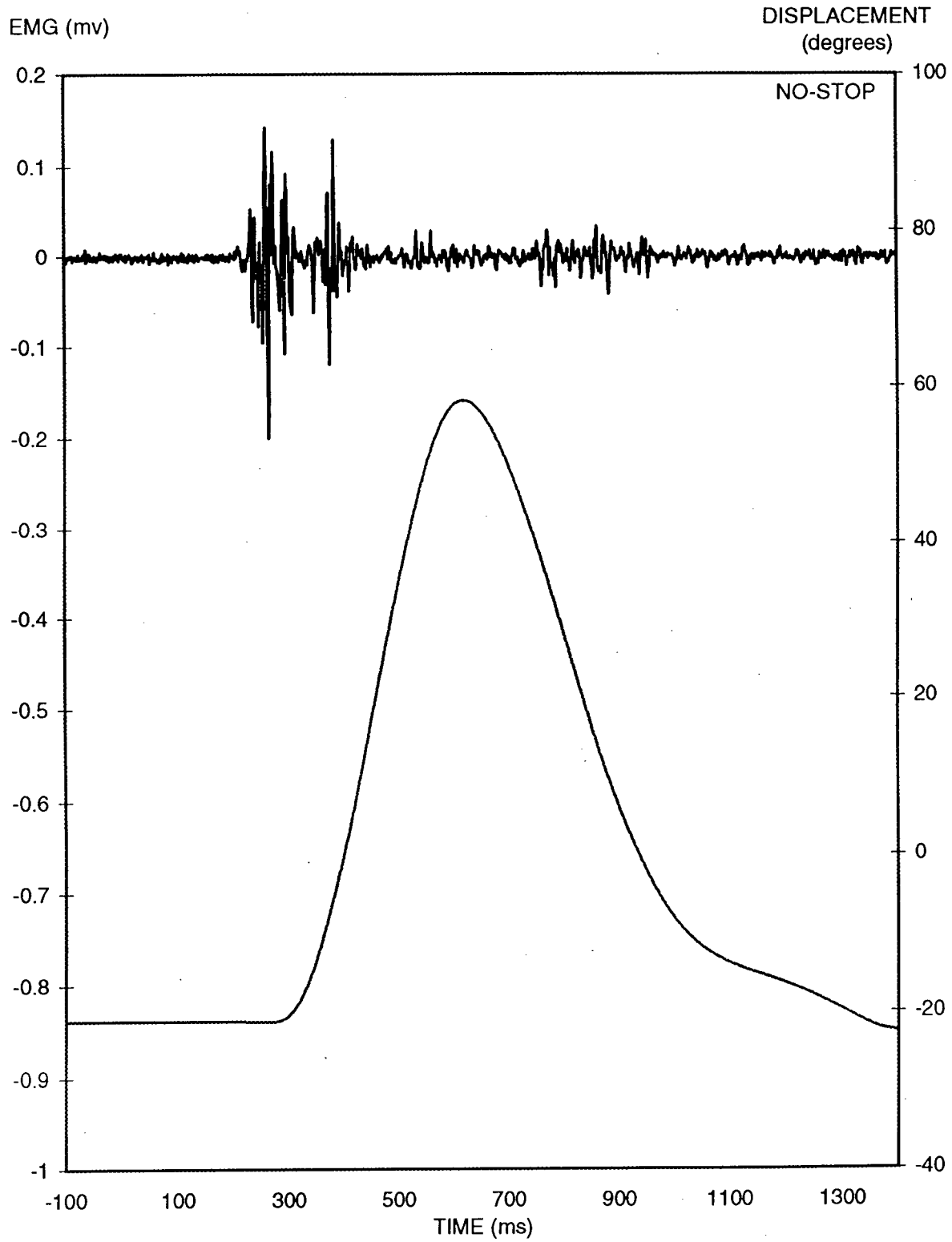
To recap, a response was classified as; (a) full if its extension displacement was more than $\overline{X}_{\text{No-stop}}$ minus one $SD_{\text{No-stop}}$, and (b) non-full if its extension displacement was less than $\overline{X}_{\text{No-stop}}$ minus two and a third $SD_{\text{No-stop}}$s. Non-full responses were subsequently classified as interrupted responses or partial responses from visual analysis of their triceps EMG data, both untreated EMG and rectified-filtered (20 Hz) EMG. Non-full responses that were not considered as partial responses were considered as interrupted responses by exclusion. However, those interrupted responses as classified that failed to

show the typical EMG characteristics were discarded from further analysis. This procedure was undertaken in order to preserve the integrity of the interrupted responses. Three responses from 725 non-full responses were discarded on this basis. Nine additional responses from a total of 4686 responses were excluded from the EMG analysis.

Visual analysis was used to delineate the partial responses from the interrupted responses because the extension displacement is not necessarily a satisfactory discriminator in this regard. If, for example, an early interrupted response and a partial response yielded similar extension displacements then a secondary measure, perhaps velocity, acceleration or EMG onset, would necessarily be required to adjudicate on the classification of that trial. In addition, we note that the various response types first identified by McGarry and Franks (1997) were classified on the basis of their EMG onsets rather than on the consequent kinematics of that action. For these reasons, we chose to use visual analysis as per McGarry and Franks (1997) in order to further segregate partial responses from interrupted responses. One might suspect circularity if the partial responses are later shown to be statistically different from the interrupted responses on the basis of their identity on that same measure (i.e., Q_t). We suggest that, in the absence of a better discriminator, any statistical difference between the partial responses and the interrupted responses would simply affirm our earlier visual observation that these two types of responses are indeed distinct (McGarry & Franks, 1997).

Figure 4.2a-f details the untreated triceps EMG (upper trace) and the associate displacement trace (lower trace) for a no-stop (Figure 4.2a), full (Figure 4.2b), late interrupted (Figure 4c), early interrupted (Figure 4d), partial (Figure 4.2e) and stopped (Figure 4.2f) response from Participant 1. The first (left) y-axis relates to the triceps EMG data and the second (right) y-axis relates to the displacement data. Visual analysis of these data provides for the following description.
Figure 4.2a-f indicates no observed difference between the no-stop response (Figure 4.2a) and the full response (Figure 4.2b) in their EMG traces, so suggesting no effect of stopping on the go process in the latter instance. In contrast, the triceps EMG for the late interrupted response (Figure 4.2c) and the early interrupted response (Figure 4.2d) were seemingly cut-off late and early in their rise from

Figure 4.2 Untreated triceps EMG (upper trace) and displacement (lower trace), detailing (a) a no-stop response, (b) a full response, (c) a late interrupted response, (d) an early interrupted response, (e) a partial response, and (f) a stopped response for Participant 1. The left y-axis reflects EMG (mv) and the right y-axis reflects displacement (degrees).



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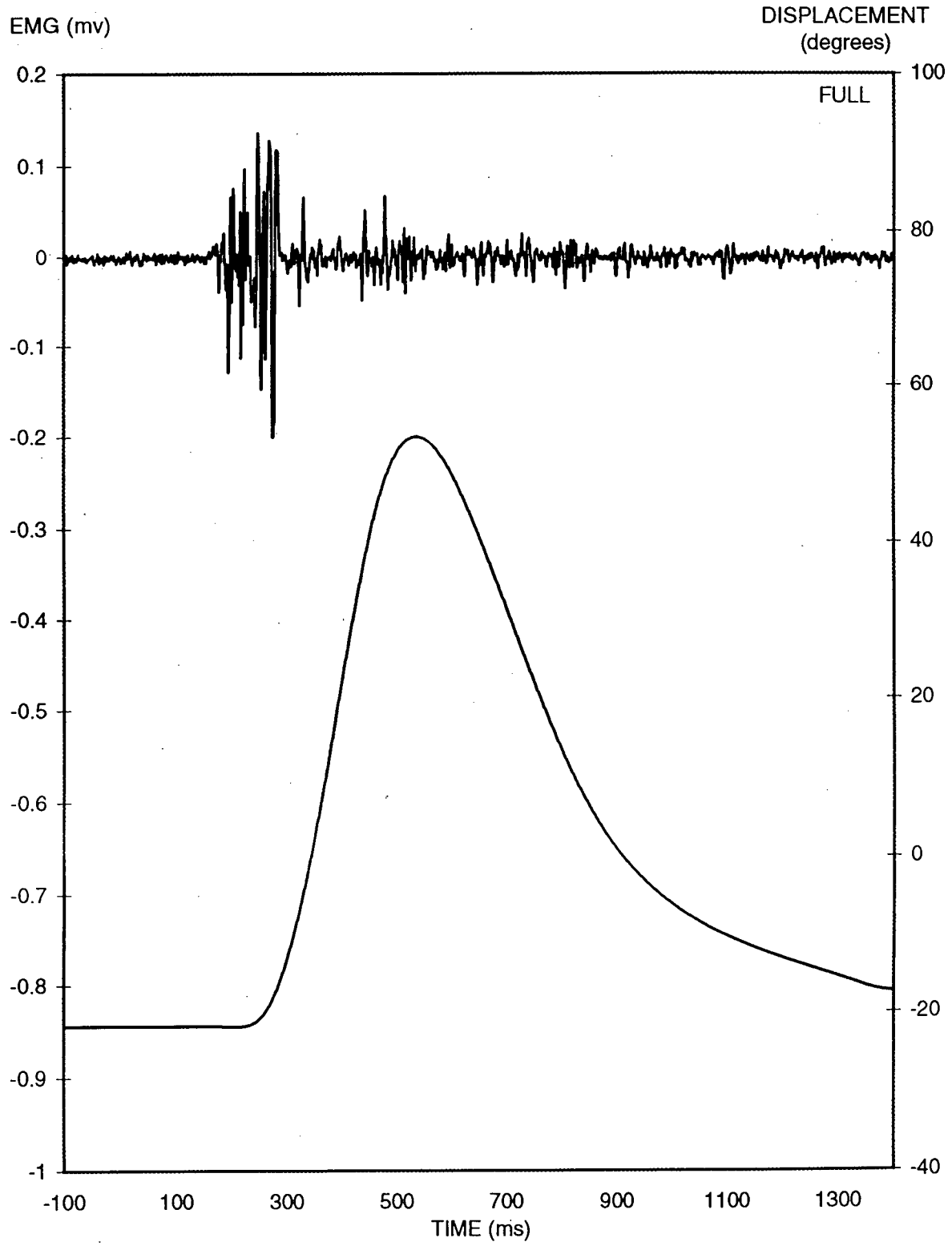

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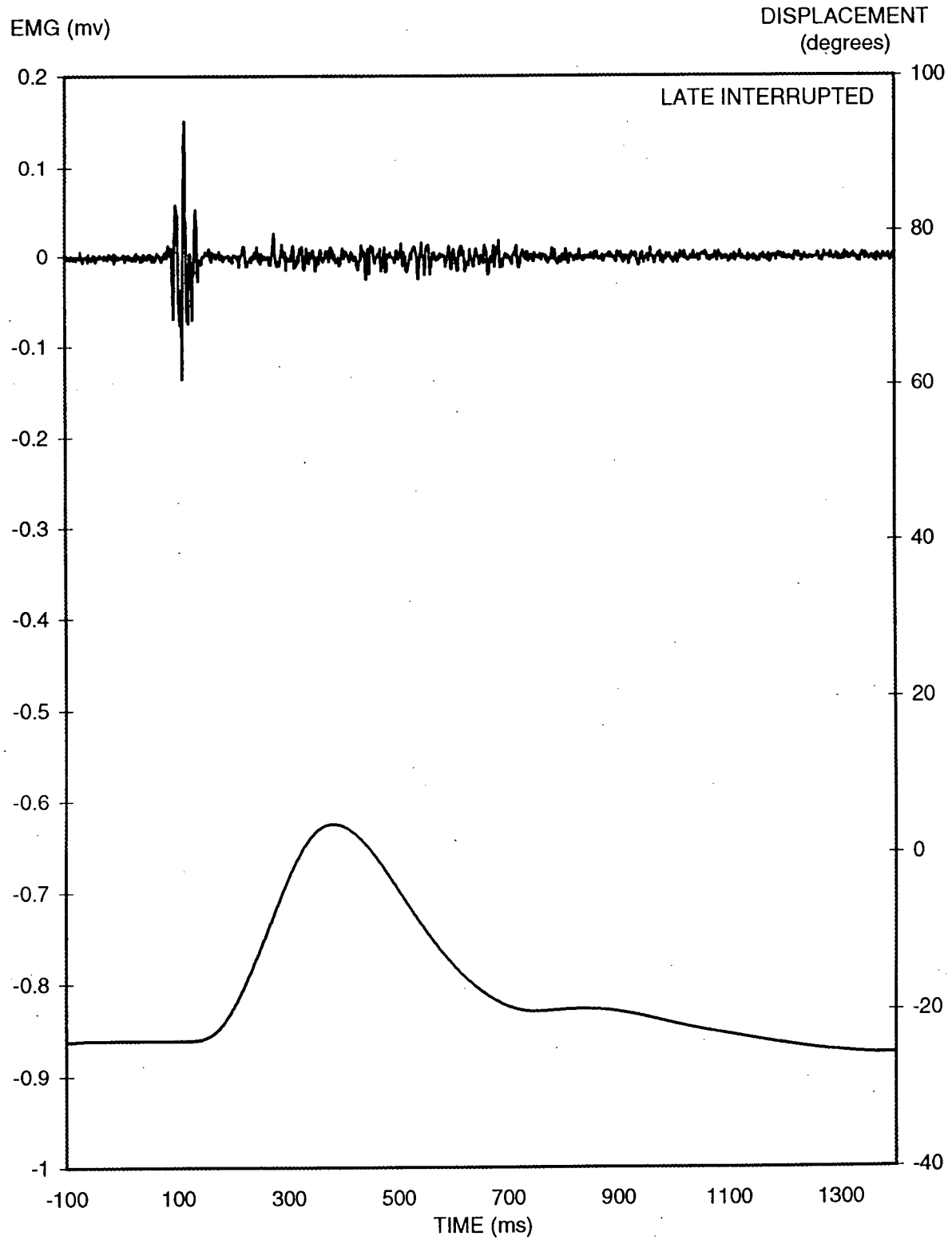

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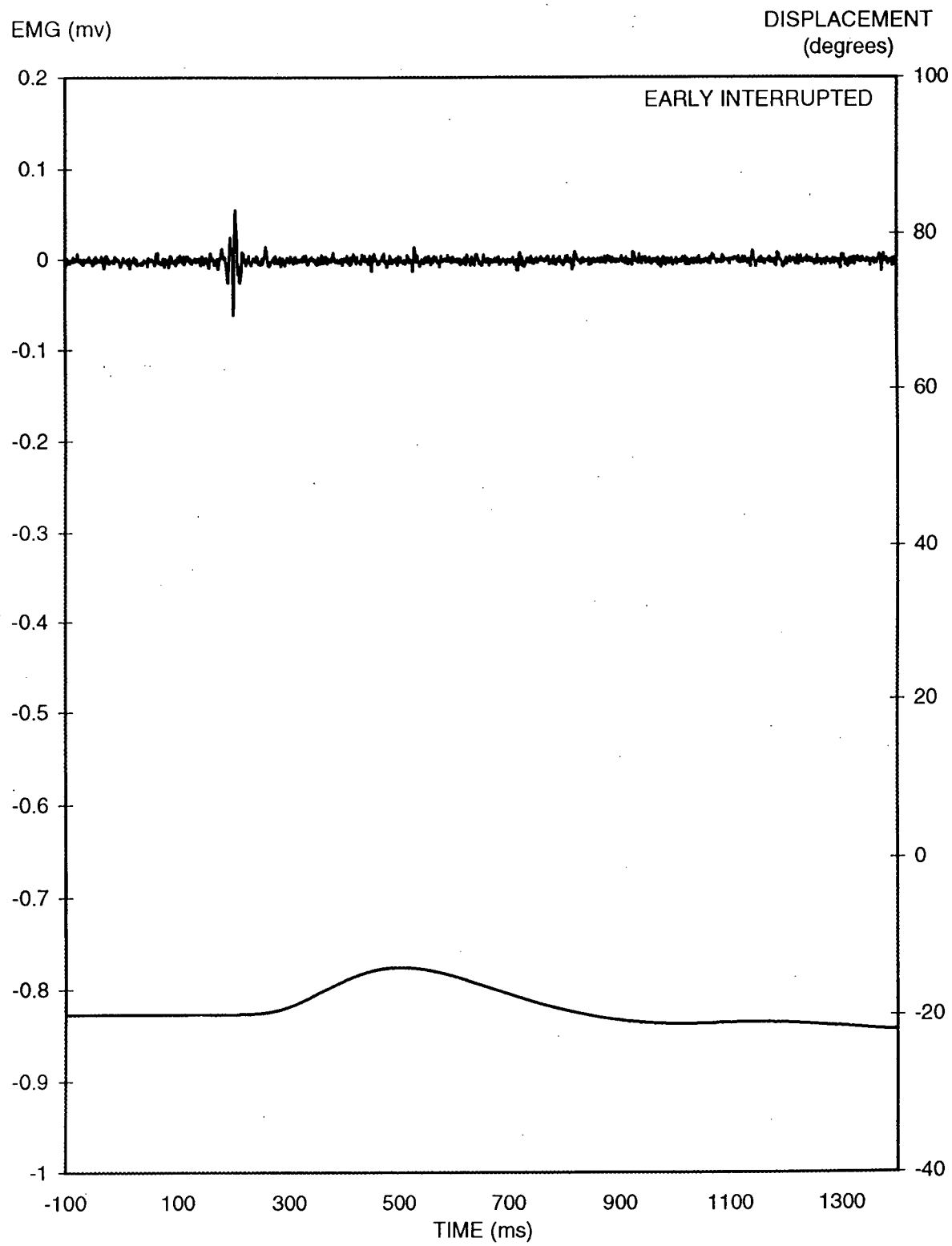

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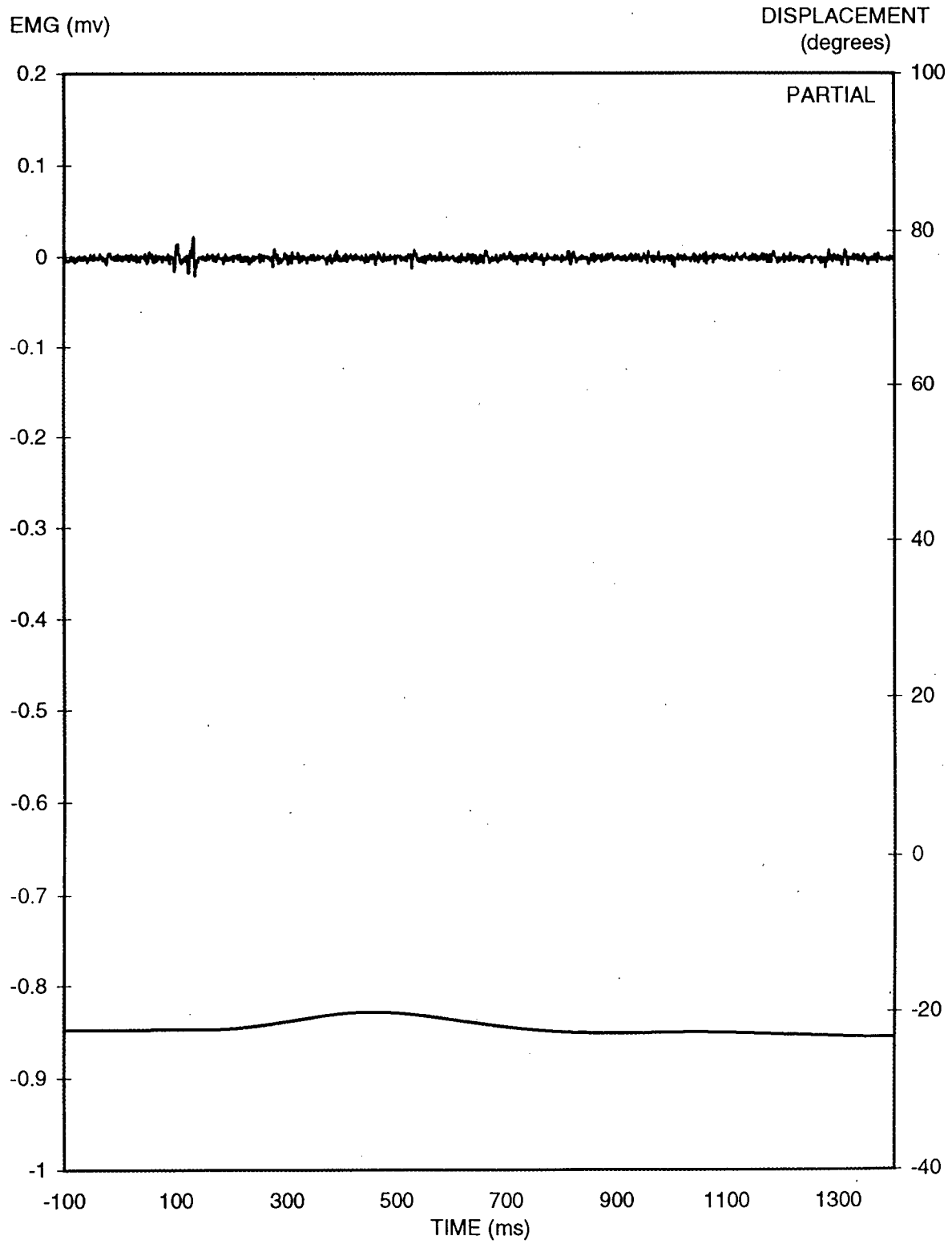

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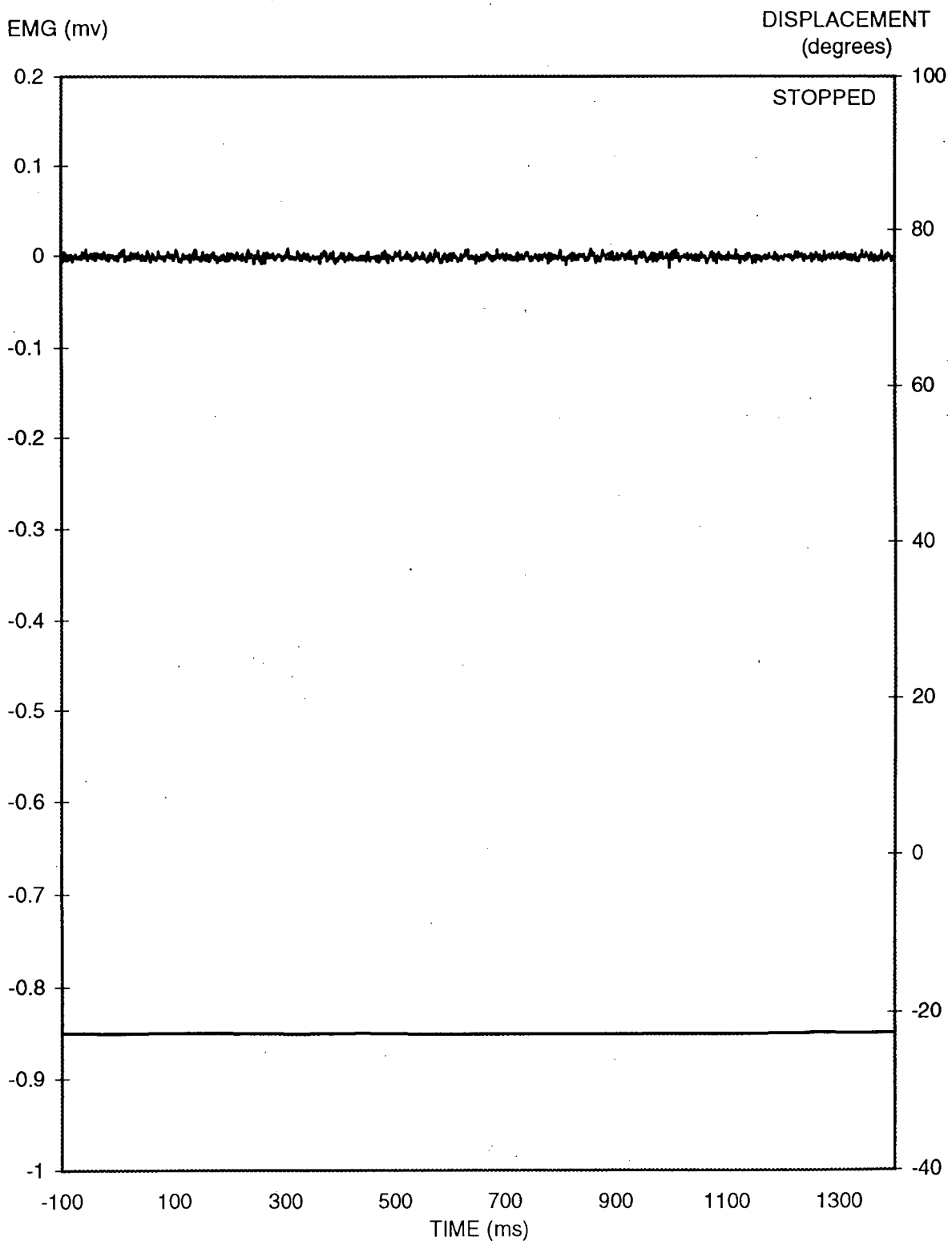

onset as compared with the no-stop response (Figure 4.2a) and the full response (Figure 4.2b). This observation is based on the untreated triceps EMG rather than the rectified-filtered (20 Hz) EMG which is not shown in these figures. Likewise, a reduced triceps EMG at onset is observed for the partial response (Figure 4.2e) as contrasted to the late interrupted response (Figure 4.2c) and the early interrupted response (Figure 4.2d). In addition, as in the previous studies (McGarry & Franks, 1997, in review-a), an associate extension displacement is observed to accompany the sub-maximal triceps EMG data that typifies a partial response. This result is not repeated for the stopped response (Figure 4.2f) which is characterised by the absence of triceps EMG above baseline as well as by the absence of a change in displacement. These example results (Figure 4.2a-f) indicate that each response type possesses the unique triceps EMG characteristics that describe it in support of the classification procedure used in this study.

4.2.7 Quantitative measures of the rise in EMG from onset (Q_{10}, Q_{20} and Q_{30})

Each dependent variable for each response type was treated for outliers preceding its use in statistical analysis. This statistical treatment, which was applied to all data in independent fashion, used an iterative procedure that rejected the highest (lowest) datum item that fell three SDs either side of $\overline{X}$ until all the datum items fell within the statistical criteria.

Following Gottlieb et al. (1989a), we used Q_t as an index of the rate of rise of EMG from onset. Gottlieb et al. (1989a) used Q_{30}, (i.e., $\underline{t}$ = 30 ms) obtained from trial and error, which they considered to be a satisfactory measure of the central drive as indexed in the EMG onset. We analysed EMG over three different time windows, $\underline{t}$ = 10 ms, $\underline{t}$ = 20 ms and $\underline{t}$ = 30 ms (i.e., Q_{10}, Q_{20} and Q_{30}), in order to test the hypothesis that the partial responses have distinct sub-maximal EMG onsets, while the interrupted responses and the full responses remain indistinct from the no-stop responses with maximal EMG onsets. While it might be objected that the first 10 ms of EMG data are of little consequence to the control of an action, this argument is specious as it pertains to the purpose of this study. While Q_{10} would describe little of the first EMG burst for a full response and, perhaps, little of the first EMG burst for an interrupted response, depending on how early that response is

interrupted, it might well describe much of the first (and often only) EMG burst for a partial response. The important point for this study is that Q_{10} describes the common initial EMG onsets of each type of response, as detected from the surface electrodes.

We use Q_{10} and Q_{20} as well as Q_{30} since partial responses are typically of short duration and hence might reach peak integrated EMG before 30 ms, in which case Q_{30} would under-estimate the rate of rise from EMG onset. We guard against this by rejecting any Q_t value whose time to peak rectified-filtered EMG (20 Hz) is less than $\underline{t}$. The addition of Q_{10} and Q_{20} is justified on three counts. First, Q_{10} and Q_{20} augment Q_{30}. Second, increasing frequencies are to be expected as $\underline{t}$ approaches zero since fewer partial responses will be excluded on the basis that $\underline{t}$ exceeds the time to peak integrated EMG (see Table 4.1). Third, if, as hypothesized, partial responses differ from no-stop, full and interrupted responses in their EMG onsets, then, smaller values of $\underline{t}$ in fact provide a stronger test in this regard.

4.3 Results and Discussion

4.3.1 Analyzing the rise of EMG from onset

Q_{10}, Q_{20}, and Q_{30} were subjected to a one-way (response type) repeated measures ANOVA (see Table 4.1). Significance was established for Q_{10}, $\underline{F}(3,48) = 14.800$, $\underline{p} < .001$, Q_{20}, $\underline{F}(3,45) = 31.658$, $\underline{p} < .001$, and Q_{30}, $\underline{F}(3,24) = 15.552$, $\underline{p} < .001$, and so each ANOVA was followed with a post hoc analysis using the studentized range test (Howell, 1997). Table 4.1 shows the same pattern of results across Q_t in that, as hypothesized, the partial responses were significantly reduced in their rate of EMG rise from those of the no-stop responses ($\underline{p} < .01$), full responses ($\underline{p} < .01$) and interrupted responses ($\underline{p} < .01$). These results are independent of Q_t and provide confirmatory evidence in support of the claim that the go process is markedly affected by the stop process before its observance in EMG onset. The pattern of results change a little with Q_t in that a reduced EMG gradient for the interrupted responses emerges for Q_{20} and Q_{30}. In general terms, these data tend towards, but fail to reach, significance at the level of $\underline{p} < .05$ (except for the Q_{20} no-stop and interrupted difference) and are to be expected if a continuum of response reductions exist (c.f., McGarry & Franks, in review-a). These data are also inconsistent with the earlier identities of the interrupted responses as detailed by

Table 4.1 Q_{10}, Q_{20} and Q_{30} descriptive and statistical data for the no-stop, full, interrupted and partial responses.

	Response Type											
	NS			F			I			P		
	n	$\overline{X}$	SD	n	$\overline{X}$	SD	n	$\overline{X}$	SD	n	$\overline{X}$	SD
Q_{10}	[a]17	0.12	.04	17	0.12	.05	17	0.10	.04	17	0.07	.04
Q_{20}	[ab]16	0.39	.12	16	0.34	.14	16	0.30	.09	16	0.15	.09
Q_{30}	[abc]9	0.88	.30	9	0.87	.40	9	0.64	.25	9	0.33	.20

	Q_{10}				Q_{20}				Q_{30}			
	NS	F	I	P	NS	F	I	P	NS	F	I	P
NS	-	0.00	0.02	0.05**	-	0.05	0.09**	0.23**	-	0.02	0.24	0.55**
F		-	0.01	0.05**		-	0.04	0.19**		-	0.23	0.53**
I			-	0.03**			-	0.14**			-	0.31*
P				-				-				-

Note. NS = No-stop. F = Full. I = Interrupted. P = Partial. n = number of participants. $\overline{X}$ = mean. SD = standard deviation. Q_{10} = rectified-integrated EMG data for the first 10 ms. Q_{20} = rectified-integrated EMG data for the first 20 ms. Q_{30} = rectified-integrated EMG data for the first 30 ms. [a] Participants 10 and 11 excluded from ANOVA on account of absent response types (see Table 4.2). Participant 12 excluded from ANOVA on account of integrated time exceeding time to rectified-filtered peak (see text). [b] Participants 14 excluded from ANOVA on account of integrated time exceeding time to rectified-filtered peak (see text). [c] Participants 4, 6, 13, 16, 19 and 20 excluded from ANOVA on account of integrated time exceeding time to rectified-filtered peak (see text). * $p < .05$. ** $p < .01$. Units are mvolts seconds for time t = 10, 20 and 30 msecs respectively.

McGarry and Franks (1997) from visual analysis of the rectified-filtered EMG data. This situation arises as a result of the different classification systems used to delineate interrupted responses between the two studies. In particular, we expect that many candidate interrupted responses would have been excluded from this study since they failed to reach the displacement criterion of being less than two and a third $SD_{no\text{-}stop}$s less than $\overline{X}_{no\text{-}stop}$. The effect of this expulsion would be to bias the interrupted responses towards lower Q_ts.

We reason that Q_t constitutes an exclusive measure of the contribution of the central (i.e., efferent) processes on the basis that the peripheral (i.e., afferent) processes are unable to operate within this time frame. De Jong, Coles and Logan (1995) argued from LRP measures for two different stopping mechanisms, one that comprises of central processes and one that comprises of peripheral processes.[9] In their view, central stopping processes act to suppress central motor planning, as evidenced from the inhibition of cortical activity before the stopping of a voluntary action, while peripheral stopping processes act to suppress motor activity at the periphery, as evidenced in the occasional observance of failure to inhibit cortical activity while still managing to inhibit motor activity in some cases. Our point is not to the nature of these different aspects of stopping, other than to affirm that the stopping effects take place seemingly at all times up to motor discharge, so yielding EMG onsets that span from maximum through zero. The earlier reported findings of single motor discharge provided good support for this proposition (see Figures 3.2 and 3.3).

4.3.2 Evidence for graded EMG onsets as a function of the latency relations between the go process and the stop process

The distributions of the full responses, interrupted responses and partial responses as a function of SOA are detailed in Table 4.2. Since each response type distribution across SOA varies by participant, it is first necessary that the distribution be subject to some kind of transformation before

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[9] De Jong et al. (1995) do not define what they consider central and peripheral processes to be. Our reading is that they do not constitute efferent and afferent processes as we would define them.

it is collapsed (reduced) across participants. Once transformed and reduced, the distribution of each response type (full, interrupted and partial) as a function of SOA will be analysed statistically for evidence of difference in the hypothesised direction (see Figure 4.1). The data were therefore subject to two procedures, the collapsing of frequencies across SOA bins (transformation) and the collapsing of SOA bins across participants (reduction). First, the SOA bins were collapsed within each participant into two bins, a low bin and a high bin. This has two advantages. Individual differences in response type distributions are preserved somewhat and the floor-ceiling effect provided by the lower-upper SOA bounds is for the most part assuaged. Second, the low bins and the high bins were collapsed across participants, thus yielding the respective frequencies for the full responses, interrupted responses and partial responses. These data were then subjected to a two-way (response type, bin) χ^2 analysis for evidence of difference in distribution between response types as a function of SOA.

Suppose that we wish to test the experimental hypothesis that more full responses than interrupted responses will be evidenced at the higher end of the SOA spectrum (Hypothesis A: $F_H > I_H$. See Table 4.2). In order to collapse the data into a low bin and a high bin for both the full responses and the interrupted responses, we first defined the two bins contingent on the hypothesis. In this example, the high bin contains more than half of the full responses per participant and the low bin, by exclusion, contains the rest of the full responses for that same participant. The high bin and the low bin for the full responses was then contrasted against the same high bin and low bin for the interrupted responses on a participant by participant basis. For example, Table 4.2 shows that of the seven full responses for participant 1, at least four (i.e., more than half) of the observations lie in the high bin, as indicated by the superscript that corresponds to the experimental hypothesis (i.e., A). In this case, the SOA bins from the highest bin (i.e., 150 ms) through to the lowest bin that contain at least four observations (i.e., -90 ms) constituted the high bin. The remaining bins (i.e., -105 ms) constituted the low bin which, in this example, are indicated by the absence of the superscript A.

Table 4.2 Frequencies of the full, interrupted and partial responses grouped into high bins and low bins by participant as per the experimental hypothesis.

Full	SOA (ms)																		
P	-105	-90	-75	-60	-45	-30	-15	0	15	30	45	60	75	90	105	120	135	150	Σ
1	$^{B}3$	$^{BA}1$	$^{BA}1$		$^{BA}1$	$^{A}1$													$^{BA}7$
2																	$^{A}3$	$^{A}1$	$^{A}4$
3						$^{B}1$						$^{B}1$	1			1	$^{A}4$	$^{A}1$	$^{BA}9$
4			$^{B}1$	$^{B}2$	$^{B}2$	$^{B}2$	$^{B}1$		$^{B}1$	1		3	1	1	$^{A}3$	$^{A}1$	$^{A}4$	$^{A}13$	$^{BA}36$
5			1		1										1	1	3	$^{A}11$	$^{A}18$
6	$^{B}3$	1																	$^{B}4$
7					$^{B}2$	$^{B}1$	$^{B}1$	2					$^{A}1$	$^{A}3$	$^{A}1$	$^{A}1$		$^{A}1$	$^{BA}13$
8									$^{B}1$						$^{B}1$		1	$^{A}8$	$^{BA}11$
9					$^{B}1$	$^{B}1$		$^{B}2$		$^{B}1$		$^{BA}2$	$^{BA}2$			$^{A}1$		$^{A}2$	$^{BA}12$
10																			-
11																			-
12	$^{B}1$	$^{B}2$		$^{B}1$	$^{B}1$	$^{B}1$			$^{B}2$	$^{A}1$		$^{A}1$		$^{A}2$		$^{A}1$		$^{A}4$	$^{BA}17$
13	$^{B}2$					$^{BA}1$	$^{BA}1$	$^{A}1$	$^{A}1$										$^{BA}6$
14	$^{B}4$	$^{A}2$			$^{A}1$	$^{A}2$													$^{BA}9$
15						$^{B}1$	$^{B}1$		$^{B}1$		$^{BA}2$		$^{BA}1$	$^{A}1$	$^{A}1$				$^{BA}8$
16										$^{B}2$						$^{B}1$	$^{B}1$	$^{A}6$	$^{BA}10$
17											$^{B}1$	$^{B}2$	$^{B}2$	$^{B}2$	1	1	$^{A}4$	$^{A}8$	$^{BA}21$
18		1		1												1		$^{A}8$	$^{A}11$
19						$^{B}1$	$^{B}1$		$^{B}3$	$^{B}3$	$^{A}1$	$^{A}4$	$^{A}1$	$^{A}2$		$^{A}1$			$^{BA}17$
20						$^{B}3$	$^{B}1$	$^{B}2$	$^{B}5$	$^{B}2$	$^{B}2$	$^{BA}6$	$^{A}6$	$^{A}5$	$^{A}6$	$^{A}2$			$^{BA}40$

Interrupted	SOA (ms)																		
P	-105	-90	-75	-60	-45	-30	-15	0	15	30	45	60	75	90	105	120	135	150	Σ
1	$^{DB}11$	$^{BA}1$	$^{BA}1$	$^{BA}3$	$^{CBA}1$	$^{CA}1$		$^{CA}6$	$^{CA}1$	$^{CA}1$	$^{CA}2$	$^{CA}3$	$^{CA}2$						$^{DCBA}33$
2							$^{DB}1$			$^{DB}1$	$^{DB}3$	$^{DB}3$	$^{DB}2$	$^{DB}4$	$^{CB}5$	$^{C}5$	$^{CA}5$	$^{CA}2$	$^{DCBA}31$
3							$^{DB}1$		$^{B}5$	$^{B}4$	$^{CB}4$	$^{CB}8$	$^{C}2$	$^{C}2$	$^{C}1$			$^{CA}1$	$^{DCBA}28$
4					$^{DB}1$		$^{DB}1$	$^{DCB}2$	$^{DCB}1$					$^{DC}1$			$^{DCA}1$	$^{CA}1$	$^{DCA}8$
5				$^{D}2$		$^{D}1$			$^{D}1$	$^{D}1$		$^{D}1$	$^{D}1$	$^{D}2$	$^{D}1$	$^{D}1$	4	$^{CA}18$	$^{DCBA}33$
6	$^{DB}36$			1	1	1													$^{DB}39$
7						$^{DB}1$	$^{DB}2$	$^{DB}2$	$^{DB}5$	$^{DB}1$	$^{DCB}6$	$^{DC}4$	$^{DCA}2$			$^{CA}2$	$^{CA}1$	$^{CA}3$	$^{DCBA}29$
8						$^{B}1$	$^{B}1$		$^{B}1$		$^{B}1$	$^{B}1$	$^{B}2$	$^{B}2$	$^{B}2$	$^{CB}3$	$^{C}3$	$^{CA}8$	$^{CBA}25$
9				$^{DB}1$	$^{DB}1$				$^{DB}1$	$^{DB}3$	$^{B}3$	$^{CBA}1$	$^{CBA}2$	$^{CA}3$	$^{CA}1$		$^{CA}1$	$^{CA}3$	$^{DCBA}20$
10												$^{D}1$	$^{D}4$	$^{D}2$		1	$^{C}8$	$^{C}13$	$^{DC}29$
11														$^{D}3$	$^{D}3$	5	$^{C}6$	$^{C}8$	$^{DC}25$
12	$^{DB}4$		$^{DB}1$	$^{DB}1$		$^{DB}2$	$^{DB}1$	$^{B}1$	$^{CB}3$	$^{CA}1$				$^{CA}2$		$^{CA}2$	$^{CA}3$		$^{DCBA}21$
13	$^{DB}4$	$^{DB}1$	$^{B}1$	$^{B}2$	$^{B}1$		$^{CBA}1$	$^{CA}2$	$^{CA}3$	$^{CA}1$	$^{CA}3$								$^{DCBA}19$
14	$^{DB}24$	$^{DCA}2$	$^{DCA}1$			$^{CA}1$	$^{CA}1$	$^{CA}2$	$^{CA}1$	$^{CA}3$	$^{CA}2$	$^{CA}5$	$^{CA}2$						$^{DCBA}44$
15					$^{DB}1$		$^{DB}1$	$^{DB}1$	$^{DB}1$	$^{DB}1$		$^{DBA}4$	$^{CBA}3$	$^{CA}2$	$^{CA}5$	$^{CA}2$			$^{DCBA}21$
16									$^{B}2$	$^{B}1$			$^{B}1$	$^{B}1$		$^{B}1$	$^{CB}7$	$^{CA}12$	$^{CBA}25$
17							$^{DB}1$					$^{DB}5$	$^{DCB}3$	$^{DCB}1$	$^{C}1$	$^{C}4$	$^{CA}1$	$^{CA}2$	$^{DCBA}18$
18			1															$^{CA}5$	$^{CA}6$
19				$^{DB}1$	$^{DB}1$	$^{DB}1$	$^{DB}1$		$^{CB}1$	$^{CB}3$			$^{CA}1$	$^{CA}1$					$^{DCBA}10$
20							$^{DB}1$		$^{DB}1$			$^{DCBA}2$		$^{CA}1$	$^{CA}1$				$^{DCBA}6$

Table 4.2 continued.

Partial	SOA (ms)																		
P	-105	-90	-75	-60	-45	-30	-15	0	15	30	45	60	75	90	105	120	135	150	Σ
1	$^{D}9$				$^{C}1$	$^{C}1$					$^{C}1$								$^{DC}12$
2								$^{D}1$	$^{D}1$					$^{D}2$		$^{C}2$			$^{DC}6$
3							$^{D}1$	$^{D}1$						$^{C}1$					$^{DC}3$
4				$^{D}1$		$^{D}1$	$^{D}1$										$^{DC}1$	$^{C}2$	$^{DC}6$
5		$^{D}1$					$^{D}1$							$^{D}1$		$^{D}1$		$^{C}3$	$^{DC}7$
6	$^{D}17$		1																$^{D}18$
7									$^{D}1$	$^{D}1$			$^{DC}2$						$^{DC}4$
8						1		1	1	1		1	1	2				$^{C}8$	$^{C}16$
9							$^{D}2$	$^{D}1$	$^{D}1$	$^{D}2$	1	$^{C}2$							$^{DC}9$
10								$^{D}1$			$^{D}1$	$^{D}1$	$^{D}3$	$^{D}3$	$^{D}5$	2		$^{C}5$	$^{DC}21$
11										$^{D}2$	$^{D}3$		$^{D}2$	$^{D}3$	$^{D}4$	5	$^{C}5$	$^{C}1$	$^{DC}25$
12							$^{D}1$												$^{D}1$
13	$^{D}11$	$^{D}3$	4		1		$^{C}2$	$^{C}1$											$^{DC}22$
14	$^{D}5$	$^{DC}1$	$^{DC}1$		$^{C}1$				$^{C}1$	$^{C}1$		$^{C}2$							$^{DC}12$
15				$^{D}1$		$^{D}1$	$^{D}1$			$^{D}1$	$^{D}3$	$^{D}2$	$^{C}3$	$^{C}1$	$^{C}1$	$^{C}1$			$^{DC}15$
16								1						1	1	3	$^{C}3$	$^{C}11$	$^{C}20$
17						$^{D}1$						$^{D}1$	$^{DC}4$	$^{DC}3$		$^{C}1$	$^{C}1$	$^{C}3$	$^{DC}14$
18		2											3	1		3	1	$^{C}11$	$^{C}21$
19			$^{D}1$	$^{D}1$	$^{D}2$	$^{D}2$		$^{D}3$	$^{C}2$		$^{C}1$	$^{C}1$	$^{C}2$						$^{DC}15$
20									$^{D}1$			$^{DC}1$	$^{DC}1$	$^{C}1$		$^{C}1$			$^{DC}5$

Note. P = Participant. Σ = Total. [A] Hypothesis A: $F_H > I_H$. Frequencies of the full responses are segregated by the high bin and the corresponding interrupted responses so segregated. [B] Hypothesis B: $I_L > F_L$. Frequencies of the interrupted responses are segregated by the low bin and the corresponding full responses so segregated. [C] Hypothesis C: $I_H > P_H$. Frequencies of the interrupted responses are segregated by the high bin and the corresponding partial responses so segregated. [D] Hypothesis D: $P_L > I_L$. Frequencies of the partial responses are segregated by the low bin and the corresponding interrupted responses so segregated.

The same high bin and low bin contain 22 and 11 interrupted responses as indicated from the presence and absence of superscript A respectively.

This analysis was repeated for each participant and the data then collapsed across participant. Those full responses that failed to meet the standard for segregation into a high bin and a low binwere discarded from further analysis for that experimental hypothesis, along with their corresponding interrupted responses. In the case of Hypothesis A, the low bin (-105 ms) would contain equal or more full responses than the high bins (-90 ms through 150 ms) in order for it to be rejected from the analysis. The discarded responses are detailed from the complete absence of the superscript in a row, as identified from the total column at the end of each row. For example, the absence of the superscript A in Table 4.2 shows that of the four full responses credited to participant 6, and the zero full responses credited to participants 11 and 12, zero full responses were assigned to their respective low bins and high bins. Zero interrupted responses were assigned to the respective low bins and high bins for participants 6, 10 and 11 by extension. In total, four (from 253) full responses were discarded on the above basis together with 93 (from 470) interrupted responses.

If, on the other hand, suppose that we wish to test the reverse experimental hypothesis (i.e., Hypothesis B: $I_L > F_L$. See Table 4.2), namely that more interrupted responses than full responses will be evidenced at the lower end of the SOA spectrum. Here, a low bin contains more than half of the interrupted responses and the high bin, by exclusion, contains the rest of the interrupted responses for that same participant. The low bin and the high bin for an interrupted response was then contrasted against the same low bin and high bin for a full response.

For example, Table 4.2 shows that of the 33 interrupted responses for participant 1, at least 17 (i.e., more than half) of the observations lie in the low bin. The SOA bins from the lowest (i.e., -105 ms, Table 4.2) through to the highest (i.e., -45 ms, Table 4.2) that contain at least 17 observations constituted the low bin, as indicated by the superscript B. The remaining bins (i.e., -30 ms through 150 ms, Table 4.2) constituted the high bin, as indicated by the absence of the superscript B. The same low bin and high bin contains six full responses and one full response respectively. Once again, this analysis was repeated within participants and the data then collapsed across participants

and treated to χ^2 analysis. In this way, the data that pertain to the first experimental hypothesis (i.e., A: $F_H > F_L$) and to the second experimental hypothesis (i.e., B: $I_L < F_L$) are dependent on the analytic procedure, that is whether a high bin or a low bin is the criterion for segregation. The data, collapsed into the high bins and the low bins for each experimental hypothesis are detailed for each participant in Table 4.3. The data, further collapsed across participants (see total column, Table 4.3) were then subjected to a two-way (response type, bin) χ^2 statistical analysis.

Table 4.3 provides statistical evidence that significantly more full responses than interrupted responses gathered at the high end of the SOA spectrum, χ^2 (1, $\underline{N}$ =626) = 20.751, $\underline{p} < .001$, and, also, that significantly more interrupted responses than full responses gathered at the low end of the SOA spectrum, χ^2 (1, $\underline{N}$ =601) = 19.289, $\underline{p} < .001$. Likewise, the data showed that significantly more interrupted responses than partial responses were observed at the high end of the SOA spectrum, χ^2(1, $\underline{N}$ = 609) = 20.922, $\underline{p} < .001$, while significantly more partial responses than interrupted responses were observed at the low end of the SOA spectrum, χ^2 = (1, $\underline{N}$ = 609) = 45.845, $\underline{p} < .001$. These results yielded good evidence to suggest that the response types were differentially distributed across SOA in the hypothesized direction. These results are consistent with the varying latency relations expressed by varying the SOA in Figure 4.1. We investigate, via computer analysis, a mechanism of excitatory-inhibitory interaction that seeks to explain the data presented thus far, that is the various EMG onsets as a function of SOA.

4.3.3 On a theory of control for stopping a voluntary action

That the pattern of motor neuron discharge is a neuro-physiological consequence of the synaptic drive to reach the motor pools affords a distinct advantage in the control of force production. The higher centres need only specify the level of synaptic drive for delivery to the motor pools, and not the combinations of motor neurons that are necessary in order to produce a given force (Ghez, 1991). This method of control addresses Bernstein's (1935) degrees of freedom problem, which might be expressed in general terms as to how a motor problem of high dimensions can be explained

Table 4.3 Frequencies of the full, interrupted and partial responses grouped into high bins and low bins (from Table 4.2) grouped across participants as per the experimental hypothesis.

			Participant																				
Hypothesis	R	B	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	Σ
A: $F_H > I_H$	F	H	4	4	5	21	11	-	7	8	7	-	-	9	4	5	5	6	12	8	9	25	150
	F	L	3	-	4	15	7	-	6	3	5	-	-	8	2	4	3	4	9	3	8	15	99
	I	H	22	7	1	2	18	-	8	8	11	-	-	8	10	20	16	12	3	5	2	4	157
	I	L	11	24	27	6	15	-	21	17	9	-	-	13	9	24	5	13	15	1	8	2	220
B: $I_L > F_L$	I	L	17	19	22	5	-	36	17	14	12	-	-	13	10	24	12	13	10	-	8	4	236
	I	H	16	12	6	3	-	3	12	11	8	-	-	8	9	20	9	12	8	-	2	2	141
	F	L	6	-	2	9	-	3	6	2	9	-	-	8	4	4	6	4	7	-	8	21	99
	F	H	1	4	7	27	-	1	7	9	3	-	-	9	2	5	2	6	14	-	9	19	125
C: $I_H > P_H$	I	H	17	17	18	6	18	-	18	14	11	21	14	11	10	-	12	19	12	5	6	4	233
	I	L	16	14	10	2	15	-	11	11	9	8	11	10	9	-	9	6	6	1	4	2	154
	P	H	3	2	1	3	3	-	2	8	2	5	6	-	3	7	6	14	12	11	6	4	91
	P	L	9	4	2	3	4	-	2	8	7	16	19	1	19	5	9	6	2	10	9	1	131
D: $P_L < I_L$	P	L	9	4	2	4	4	17	4	-	6	14	14	1	14	7	9	-	9	-	9	3	130
	P	H	3	2	1	2	3	1	-	-	3	7	11	-	8	5	6	-	5	-	6	2	65
	I	L	11	14	1	7	11	36	23	-	6	7	6	9	5	27	9	-	10	-	4	4	190
	I	H	22	17	27	1	22	3	6	-	14	22	19	12	14	17	12	-	8	-	6	2	224

Note. R = Response type. B = Bin. F = Full response. I = Interrupted response. P = Partial response. H = High bin. L = Low bin. Σ = Total.

satisfactorily with a motor solution of low dimensions. In this case, how the inordinate array of inter-connecting neurons might be managed through the specification of synaptic drive. Thus, the control of an action is more easily explained if the motor discharge pattern is an *a posteriori* result from the synaptic drive rather than an *a priori* result of neural instruction. For our purposes, it is sufficient to note that an action can be stopped by simply preventing the synaptic drive to reach the motor pools, rather than by specifying a change in a motor program in order to accomplish this objective.

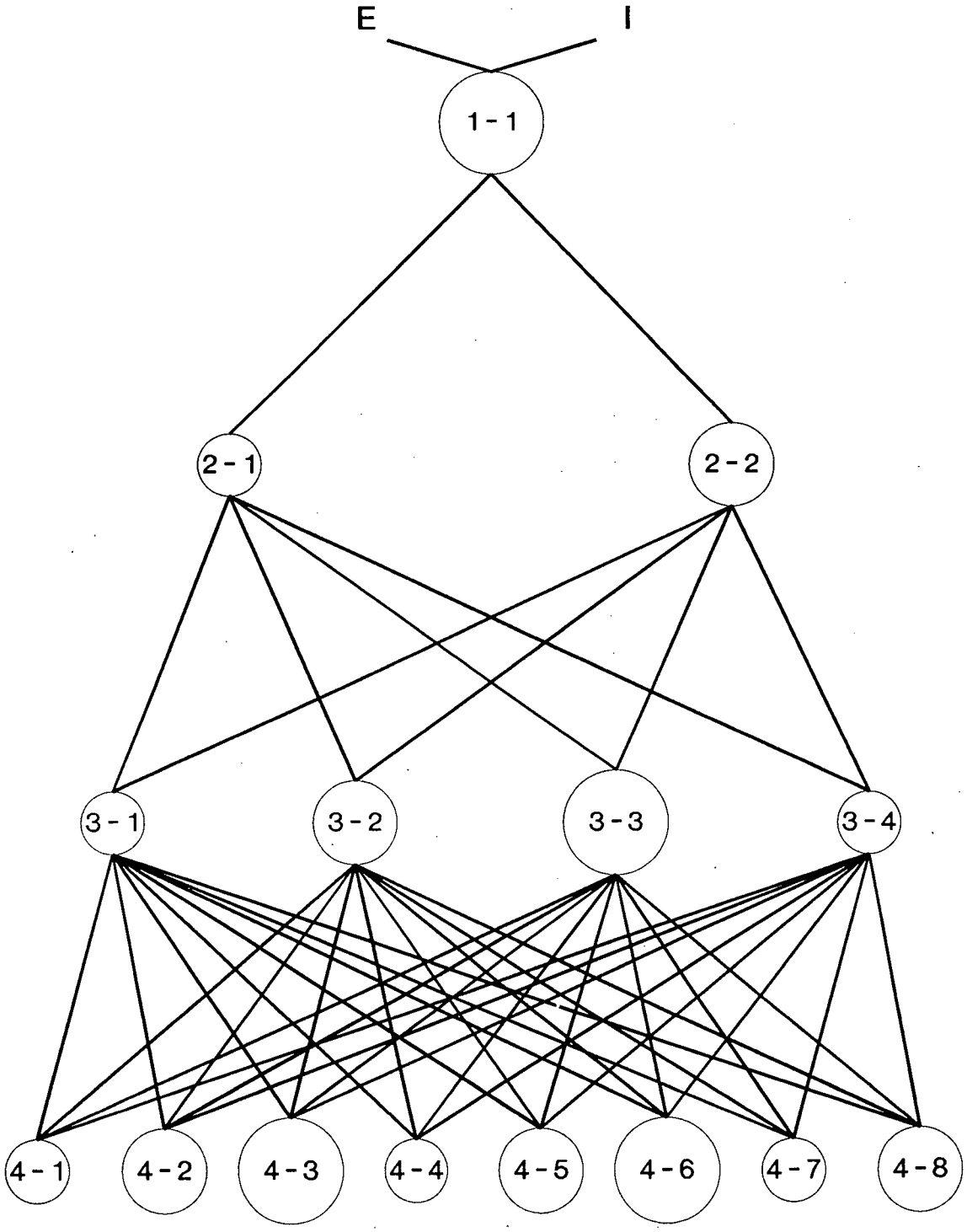
The spatial-temporal sum of each muscle fibre action potential that attaches to a motor neuron constitutes a motor unit action potential. Surface EMG is the spatial-temporal sum of each motor unit action potential or, alternatively, the spatial-temporal sum of each motor neuron to discharge as detected by the surface electrodes. This pattern of motor discharge (i.e., the order and frequency) is dependent on the level of synaptic drive to reach the motor pools. To recap, small (slow) motor neurons discharge early and at low frequency because of low synaptic threshold and large (fast) motor neurons discharge late and at high frequency because of high synaptic threshold (Hennemann, 1957). Small motor neurons attach to slow twitch muscle and large motor neurons attach to fast twitch muscle. Increased recruitment of motor neurons and, later, increased firing rates lead to increased force of muscle contraction.

We have posited elsewhere (McGarry & Franks, 1997) that the synaptic input to the motor pools, as reflected in the EMG onset, is the net effect of excitatory-inhibitory processes that compete at all times before their arrival at the motor pools. Hence the varying inputs to the motor pools, as a result of preceding excitatory-inhibitory interaction, leads to varying discharge rates and, consequently, varying types of EMG onset. We have shown, albeit in brief form, that some excitatory-inhibitory interaction at the level of a neuron in a hierarchy of neurons can capture the stochastic independence of the race model (McGarry & Franks, in review-c) and, at the same time, yield graded EMG onsets. We seek to analyse this result further by developing various computer models and comparing the generated data with the empirical data reported thus far.

Following McGarry and Franks (in review-a), we posit a connectionist account in which neurons, excitatory and inhibitory, interact with each other at all levels in the hierarchy. We make no claim that the neuron is the fundamental unit of control, though we do consider it to be the fundamental unit through which such control is expressed. We retain the binary architecture of McGarry and Franks (in review-a) in which each neuron (or cell) propagates in stochastic fashion throughout the structure in random time (see Figure 3.4). Once more, we liken the architecture as comprising two families of cells which we refer to as the E-family (excitation) and the I-family (inhibition) respectively whose first parent (i.e., E : 1 - 1, I : 1 - 1) is sibling related. In each family, the collective cells within a level constitute a generation and each cell within a generation constitutes a member within a sibling order indexed left to right. Thus, each cell within the architecture is uniquely accessed by its membership to family (f), generation (g) and sibling order (s). We retain the familial references of parent, child, uncle (or aunt), nephew (or niece), sibling and cousin as used in the earlier experiment. (Like cousins occupy the same generation - sibling reference in each family i.e., E : x - y, I : x - y). For further detail, see Section 3.3.8 Excitatory-inhibitory interaction versus a race between excitatory (go) and inhibitory (stop) processes.

Once again, each cell in a family is limited to a single synapse (input) and to a single axon (output) that connects each cell in a generation to each cell in the preceding and subsequent generation. Solid lines denote excitatory pathways while dashed lines denote inhibitory pathways. Inhibitory pathways project uni-laterally from the I-family to the E-family in an avuncular relation (see Figure 3.4). The E-family and the I-family are superimposed in a single structure in Figure 4.3 for ease of presentation. This hierarchy of control is the formal equivalent of Figure 3.4 except for the introduction of neurons of varying size (small, intermediate, large), as well as the addition, in some cases (see later), of inhibitory pathways that project uni-laterally from cousin (I : x - y) to like cousin (E : x - y). The effect of varying the size of a neuron is to vary its threshold and discharge (see later). E-excitation propagates in the E-family and I-excitation propagates in the I-family each in stochastic fashion on presentation of its own signal. The effect of increasing I-excitation in the

Figure 4.3 Hypothesized hierarchical structure of control for producing and stopping a speeded voluntary action updated (see Figure 3.4). The structure - a superimposition of the E-family and the I-family - contains only three levels for ease of presentation. Neurons of three sizes (small, intermediate, large) are distributed throughout the hierarchy. Each neuron excites or inhibits the next generation, given its predisposition, as a function of its size (see text). Once again, the processes of excitation and inhibition propagate the hierarchy in random fashion and the pattern of discharge of the motor neurons (last generation, E-family) determines the various EMG onsets. Note. From T. McGarry and I.M. Franks (in review-b).



I-family is to exert increasing inhibitory influence (I-inhibition) on the E-family by virtue of the uni-lateral pathways that project from the I-family to the E-family. The result is excitatory-inhibitory interaction in the E-family at all generations.

The general rules of operation for Figure 4.3 are as follows. The present status and the future status of each cell is updated on each time step. The unit of time is arbitrary and increases in a positive direction. Units of excitation and inhibition are also arbitrary. E-excitation is indexed by positive sign whereas I-excitation and E-inhibition are indexed by negative sign. That the I-excitation is signed negative is inconsequential to its own excitation and simply affords easier tracking within the computer program. Each cell in the I-family performs double duty in that it excites each child and it inhibits each nephew (or niece). This duality is achieved by "carbon copying" the I-excitation pulse that is dispatched in random time to a child as the I-inhibition pulse that is dispatched in the same random time to the like nephew (or niece).

The status of a cell decays linearly in time towards a nil value that represents a neutral, or resting, state for that cell. The effect of any cell on any other connected cell is tallied as the net algebraic sum in the receiving cell at any instant. In other words, the status of a cell is the sum of its present status and the discharge pulses (excitatory and or inhibitory) that are received at that time. If the status of a cell equals or exceeds threshold, then a discharge pulse, equal to threshold, is generated by the parent and sent to each child in random time. Small cells have low threshold, intermediate cells have intermediate threshold and large cells have high threshold, thus reflecting the physiological properties of a nerve cell as it relates to size (Hennemann, 1957).

Each parent enters refractory state on the dispatch of a discharge pulse to each child. The refractory state of a cell is a state of inactivity for an interval of time, the inverse of its discharge frequency, in which discharge pulses cannot be traded (i.e., received or sent). Small cells have low discharge frequency, intermediate cells have intermediate discharge frequency and large cells have high discharge frequency. Each cell can receive a discharge pulse providing that the cell is not already in a refractory state at the time of its receipt. If the cell is in refractory state, then the effect of the discharge pulse on that cell is forfeited, regardless of whether the discharge be excitatory or

inhibitory. (In fact, how a cell that is in refractory state handles the incoming discharge of a particular pulse, be it excitatory or inhibitory, is specified in the rules of interaction for that model as described below.) If the cell is not in a refractory state, then the status of the cell is updated (i.e., the discharge pulse is added to the present status of that cell). In this way, the architecture self-excites and self-inhibits as increasing cells are recruited within each generation. The process ends when each sibling in the last generation (i.e., motor neuron) is excited or inhibited.

The uni-lateral projections available (Figure 3.4) were invoked in varying degrees in order to examine the effect of independence and non-independence on the various outcomes. Five models (A through E) with varying degrees of excitatory-inhibitory interaction were used in this analysis. The specific rules of interaction, as they pertain to each model, were as follows:

A. Independence. There are no projections from the I-family to the E-family. (See the Appendix for a much abridged walk-through example.)

B. Semi-independence. There are like cousin lateral projections only from the I-family to the E-family. These like cousin projections might be thought of as sovereign as the inhibition cell exerts inhibitory influence on its like cousin preventing further excitation. The excitation refractory state of this cousin is not respected. (Note. Like cousin uni-lateral projections are not detailed in Figure 3.4.)

C. Semi-independence. There are avuncular projections only from the I-family to the E-family (see Figure 3.4). The inhibition cell exerts inhibitory influences on its nephews (nieces). The excitation refractory state of each nephew (niece) is respected.

D. Semi-independence. There are like cousin projections as well as avuncular projections from the I-family to the E-family (see Figure 3.4). The like cousin projections might be thought of as sovereign as the inhibition cell exerts inhibitory influence on its like cousin preventing further excitation. The excitation refractory state of this cousin is not respected. In addition, the inhibition cell exerts inhibitory influences on its nephews (nieces). The excitation refractory state of each nephew (niece) is respected. (Note. Like cousin uni-lateral projections are not detailed in Figure 3.4.)

E. Dependence. Each cell is updated in common by the E-family and the I-family. That is, the E-family exerts excitatory influence and the I-family exerts inhibitory influence on a common cell. Excitation and inhibition refractory states are respected. (This type of structure is most easily imaged as a single structure in which each cell possesses excitatory-inhibitory duality. While one might object on the grounds of physiology, it is, we think, equal to each family exerting mutual inhibition on the other by way of bi-lateral avuncular projections.)

Imagine a single structure (see Figure 4.3) in which an E-excitation pulse (E) triggers a green light in the body of the first cell to discharge (i.e., E : 1 - 1) and that each cell to discharge remains green until its refractory state is lifted. In time, a random sequence of green lights which reflects the go process (i.e., E-excitation) propagates the hierarchy. Imagine the same single structure in which an I-excitation pulse (I) triggers a red light in the body of the first cell to discharge (i.e., I : 1 - 1). Once again, the discharged cell remains red until its refractory state is lifted and, in time, a random sequence of red lights which reflects the stop process (i.e., I-excitation) propagates the hierarchy. Importantly, the I-excitation in the I-family is observed as I-inhibition in the E-family by way of the uni-lateral projections and the dual nature of the discharge properties. Thus, if E-excitation and I-inhibition each begin to propagate the E-family on presentation of their own signal, separated by an SOA, then the effect of I-inhibition on E-excitation, if any, is observed at the level of motor discharge (i.e., the last generation, E-family).

The first motor neuron to light green marks the onset of motor discharge as indexed in EMG. In fact, the time-order sequencing of these discharges determine the type of EMG onsets observed at onset (see later). The time interval from the first E-excitation pulse (E : 1 - 1) to the first motor neuron to discharge (E : g - 1 through E : g - $\underline{s}$) is indexed as Go-EMG. Likewise, the time interval from the first I-excitation pulse (I : 1 - 1) to the first motor neuron to light red (I : g - 1 to I : g - $\underline{s}$, or E : g - 1 to E : g - $\underline{s}$) is indexed as Stop-TEMG. On occasion, a Go-EMG and Stop-EMG will register on the same trial by virtue of vacant states within the motor neurons ($\underline{s} = 2^{g-1}$) available for discharge. These occasions constitute some form of EMG onset, regardless of the colour of the first motor neuron to discharge.

In passing, we note that the process of stochastic propagation to threshold as outlined above is in keeping with Hanes and Schall's (1996) position, taken from an analysis of single cell activities in the frontal eye field (frontal cortex) of the rhesus monkey (Macaca mulatta). These authors favoured the stochastic sampling of variable rate as opposed to that of variable threshold in order to explain the variance in reaction (go) latencies. Hence, short latencies and long latencies result from high rates and low rates of growth to a constant threshold, rather than constant rates of growth to low threshold and high threshold. To this, we add that the rate at which the go process crosses threshold would explain the gradations of EMG onset. This would predict that sub-maximal rates to threshold would yield sub-maximal EMG onsets at longer latencies, whereas maximal rates to threshold would yield maximal EMG onsets at shorter latencies (c.f., the latency relations detailed in Figure 2.5). This reasoning is consistent with the inverse relation that exists between the latency reaction and the average velocity of that action (Falkenberg & Newell, 1980).

4.3.4 On latencies and probabilities of stopping a voluntary action

Since the time for each pulse to E-excite and to I-excite (I-inhibit) the next generation is random, the time for each process to traverse the hierarchy is random also. In addition, since a specific result is produced from each starting seed within the computer generated pseudo-random stream, $\underline{r}$ starting seeds ($\underline{r}_1 = 1000$) were used to provide descriptive statistics for Go-EMG and Stop-EMG by allowing each process to traverse the hierarchy ($\underline{b} = 2$, $\underline{g} = 9$) in the absence of the other. Using the initial conditions for time-to-excite (25 ± 10) and time-to-inhibit (20 ± 10) resulted in the following distributions parenthesised ($\overline{X}_{\text{Go-EMG}} = 219.82$, $SD_{\text{Go-EMG}} = 73.38$, $\overline{X}_{\text{Stop-EMG}} = 127.10$, $SD_{\text{Stop-EMG}} = 34.41$). (Note. Time-to-excite and time-to-inhibit are linear distributions scaled to yield integer values selected at random from 15 through 35 and from 10 through 30 respectively. Units of time are arbitrary.) We used SOAs from 10 through 200 in steps of 10 and contrasted the outcome probabilities obtained from each model ($\underline{r}_1 = 1000$, A through E) against the predicted probabilities obtained from the race model. (For further detail, see Section 3.3.7 Excitatory-inhibitory interaction versus a race between excitatory [go] and inhibitory [stop] processes).

Table 4.4 details the probabilities of an action (i.e., motor discharge) as a function of SOA for the race model and the five models described in this study (i.e., A through E). Since probability is bounded at 0 and 1, the results were subjected to Friedman's rank test for $\underline{k}$ correlated samples. This is a non-parametric test of significance that is closely related to a repeated measures ANOVA as applied to ranks instead of raw scores (Howell, 1997). Friedman's test is especially sensitive to differences in central tendency and, importantly for our purposes, eliminates SOA differences by virtue of the ranking procedure. The null hypothesis is that the scores for each treatment (model) are drawn from identical populations which, if true, would be expected to yield within SOA rankings that are randomly distributed. The ranking of raw scores within each SOA are detailed in parentheses (Table 4.4).

The results, $\chi^2_F[5, \underline{N} = 120] = 73.664$, $\underline{p} < .001$, reject the null hypothesis that the scores from each model sample from identical populations, a result that is repeated, $\chi^2_F[4, \underline{N} = 100] = 43.130$, $\underline{p} < .001$, if model E is excluded from the analysis on the grounds of extremity. Three points are of interest from a follow-up visual analysis of the data. First, an account in which the stop process inhibits the E-family at the same time and in the same way that it excites the I-family (model C) best approximates the latency relations between the go process, the stop process, the SOA and the outcome probability. Second, an account in which the go process and the stop process share a common state (model E) produces an unsatisfactory account of the data. In other words, an independent (functional) go process is not a necessary condition, whereas an independent (functional) stop process is a necessary condition, if the latency relations as expressed in the race model are to be satisfied. Third, each model underestimates the response probability at the extreme low end of the SOA spectrum, a result that derives presumably from the excitation-inhibition processes beginning with very few cells being recruited in the early stages. This situation benefits the stopping process at the expense of the go process, although this advantage is mitigated increasingly as more neurons become active. The consequent run-away effect tends to mimic independent (stochastic) processes.

Table 4.4 Predictions of the probabilities of an initiated action from the race model as contrasted with the probabilities obtained from models A through E. The probabilities are detailed as a function of the time from presentation of the go signal to presentation of the stop signal (SOA).

			Model									
	RM		A		B		C		D		E	
SOA	p	(R)	p	(R)	p	(R)	p	(R)	p	(R)	p	(R)
10	.130	(6)	.092	(5)	.030	(2.5)	.042	(4)	.030	(2.5)	.000	(1)
20	.161	(6)	.149	(5)	.120	(3)	.128	(4)	.118	(2)	.002	(1)
30	.196	(2)	.245	(6)	.221	(4)	.226	(5)	.218	(3)	.027	(1)
40	.236	(2)	.308	(6)	.292	(3.5)	.295	(5)	.292	(3.5)	.105	(1)
50	.280	(2)	.368	(6)	.362	(4)	.363	(5)	.359	(3)	.228	(1)
60	.328	(2)	.374	(6)	.372	(3.5)	.373	(5)	.372	(3.5)	.327	(1)
70	.378	(2)	.441	(6)	.395	(3.5)	.414	(5)	.395	(3.5)	.377	(1)
80	.431	(5)	.448	(6)	.410	(3)	.435	(4)	.409	(2)	.384	(1)
90	.485	(4)	.492	(6)	.472	(3)	.486	(5)	.471	(2)	.397	(1)
100	.539	(6)	.528	(5)	.518	(4)	.517	(3)	.515	(2)	.413	(1)
110	.593	(4.5)	.602	(6)	.581	(3)	.593	(4.5)	.580	(2)	.475	(1)
120	.645	(5)	.648	(6)	.637	(3)	.641	(4)	.636	(2)	.576	(1)
130	.694	(6)	.677	(5)	.664	(3)	.667	(4)	.661	(2)	.627	(1)
140	.740	(6)	.717	(5)	.687	(2.5)	.699	(4)	.687	(2.5)	.661	(1)
150	.782	(6)	.751	(5)	.735	(2.5)	.746	(4)	.735	(2.5)	.674	(1)
160	.820	(6)	.807	(5)	.794	(2.5)	.801	(4)	.794	(2.5)	.686	(1)
170	.854	(6)	.844	(5)	.834	(3)	.837	(4)	.833	(2)	.720	(1)
180	.883	(3)	.892	(6)	.883	(3)	.884	(5)	.883	(3)	.785	(1)
190	.908	(2)	.917	(6)	.914	(5)	.913	(4)	.911	(3)	.842	(1)
200	.928	(6)	.926	(5)	.921	(2.5)	.922	(4)	.921	(2.5)	.889	(1)
Total		(87.5)		(111)		(64)		(86.5)		(51)		(20)

Note. RM = race model. p = probability of an action. (R) = rank order within SOA.

Interestingly, the results from this study shows that a non-independent (functional) go process best captures the stochastic independence that the race model expresses (see Table 4.4). This relation is further detailed in Table 4.5, which provides the frequency distribution of the various response types - full, interrupted, partial - as a function of SOA for model A and model C. (See later as to how the full, interrupted and partial responses were classified from the excitation histories of the respective models.) The combined latency distributions of each of the full, interrupted and partial responses (i.e., go latencies) are also presented as a function of SOA for model A and model C. The latencies are included to show that the principal relations expressed in the race between stochastic processes, namely that of shorter go latencies at shorter SOAs hold for model A and for model C also. These results replicate those of the earlier study (McGarry & Franks, in review-a) and show that the relations as described in the race model can be produced from semi non-independent processes (i.e., the go process is non-independent while the stop process is independent).

We now investigate whether semi non-independent processes can account for the distribution of response types as a function of SOA (Figure 4.1, Table 4.3) as well as their characteristic graded EMG onsets (Table 4.1). We will examine each issue in turn and contrast the results from semi non-independent processes (Model C) with those obtained from independent processes (Model A).

The full, interrupted and partial response data were subjected to the same statistical procedure (not shown) as the empirical data for segregating the responses into low bins and high bins respectively (see Section 4.3.2 Evidence for graded movement as a function of the latency relations between the go process and the stop process, for further detail). The results (from Table 4.5) for model A yielded significantly more full responses than interrupted responses at the high end of the SOA spectrum, χ^2 (1, $\underline{N}$ = 10,896) = 162.615, $\underline{p}$ < .001, and, also, significantly more interrupted responses than full responses at the low end of the SOA spectrum, χ^2 (1, $\underline{N}$ = 10,896) = 179.317, $\underline{p}$ < .001. However, this pattern was not repeated for the interrupted responses and the partial responses. Instead, Model A yielded no significant difference between the interrupted responses and the partial responses at the high end of the SOA spectrum, χ^2(1, $\underline{N}$ = 800) = 1.096, $\underline{p}$ = .295, and

Table 4.5 Frequency distribution of the full, interrupted and partial responses as well as the respective go latency distributions as a function of the time between presentation of the go signal and the stop signal (SOA) for model A and model C.

	Model A						Model C					
				Go						Go		
SOA	F	I	P	n	$\overline{X}$	SD	F	I	P	n	$\overline{X}$	SD
10	67	10	15	92	144.0	11.3	26	7	9	42	138.7	8.8
20	86	33	30	149	142.4	9.2	76	32	20	128	142.3	9.7
30	147	55	43	245	143.7	8.4	146	54	26	226	143.3	8.4
40	219	52	37	308	144.1	8.1	218	52	25	295	143.8	8.1
50	318	34	16	368	146.0	9.4	319	33	11	363	145.7	8.7
60	355	14	5	374	147.1	9.7	355	14	4	373	147.1	9.7
70	428	11	2	441	155.4	24.0	402	10	2	414	151.5	18.4
80	434	5	9	448	157.4	25.8	424	6	5	435	155.5	23.8
90	454	19	19	492	162.1	29.0	453	20	13	486	161.5	28.6
100	470	37	21	528	165.8	31.0	471	38	8	517	164.6	30.3
110	530	47	25	602	172.6	34.6	532	46	15	593	171.7	34.1
120	603	27	18	648	176.0	35.6	601	30	10	641	175.5	35.4
130	657	12	8	677	179.5	38.4	649	13	5	667	178.3	37.5
140	701	7	9	717	184.0	42.4	682	13	4	699	181.7	40.3
150	724	12	15	751	187.8	45.0	724	12	10	746	187.3	44.7
160	758	29	20	807	193.9	48.7	760	28	13	801	193.3	48.5
170	804	28	12	844	197.9	51.4	802	27	8	837	197.2	50.9
180	852	24	16	892	202.8	53.9	851	24	9	884	202.0	53.5
190	899	10	8	917	205.4	55.4	898	9	6	913	204.9	55.1
200	920	4	2	926	206.4	56.1	915	7	-	922	205.9	55.6
Σ	10426	470	330				10304	475	203			

F = full. I = interrupted. P = partial. n = number of observations. $\overline{X}$ = mean. SD = standard deviation. Σ = Total.

no significant difference between the partial responses and the interrupted responses at the low end of the SOA spectrum either, $\chi^2 = (1, \underline{N} = 800) = 1.096$, $\underline{p} = .295$. The results for model C, on the other hand, yielded a different pattern of behaviour. Significantly more full responses than interrupted responses gathered at the high end of the SOA spectrum, χ^2 (1, $\underline{N}$ =10,779) = 157.654, $\underline{p} < .001$, as well as significantly more interrupted responses than full responses at the low end of the SOA spectrum, χ^2 (1, $\underline{N}$ =10,779) = 171.341, $\underline{p} < .001$. Likewise, significantly more interrupted responses than partial responses gathered at the high end of the SOA spectrum, χ^2 (1, $\underline{N}$ =678) = 4.257, $\underline{p} = .039$, although the reverse analysis - that of more partial responses than interrupted responses at the low end of the SOA spectrum - failed to reach significance, χ^2 (1, $\underline{N}$ =678) = 2.389, $\underline{p} = .122$.

In short, these results demonstrated that a semi non-independent go process best approximates the latency relations detailed in Figure 4.1. This type of excitatory-inhibitory interaction has also been hypothesized to explain the sub-maximal EMG onsets (McGarry & Franks, 1997, in review-a). We therefore investigated, from the excitation histories of motor neuron discharge, the types of EMG onsets as produced from semi non-independent processes (model C) as contrasted with those from independent processes (model A). First, we explain how EMG patterns were generated from the history of motor neuron discharge.

4.3.5 Generation of EMG from the pattern of motor neuron discharge

In brief, we assume that each motor neuron discharge generates a motor unit action potential whose amplitude and frequency are contingent on the size of the cell. Small cells (size = 1) generate action potentials of small amplitude and large period (or low frequency), intermediate cells (size = 2) generate action potentials of intermediate amplitude and intermediate period (or intermediate frequency) and large cells (size = 3) generate action potentials of large amplitude and small period (or high frequency). The sign of the action potential (positive, negative) is contingent on its direction of travel along the muscle fibre with reference to the surface electrodes. For example, an action potential that travels from left to right along a muscle fibre might register a positive sign, or vice

versa, as it approaches passing under the surface electrodes. The sign is reversed as the action potential travels away from the surface electrodes after passing under them.

If, for convenience, a sine wave approximates an action potential, then an EMG profile can be generated from the spatial-temporal sum of each sine wave as specified in the history of motor discharge. For example, Figure 4.4 presents three motor neurons (size = 1, size = 2 and size = 3) that discharge at various times (t_1, t_2 and t_3, where $t_1 < t_2 < t_3$). Since we have no information with respect to the location of the attachment of each motor neuron to its muscle fibres, and therefore no information with regard to the sign of its action potential, we assume the sign to be equi-probable (i.e., $p = .500$). Each sign combination of the three action potentials ($2^3 = 8$) is presented in panels a-h. In each panel, the upper trace details the three action potentials, the middle trace details the surface EMG wave form generated from the three action potentials added together on a common time base, and the lower trace details the same EMG trace on a compressed time scale, centred. In this way, an EMG trace was generated from the excitation histories of motor neuron discharge.

Two possibilities present themselves for generating an EMG trace from a given excitation history of motor discharge. The first option is to obtain an average EMG trace from the repeat generation of a given history of motor discharge. If q motor neurons discharge then 2^q EMG traces are possible but only 2^{q-1} EMG traces are produced when the rectified-average EMG measure is taken. (This is because the EMG pattern is mirrored for reverse sign combinations. See for example the left panels and the right panels in Figure 4.4.). We are not aware of the minimum repeat generation necessary in order to obtain a satisfactory EMG average for any given q, though we estimate that it is likely in the order of 2^q, an order of magnitude that quickly becomes unwieldy as q increases from 1 to 128. The second option is to assign arbitrarily each action potential to a positive (or negative) sign, thus removing the need for repeat generation of the EMG traces. While this option is unrealistic in practice, it best meets our purpose for EMG comparison between the various response types (i.e., various excitation histories) because it removes any confounds of approximation to averages. We therefore used the second option to compare EMG onsets between various response types.

Figure 4.4 Example generation of surface EMG from temporal-spatial sum of three action potentials, each of which reflect single motor neuron discharges of varying size. The first trace in panel A details the three separate action potentials, one for each size of motor neuron (small, intermediate, large), that discharge at different times. The second trace in panel A details the spatial-temporal sum of these three action potentials and the third trace in panel A details the same spatial-temporal sum on a compressed time scale. Panels A-H show the eight different sign combinations from the three motor neurons to discharge. In each row, the right panel is the mirror image of the left panel by virtue of reverse sign.

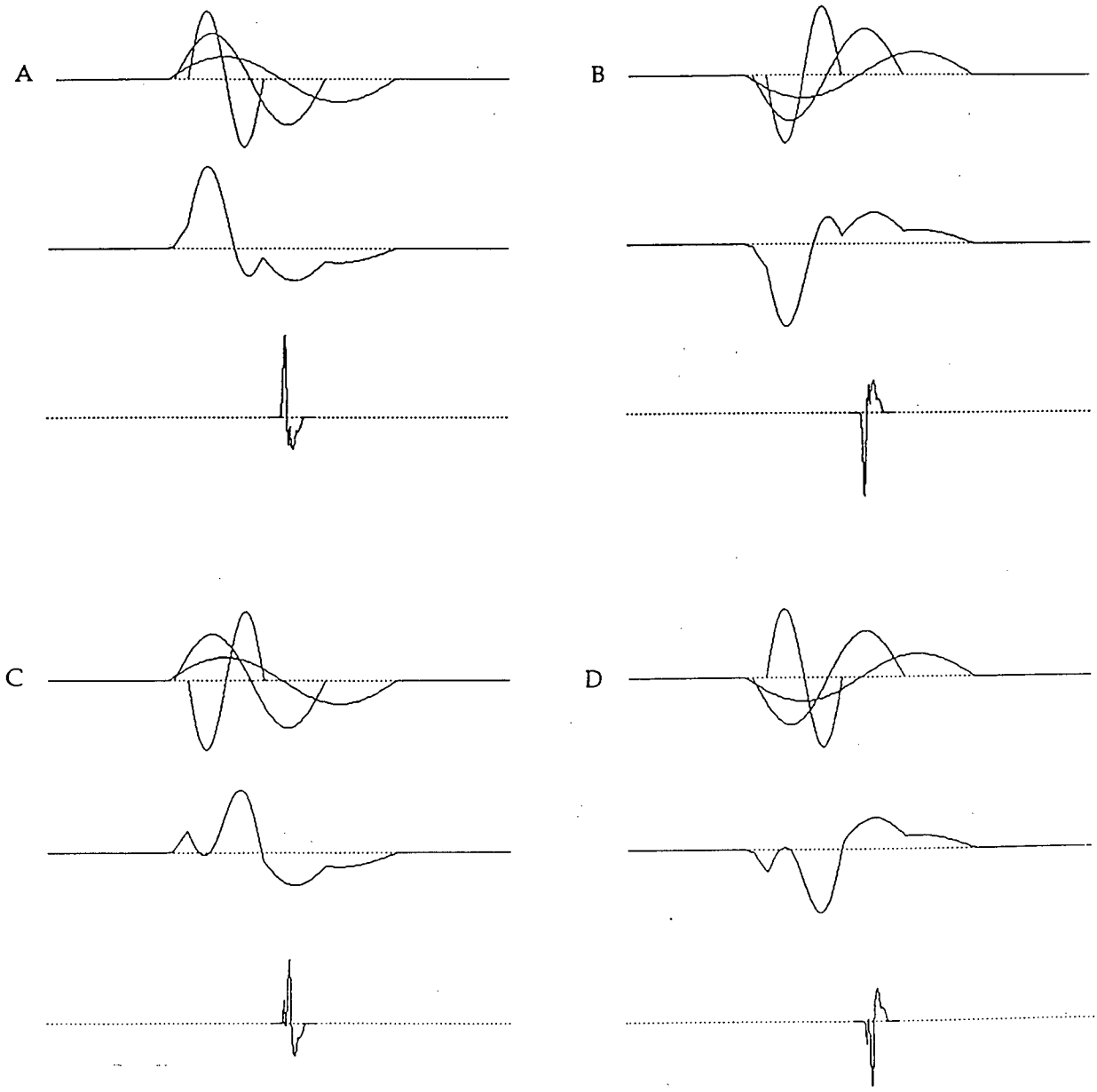
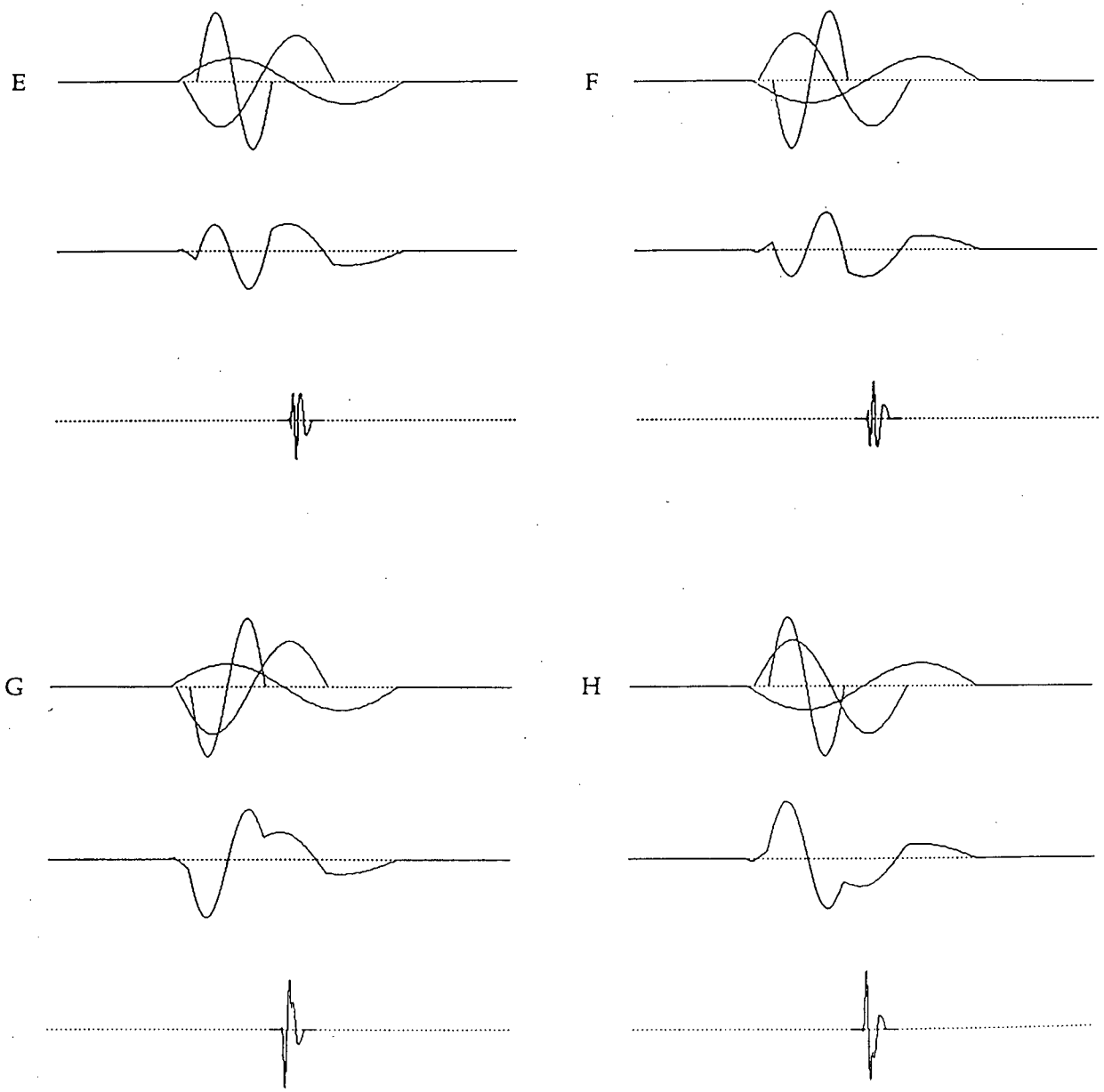


Figure 4.4 continued.



4.3.6 On EMG onsets when stopping a voluntary action

We (McGarry & Franks, 1997) have suggested that the pattern of motor neuron discharge, and hence the EMG onsets, are a result of earlier excitatory-inhibitory interaction between the go process and the stop process. In this view, full responses are produced with no effect of the stop process on the go process at the motor pool, as indexed in EMG onset. Similarly, interrupted responses are also produced with no effect of the stop process on the go process at the motor pool, although the stop process rapidly takes effect some time thereafter so yielding a response that is interrupted at various times in the course of its usual activity. The results from this study, however, suggest that the stop process indeed affects the go process before the motor pool for interrupted responses, likely because of the different classification systems used to identify the interrupted responses between the two studies. Nonetheless, the findings from this study are consistent with a continuum of reductions from maximal EMG onsets through zero EMG onsets as the stop process acts on the go process at all times from lag to bulk to lead.[10] That is, in order to stop an earlier intended maximal speeded action, the excitation lag is suppressed first, the excitation bulk is suppressed next, and the excitation lead is suppressed last. (Note. The stop process possesses its own lead, bulk and lag activity also.) The degree of suppression, or near suppression, of the excitation bulk would then explain how interrupted responses might or might not produce the same EMG onsets as that for full responses.

Regardless, the task at hand is to investigate whether the various response types as generated differ in their EMG onsets. The various response types were categorised on the basis of their motor discharge history (1 through s) from Model A and Model C. Specifically, a response was considered as: full, when the first motor neuron to discharge was excited and all subsequent motor neurons were excited; interrupted, when the first motor neuron to discharge was excited and some subsequent motor neurons were inhibited; partial, when the first motor neuron to discharge was

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[10] We introduce the concept of lead, bulk and lag activity by extending the analogy of a race in which the flow of entrants across the finish line occurs at varying rates from minimal (i.e., lead) through to maximal (i.e., bulk, or peak) through to minimal (i.e., lag).

inhibited and some subsequent motor neurons were excited; and stopped, when the first motor neuron to discharge was inhibited and all subsequent motor neurons were inhibited. (Note. No-stop responses were classified in the same way as full responses for purposes of comparison.)

The mean-rectified EMG data for each response type for SOA 100 for Model A (upper panels) and for Model C (lower panel) are detailed in Figure 4.5. (The same data are presented in Table 4.6.) Stopped responses produced no EMG and are not reported. The EMG data show that the no-stop responses and the full responses share like traces. The interrupted responses, however, differ from the no-stop responses and the full responses in their later rise of EMG from onset (left panels). We synchronised the response types to their rise in EMG onsets by moving the interrupted responses one time step back in relation to the other response types (right panels). These data better detail the cut-off from the usual EMG activity that characterises the interrupted responses, as first reported by McGarry and Franks (1997) using the same method of comparison. This latter representation of the data better demonstrates the difference in the level of interruption between Model A and Model C.

Model A (Figure 4.5, upper panel right), unlike the empirical data, yielded little cut-off in its mean-rectified EMG trace for the interrupted responses whereas Model C (Figure 4.5, lower panel right), like the empirical data, yielded earlier cut-off in its mean-rectified EMG trace for the interrupted responses. The probable reason for the earlier cut-off in Model C is the level of suppression of the excitation bulk. The excitation bulk is not affected by the stop process in Model A because of the independence of the go process and, hence, that process cannot be stopped readily once excitation lead takes effect at the motor pool. This result runs counter to empirical observation. On the other hand, the excitation bulk is reduced somewhat by the stop process in Model C because the go process is not independent of the stop process and it is this effect, presumably, that allows for the subsequent interruption in excitatory activity following lead excitation at the motor pool. The result is interrupted responses that better approximate the empirical data (c.f., Figure 3.1).

This study indicates that the EMG onsets for the interrupted responses are in fact intermediate responses between that for the full responses and that for the partial responses (see Table 4.1). In other words, full responses are steeper in their EMG onsets than are interrupted responses which, in

Figure 4.5 Linear EMG envelopes of the mean-rectified agonist EMG for the no-stop, full, interrupted and partial responses generated from the pattern of motor discharge (see Figure 4.4) for model A (upper panels) and model C (lower panels). Each response is time-synchronised to EMG onset (left panels). The interrupted response (left panels) is synchronised one time step back (right panels) for comparison (see text). Units of time and EMG are arbitrary.

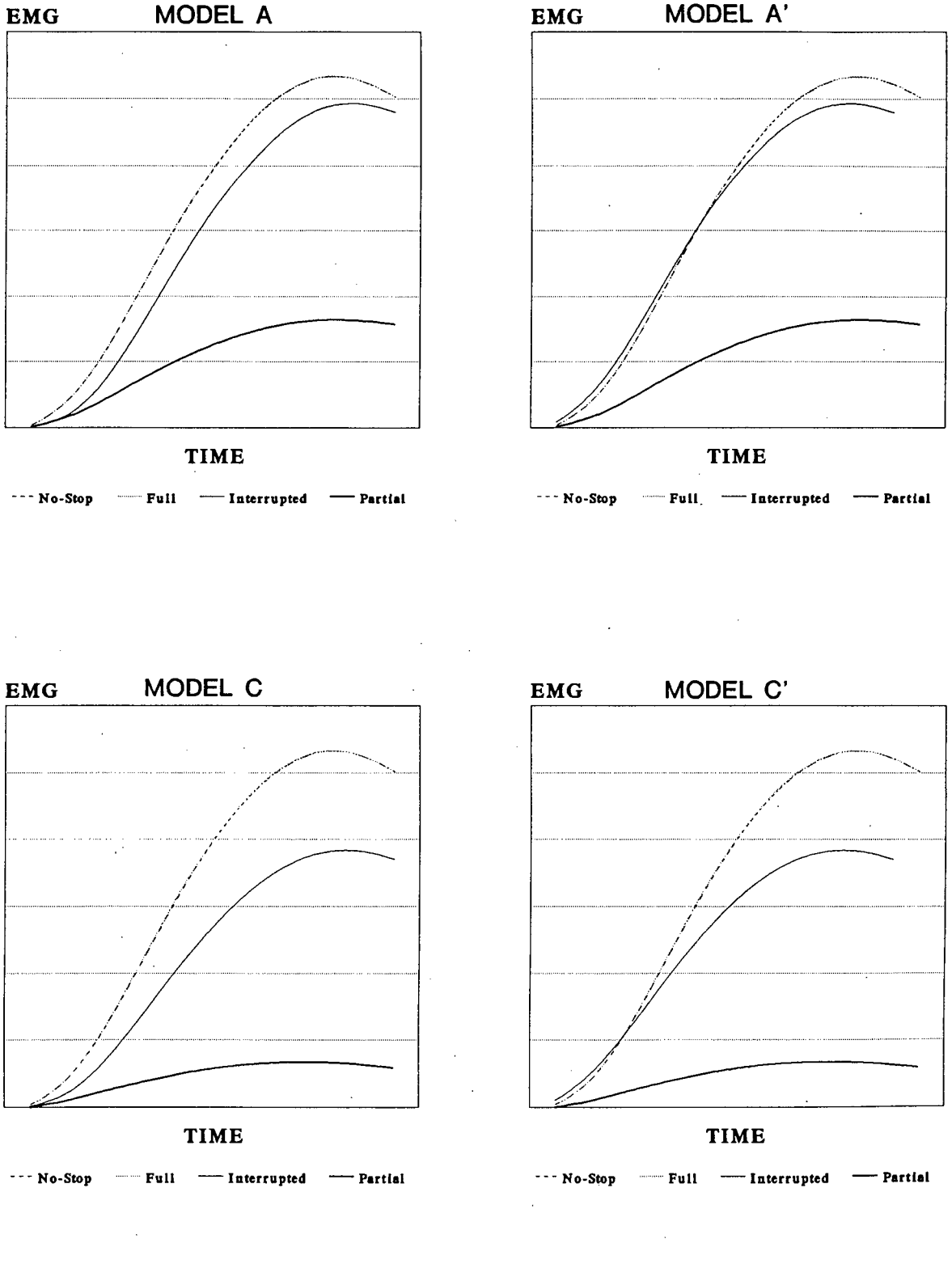

Table 4.6 Generated mean-rectified EMG data for the no-stop, full, interrupted and partial responses from Model A and Model C.

	Model A								Model C					
	No-Stop		Full		Interrupted		Partial		Full		Interrupted		Partial	
Time	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD	$\overline{X}$	SD
1	76	118	79	125	24	33	35	78	75	118	32	52	11	8
2	464	490	473	492	172	203	206	262	472	486	204	291	119	97
3	1313	984	1321	983	666	600	507	605	1330	980	689	710	320	283
4	2545	1381	2555	1378	1617	1107	939	1060	2574	1375	1485	1188	519	471
5	3979	1582	4006	1551	2862	1527	1398	1566	4028	1544	2473	1632	710	652
6	5444	1587	5478	1544	4223	1789	1826	2062	5501	1533	3528	2016	883	820
7	6815	1468	6850	1422	5543	1963	2214	2517	6873	1406	4538	2365	1031	964
8	8028	1286	8060	1246	6748	2082	2552	2913	8082	1224	5447	2685	1156	1087
9	9045	1065	9073	1033	7798	2170	2835	3240	9092	1009	6231	2968	1254	1184
10	9831	814	9854	789	8657	2233	3055	3487	9869	765	6859	3196	1316	1250
11	10366	550	10385	531	9297	2259	3201	3644	10395	508	7316	3363	1345	1284
12	10651	309	10665	300	9708	2256	3285	3712	10671	277	7595	3461	1348	1293
13	10683	220	10692	225	9890	2222	3299	3694	10692	211	7698	3490	1316	1270
14	10476	384	10480	389	9849	2159	3255	3594	10475	384	7628	3448	1259	1222
15	10042	609	10042	609	9593	2065	3152	3416	10032	603	7396	3342	1176	1153
N*	10		10		10		10		10		10		10	
$\overline{\overline{X}}$	4753.9		4775.1		3831.0		1556.6		4789.6		3148.7		731.9	

Response Type (Model A)

	No-Stop	Full	Interrupted	Partial
No-Stop	-	21.2	922.8	3197.2**
Full		-	944.0	3218.4**
Interrupted			-	2274.4**
Partial				

Response Type (Model C)

	No-Stop	Full	Interrupted	Partial
No-Stop	-	35.8	1605.1	4021.9**
Full		-	1640.9	4057.7**
Interrupted			-	2416.8**
Partial				

Note. $\overline{\overline{X}}$ = grand mean. N = number of time steps (first 10 steps only). $\overline{X}$ = mean. SD = standard deviation. *Small, intermediate and large motor neurons generate action potentials of periods 100, 50 and 33 yielding time-to-peaks from onset of 25, 12.5 and 8.3 units respectively. Hence first 10 data items would be unlikely to exceed the peak-rectified EMG on any single trial. * $p < .05$ (if difference > 1481.8 for Model A and difference > 1819.0 for Model C). ** $p < .01$ (if difference > 1840.0 for Model A and difference > 2258.7 for Model C). Units of time and EMG arbitrary.

turn, are steeper in their EMG onsets than are partial responses. Visual inspection of Figure 4.5 suggests a similar pattern to emerge for both model A and model C. This analysis is confirmed in a one-way (response type) repeated measures ANOVA of the first 10 data items (see Table 4.6). (The first 10 data items were used so as not to exceed the peak mean-rectified EMG.) The results for Model A, $F(3,27) = 15.629$, $p = < .001$, and Model C, $F(3,27) = 16.504$, $p < .001$, each yielded a significant difference between response types. Follow-up analyses using the studentized range statistic showed that in each case the difference lied between the partial responses and the interrupted responses, full responses and no-stop responses in the expected direction ($p < .01$) (Table 4.6). In addition, the EMG gradients for the interrupted responses tended to significance ($p > .05$)[11] as being less than that for the full responses and the no-stop responses (see Table 4.1). Thus, the trend to a continuum of EMG reductions as a result of stopping at various times is strongly supported from each model.

Taken together, the results from an independent stopping process and a non-independent go process that interacts at all times best explains the empirical data, as expressed in the latency relations as described in the race model (i.e., the go latency, the stop latency, the SOA and the outcome probability), as well as the graded EMG onsets that delineate the type of go response.

4.3.7 Rapprochement with the race model to explain the control of thought and action

In the race model, each process begins to race on presentation of its respective signal to a finishing post. This theory of control, which is based on a random utility model, assumes stochastic independence. The model that we favour in this study (model C) proceeds in accord with these principles, although stochastic independence is approximated *a posteriori* rather than presumed *a priori*. (That said, stochastic independence is retained at the level of the neuron.) For these reasons,

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[11] In Table 4.6 (Model C), the difference between the no-stop response and the interrupted response is 1605.1 units and the difference between the full response and the interrupted response is 1640.9 units. The requisite difference for significance at $p < .05$ is a value greater than 1819.0 units.

the computer model presented in this study is closely akin to the race model theory of control. Thus, the computer model might usefully be thought of as an extension of the processes that underlie the race model that compete at all times to the finish line, that is, to motor discharge. We think that our model makes two key contributions in this regard. First, the model shows that the latency relations that the race model describes as a function of SOA need not speak against competitive processes. Instead, a non-independent (functional) go process and an independent (functional) stop process can show the same pattern of data as that described in the race model, namely, faster go latencies that win the race with reduced probabilities at shorter SOAs (Table 4.4). Second, the graded EMG data are better explained from a reasoned account of excitatory-inhibitory interaction in line with various basic physiological principles, than can otherwise be advanced from the race model's account of a race between two discrete processes. We submit that this study is a promising first step towards meshing physiology and psychology in order to further aid our understanding of the mechanism of control that underlies the stopping of a voluntary action.

4.4 Summary

The results from this study support the interpretation that the empirical data observed when trying to stop an earlier intended maximal speeded action at various times are the result of non-independent excitatory-inhibitory processes that compete at all times up to motor discharge. We have shown, by way of computer analyses, that not only can stochastic independence be seemingly produced from an interactive account in which the stop process affects the go process at all times, but also that the complement of EMG onsets observed when stopping a maximal speeded action can be produced from the same account. Furthermore, these EMG onsets, as classified, lie on a continuum of response reductions. The important result of this study is that the empirical data - the latency relations as expressed in the race model and the EMG onsets or, alternatively, the synaptic drive as expressed in the motor neuron discharge patterns - are better explained from a non-independent go process that is subject to the effects of stopping at all times.

5 Experiment IV

5.1 On a point of no return in the control of thought and action revisited once again

Sub-maximal EMGs when trying to stop a maximal speeded voluntary action have been interpreted as evidence of an excitation process that is compromised by an inhibition process before EMG onset. This has led to the suggestion that the point of no return is phantom (McGarry & Franks, 1997) on account of the following reasoning. Sub-maximal EMG data betray a point upstream of EMG onset beyond which some EMG must be generated downstream, but, importantly, that same point fails to mark the onset of a final ballistic process if the to-be-produced EMG is subject to further effects of stopping. The alternative interpretation is that of a final ballistic process that receives sub-maximal input some time before EMG onset and that this input is faithfully preserved in sub-maximal EMG output. Thus, a final involuntary process cannot be satisfactorily ruled out on the observance of sub-maximal EMG records. The aim of this study is to analyse the validity of this alternative interpretation using the Hoffmann (H) reflex.

The H-reflex (i.e., the peak-to-peak EMG amplitude of an involuntary muscle twitch to an electrical stimulus) is facilitated in the short time window (30 ms - 80 ms) before EMG onset (Schieppati, 1987), within which a final ballistic process might be expected to locate. In a general sense, the H-reflex can be taken as a result of descending voluntary spinal control that readies the motor pools for the impending arrival of the efferent drive that produces EMG onset.

Frank (1986) analysed a simple (one choice) reaction time (SRT) task and a two choice reaction time (CRT) task, each preceded with a variable foreperiod, as well as a coincident-timing task (i.e., the timing of a response to an external event, or signal) in order to analyse the effects of latency and event (un)certainties on the onset of the H-reflex facilitation. SRT provides event certainty and temporal uncertainty, CRT provides event uncertainty and temporal uncertainty and coincident-timing provides event certainty and temporal certainty. The facilitation latencies of the H-reflexes showed the same onsets for SRT and CRT and earlier onsets for coincident-timing. These results indicated that the H-reflex onset is not time-locked to EMG onset and, instead, that it is modulated by preparatory set (i.e., the action of supra-spinal and spinal motor centres) (Frank, 1986). In

addition, the H-reflex is also facilitated in shorter time and with greater effect if the imperative signal to react is predictable rather than unpredictable (Manning & Hammond, 1990).

Of relevance to a point of no return is that the H-reflex can provide a window through which to examine the process of control upstream of EMG onset. We note at this juncture that motor pool excitability, which itself is affected by the release of certain neuro-modulators (Kiehn, 1991), is not the only factor that might contribute to the output of the motor pool (Capaday, 1997). For example, the pre-synaptic inhibition of Ia afferent terminals in the spinal cord (Capaday & Stein, 1987) and the input-output relations of various spinal neural circuits (Devanne, Lavoie & Capaday, 1997) would each bear upon motor discharge, as indexed in the H-reflex. We will return to the former point later.

Notwithstanding, we use the H-reflex as a general index of spinal control observed in real time before EMG onset. The facilitation of the H-reflex a short time before EMG onset lends a strong prediction in regard to stopping. On the one hand, a final ballistic process would be expected to shield the go process from stopping effects so yielding a preserved H-reflex. On the other hand, a final non-ballistic process would be expected to remain subject to further stopping effects so yielding an attenuated H-reflex. Thus, we reason that the presence or absence of a final ballistic process in the control of an action might be analysed directly from the effects of stopping, if any, on the H-reflex. In this study, we used a coincident-timing task (see later) to analyse the process of control for the hypothesized effects of stopping on the H-reflex shortly before EMG onset.

In like fashion, Hammond and Choo (1994) used the H-reflex taken from the soleus muscle in order to examine the mechanism of spinal control for evidence of a final ballistic process. Using a coincident-timing task, these authors reported a dissociation between the onset of facilitation of the H-reflex and the onset of EMG (i.e., the former did not obligate the latter), which they interpreted, in keeping with Osman et al. (1990), as evidence for a ballistic process very late in motor preparation. However, because stopping effects were observed, in some cases, after activity began in the spinal process, Hammond and Choo (1994, p. 194) concluded that "... any ballistic phase of motor processing must be very brief ... so brief that a distinction from controlled processes is of little behavioural significance". This study seeks to add to this finding by analysing the effects, if any, on

the H-reflex of the flexor carpi radialis (FCR) when stopping a maximal speeded wrist flexion at various times. The hypothesis, consistent with our earlier interpretation that the go process is susceptible to the effects of stopping at all times up to EMG onset, is that the H-reflex will be subject to stopping effects in the short time before EMG onset.

5.2 Method

5.2.1 Participants

Four right hand dominant participants volunteered for this study. One participant partook in Experiment III. Testing occurred in a single session. Each participant received $30 remuneration on completion of the session.

5.2.2 Apparatus and Task

Each participant sat upright in a comfortable position with his (her) right forearm fixed in a cast designed so as to isolate the forearm from the elbow to the wrist. Silver/silver chloride surface electrodes were positioned on the muscle bellies of the flexor carpi radialis (FCR) and the extensor carpi radialis (ECR). FCR-EMG was recorded in order to assess the degree to which a maximal speeded wrist flexion might be stopped at various times. The FCR-EMG signal was amplified (1-100 K range) using a multi-channel EMG system (model 544, Therapeutics Unlimited Inc.) and sampled using a 12 bit analogue-digital converter at 1000 Hz. Since ECR-EMG activity should be quiet before the onset of FCR-EMG activity so as to not adversely affect the facilitation of the FCR H-reflex through the mechanism of reciprocal inhibition (Day, Marsden, Obeso & Rothwell, 1984), the ECR-EMG was recorded at high gain (100 K) in order to detect unwanted activity in the extensor muscle before activity in the flexor muscle.

Kinematic data were recorded from an optical encoder (Dynapar E20-2500-130) and Kistler accelerometer (type 8638B50, ± 50 G) positioned 21 cm from the center of rotation. Both angular displacement and angular acceleration signals were sampled at 1000 Hz. The latter signal was filtered with an active low pass filter (Krone-Hite 3750) at 50 Hz before sampling.

Electrical stimuli were presented superficially to the median nerve using an optically isolated stimulation probe (Grass, S48). The site of stimulation was located proximal to the elbow and

identified from observance of involuntary FCR muscle twitch and participant report. The recording site was secured by a cuff that exerted sufficient constant pressure on the probe so as to reliably maintain a constant stimulation site. Voltage was set for each participant so as to yield a single involuntary muscle twitch that contained an approximate 10 % -15 % maximum M-wave (see later) and a H-reflex (see later) from each electrical pulse.

5.2.3 Experimental procedure

We used a coincident-timing task (c.f., Slater-Hammel, 1960) in order to probe the process of control for the hypothesized effects of stopping on the H-reflex facilitation shortly before EMG onset. This task was chosen so as to be able to knowingly probe the H-reflex in the short window before EMG onset. This is possible given that the onset of wrist flexion should approximate synchrony with the temporal event as specified in the experimental task.

The horizontal displacement of a response cursor in a left (wrist flexion) and right (wrist extension) direction was presented in real time on a computer monitor in front of the participant. Each participant vertically aligned the response cursor to a signal cursor that reflected a home position of 0° flexion - 0° extension in the transverse plane. Once aligned, the experimenter started each trial with a key press.

Each trial began with a start tone (sounded at 500 Hz for 150 ms) and consisted of three time marks, 500 ms, 2500 ms and 3500 ms from trial onset. The first mark (500 ms) and the third mark (3500 ms) were each preceded by an electrical pulse at a variable offset (-125 ms, -100 ms, -75 ms, -50 ms, -25 ms and 0 ms). The first pulse and the second pulse in a trial are hereafter referred to as the control pulse and the test pulse, respectively. Thus, if for example the offset for a trial was -100 ms, then the control pulse was delivered at 400 ms and the test pulse was delivered at 3400 ms for that trial. Each offset was selected randomly between trials (probability = .167) and counter-balanced across trials. The second mark (2500 ms) began the sweep hand of a clock that proceeded through one revolution anti-clockwise from its start position of 12 o'clock to its final position of 12 o'clock. (The 12 o'clock mark also doubled as the signal cursor to which the participant oriented before the

start of each trial.) The arrival of the sweep hand at its target on the third mark (3500 ms) was signaled with an end tone (sounded at 1000 Hz for 50 ms).

Each participant was instructed to produce voluntary maximal wrist flexion, over and above any involuntary muscle twitch evoked from the electrical pulse, in temporal synchrony with the arrival of the sweep hand at the 12 o'clock target (i.e., after one revolution) and the end tone. Wrist flexion from the home position (i.e., 12 o'clock) and the anti-clockwise arrival of the sweep hand at the 12 o'clock target was spatially matched so as to reflect, say, a bat striking a ball. On random trials (probability = .333), the sweep hand was stopped before it reached the 12 o'clock target, in which event the participant was instructed to withhold voluntary wrist flexion. No end tone was presented on these stop-signal trials. The time interval from stop to would-be-target (i.e., stop-target) was adjusted in accord with task performance on a trial-by-trial basis (see below).

5.2.4 Signal probabilities

The stop-target was varied experimentally, within 80 ms and 240 ms limits, in order to produce an approximate .500 probability of moving (or stopping). This was undertaken in order to get the participant on the verge of moving and stopping and so maximize stopping in the window of interest, namely in the short time interval before EMG onset. The algorithm used the probability of stopping in order to determine the stop-signal for the next stop trial. If the probability of stopping on trial n was further from .500 than the probability of stopping on trial n - 1, then the stop-signal was incremented or decremented depending on the direction of change in the updated probability, otherwise the SOA was left unchanged. The stop-target algorithm used the following nested if statement:

if p_n - .500 > 0 then

  if p_n - .500 > p_{n-1} - .500 then

    if stop-target > 80 ms then stop-target = stop-target - 10 ms

else

if p_n - .500 < 0 then

if $\underline{p}_n$ - .500 < $\underline{p}_{n-1}$ - .500 then

if stop-target < 240 ms then stop-target = stop-target + 10 ms

where $\underline{p}$ = probability of stopping and $\underline{n}$ = number of stop trials presented. For example, if $\underline{p}_{n-1}$ = .400 (e.g., 8 from 20) and $\underline{p}_n$ = .381 (i.e., 8 from 21) then the stop-target is incremented 10 ms (providing that the stop-target is not 240 ms). If $\underline{p}_{n-1}$ = .400 (e.g., 8 from 20) and $\underline{p}_n$ = .429 (i.e., 9 from 21) then the stop-target is left unchanged.

5.2.5 Data collection

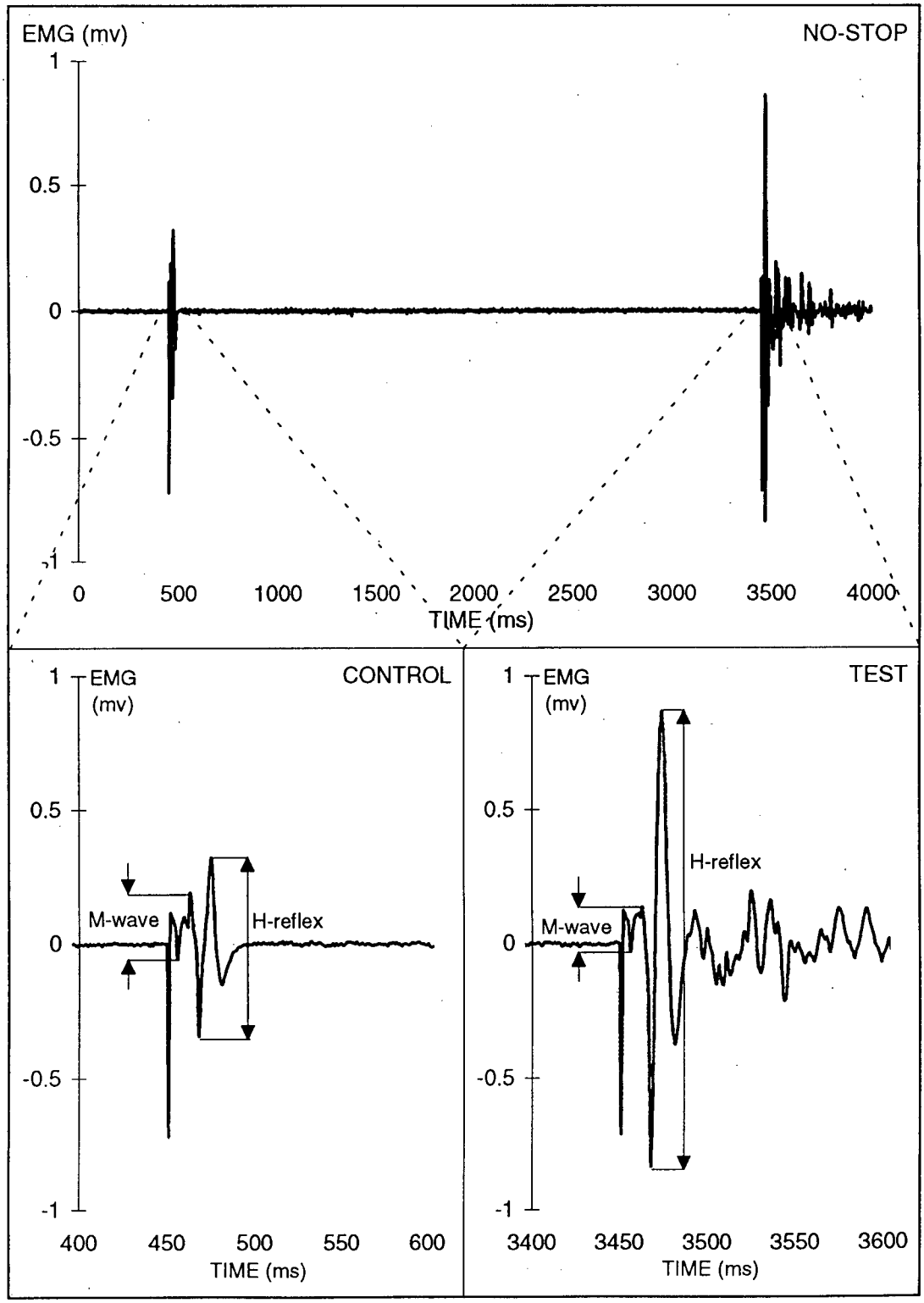
Each trial was classified as "correct" if the participant yielded voluntary EMG onset in the absence of a stop signal or no voluntary EMG onset in the presence of a stop signal, and as "incorrect" if the participant produced voluntary EMG onset in the presence of a stop signal or no voluntary EMG onset in the absence of a stop signal. Feedback with respect to the trial status (i.e., correct, incorrect) was provided to each participant on each trial, as well as the approximate error from the target, if appropriate. (This error score was approximate since the computer algorithm, which was based on a low velocity threshold, was compromised in some cases by the involuntary movement that resulted from the test pulse. Obvious errors were corrected on-line by the experimenter, thus yielding a closer approximate error score in these cases.) 150 stop-signal trials from Participant 1 and 100 stop-signal trials from Participants 2, 3 and 4 were obtained.

5.2.6 Data analysis

Trials were analysed post hoc and rejected if voluntary FCR-EMG data or voluntary ECR-EMG data preceded delivery of the test pulse. Trials were also rejected on failure to produce voluntary EMGs in the absence of a stop signal. The remaining trials ($\underline{n}$ = 920) were analysed for involuntary FCR-EMG data related to each pulse (control, test), as well as for voluntary FCR-EMG data following the test pulse. $\underline{n}$ refers to the total number of trials for each response type collapsed across participants.

Figure 5.1 (upper panel) presents an example trial from Participant 1 that shows facilitation in the test H-reflex. The upper panel details the trial EMG data in its entirety and the lower panels detail the EMG data for the control pulse (left panel) and the test pulse (right panel) on an extended broken

Figure 5.1 Example of a no-stop response from Participant 1 (upper panel). Involuntary EMG data from the control pulse (lower panel, left) and the test pulse (lower panel, right), each on an extended time line detailing the peak-to-peak amplitude of the M-wave (control = .246 mv, test = .164 mv) and the H-reflex (control = .660 mv, test = 1.702 mv) respectively. These data yielded a peak-to-peak M-wave difference between the test pulse and the control pulse of -.082 mv and a test to control H-reflex ratio of 2.58.



time line (i.e., -50 ms through 150 ms each side of the respective pulse). The first (involuntary) EMG burst results from the control pulse (upper panel, lower panel left), the second (involuntary) EMG burst results from the test pulse (upper panel, lower panel right) and the third (voluntary) EMG burst, which closely follows the second test H-reflex, results from voluntary wrist flexion (upper panel). The peak-to-peak amplitude of the M-wave and the H-reflex (lower panels) were analysed along with their corresponding peak-to-peak latencies. Those trials whose peak-to-peak latencies for the M-wave and or the H-reflex varied substantially for that participant were discarded. (Note. The peak-to-peak latencies in the M-wave changes if the double peaks reverse their order of amplitude.)

The involuntary EMG data from the control pulse (lower panel, left) yielded a peak-to-peak amplitude for the M-wave (.246 mv) and the H-reflex (.660 mv). Likewise, the involuntary EMG data from the test pulse (lower panel, right) yielded a peak-to-peak amplitude for the M-wave (.164 mv) and the H-reflex (1.702 mv). The peak-to-peak M-wave difference between the control pulse and the test pulse is thus -.080 mv and the H-reflex ratio, expressed as test to control, is 2.58.

Trials were then classified (see below) according to the various types of response - no-stop, go-stop, partial and stopped - on the basis of voluntary EMG.[12] Figure 5.2 details an example no-stop, go-stop, partial and stopped response observed from Participant 1 on a broken time line. The left and right panels detail the time window (-100 ms through 300 ms) around the control pulse and the test pulse respectively. No-stop responses ($\underline{n}$ = 599) were identified from the presence of voluntary EMGs in the absence of a stop signal (upper panel), stopped responses ($\underline{n}$ = 119) from the absence of voluntary EMGs in the presence of a stop signal (lower panel), partial responses ($\underline{n}$ = 69) from the

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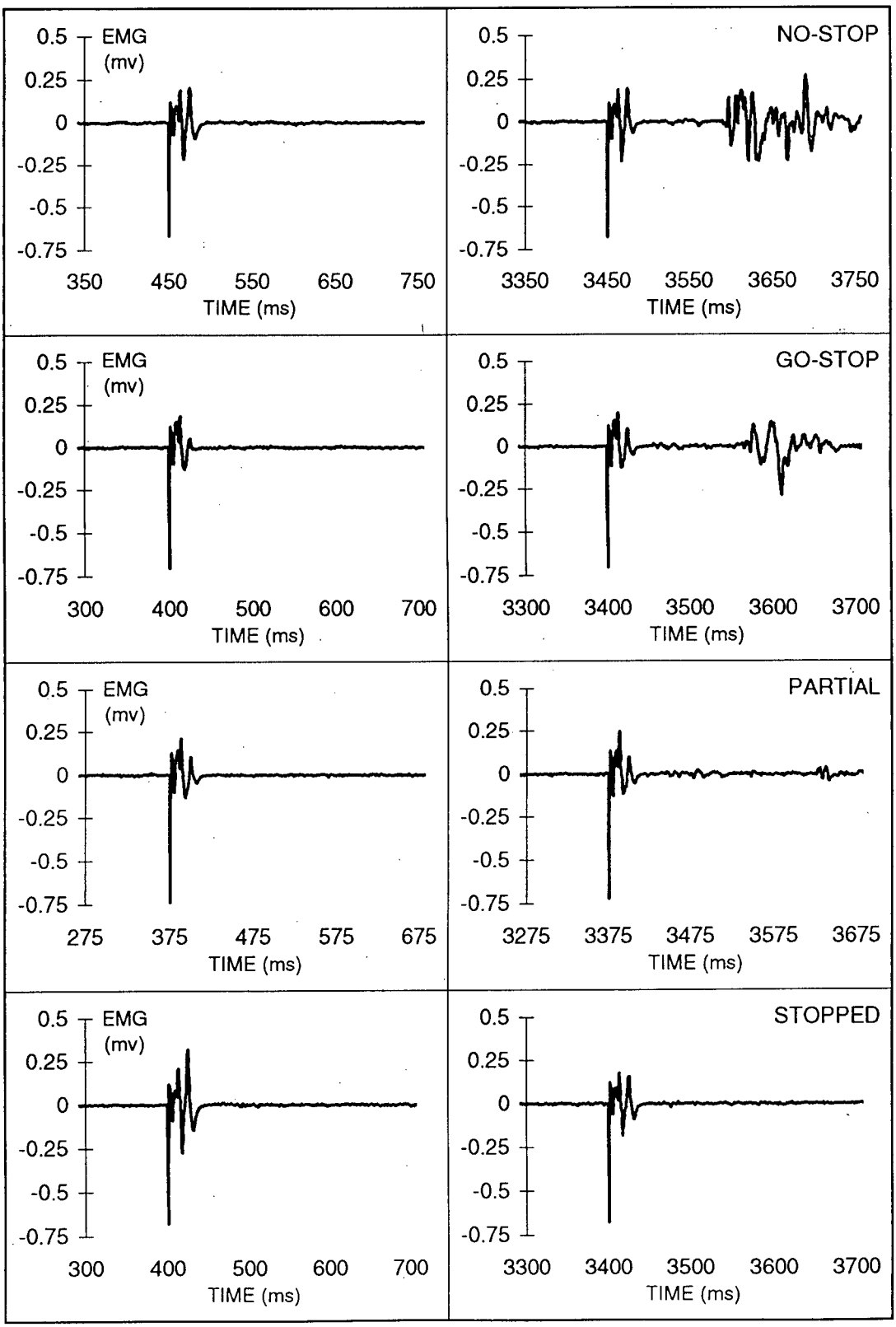
[12] In earlier studies, we segregated go responses (i.e., those actions observed in the presence of a stop signal) as either full responses, interrupted responses or partial responses on the basis of their EMG onsets (McGarry & Franks, 1997, in review-a, in review-b). In this study, we segregated go responses as either go-stop responses or partial responses. Full responses and interrupted responses were not segregated and go-stop responses therefore constitute both of these types of responses.

Figure 5.2 Example of a no-stop response (upper panel), go-stop response (upper middle panel), partial response (lower middle panel) and stopped response (lower panel) on a broken time line from Participant 1. Each trial is presented on a broken time line with a time window of -100 ms through 300 ms around the control pulse (left panel) and the test pulse (right panel).



presence of sub-maximal voluntary EMG onsets in the presence of a stop signal (lower middle panel) and go-stop responses (n = 133) from exclusion, that is from the presence of the remaining voluntary EMGs in the presence of a stop signal (upper middle panel).

5.3 Results and Discussion

5.3.1 The M-wave and the H-reflex

The M-wave results from the direct recruitment of the motor axons by the electrical pulse. In contrast, the H-reflex results from the indirect recruitment of the motor pool via the Ia afferent pathway (Schieppati, 1987). The solid circles (Figure 5.3) detail the difference in the M-wave peak-to-peak amplitude between the control pulse and the test pulse for each trial for each participant (c.f., Figure 5.1). These data show that the M-wave is consistent across trials as evidenced in their tight variance around zero. Thus, any effect on the H-reflex can reasonably be interpreted to indicate a change in the descending voluntary control, rather than to constitute an artifactual result of varying electrical stimuli.

The trials were segregated by participant, response type and offset. Figure 5.4 (upper panel) details the error from the target as a function of the offset collapsed across participants. The linear regression between the offset and the error for the no-stop responses, go-stop responses and partial responses were r = -.951, r = -.955 and r = -.968 respectively ($p < .001$). These data might be interpreted as an effect of interference in that the shorter the offset (i.e., the delivery of the test pulse to the target), the greater the error incurred. In fact, this pattern of data is an artifact of the process of rejecting those trials that showed premature voluntary FCR-EMG. This is because trials that show voluntary FCR-EMG data early with respect to the target are not rejected for trials with large offsets, but they are rejected for trials with small offsets. That is, EMG onsets before the target are increasingly likely to occur before the offset at increasingly shorter offsets. Thus, trials were rejected on the basis of premature voluntary EMG onsets with increasing frequency as the offset was reduced. The result is an increasing over-inflated positive error score as zero offset is reached, since negative error scores are more frequently rejected. Indeed, the error regression washes out when those no-stop responses rejected on the basis of premature voluntary FCR-EMG data were included

Figure 5.3 Peak-to-peak M-wave difference between the control pulse and the test pulse for each trial for each participant. Trial numbers follow data collection and are not renumbered following post hoc analysis.

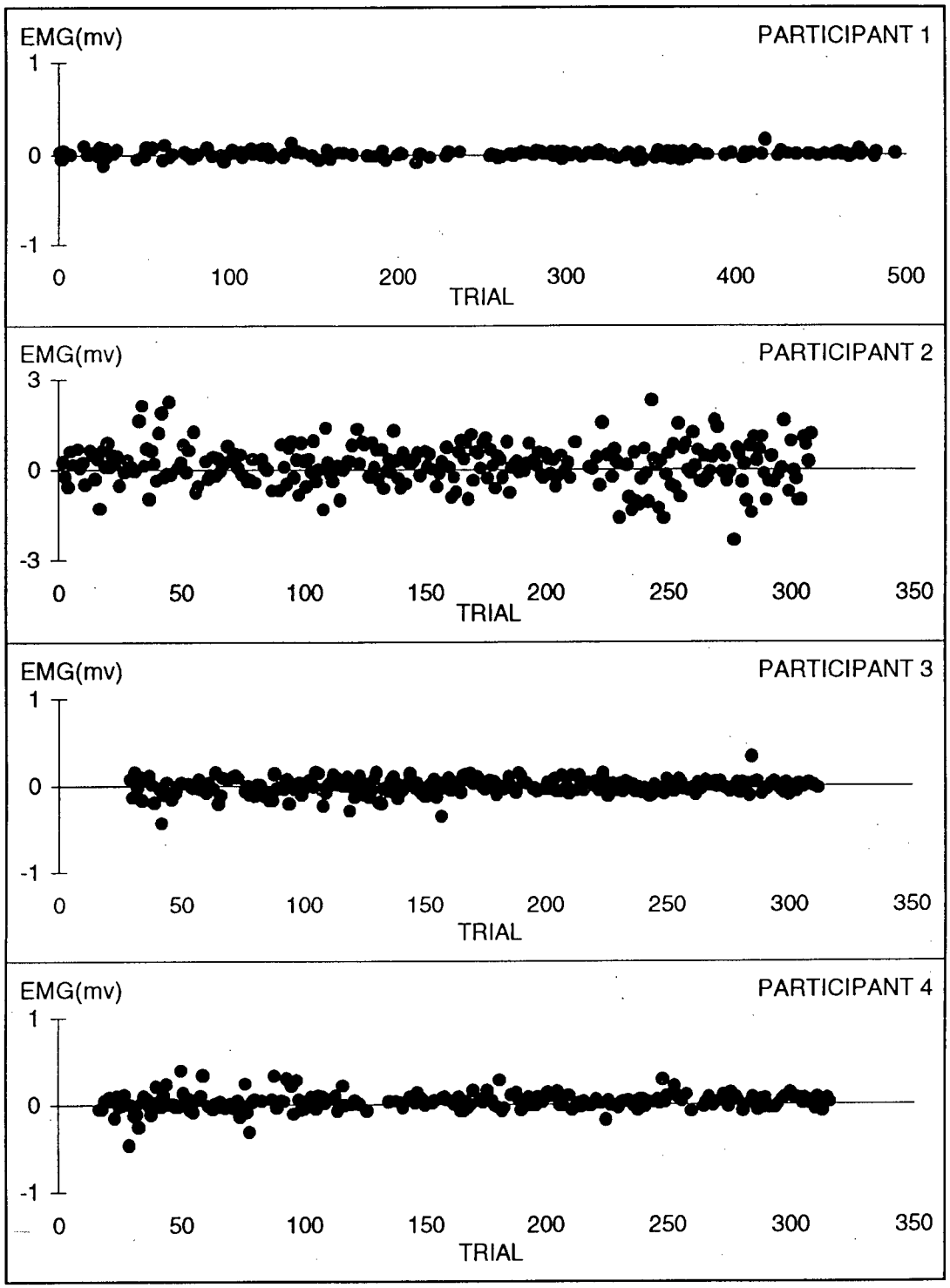
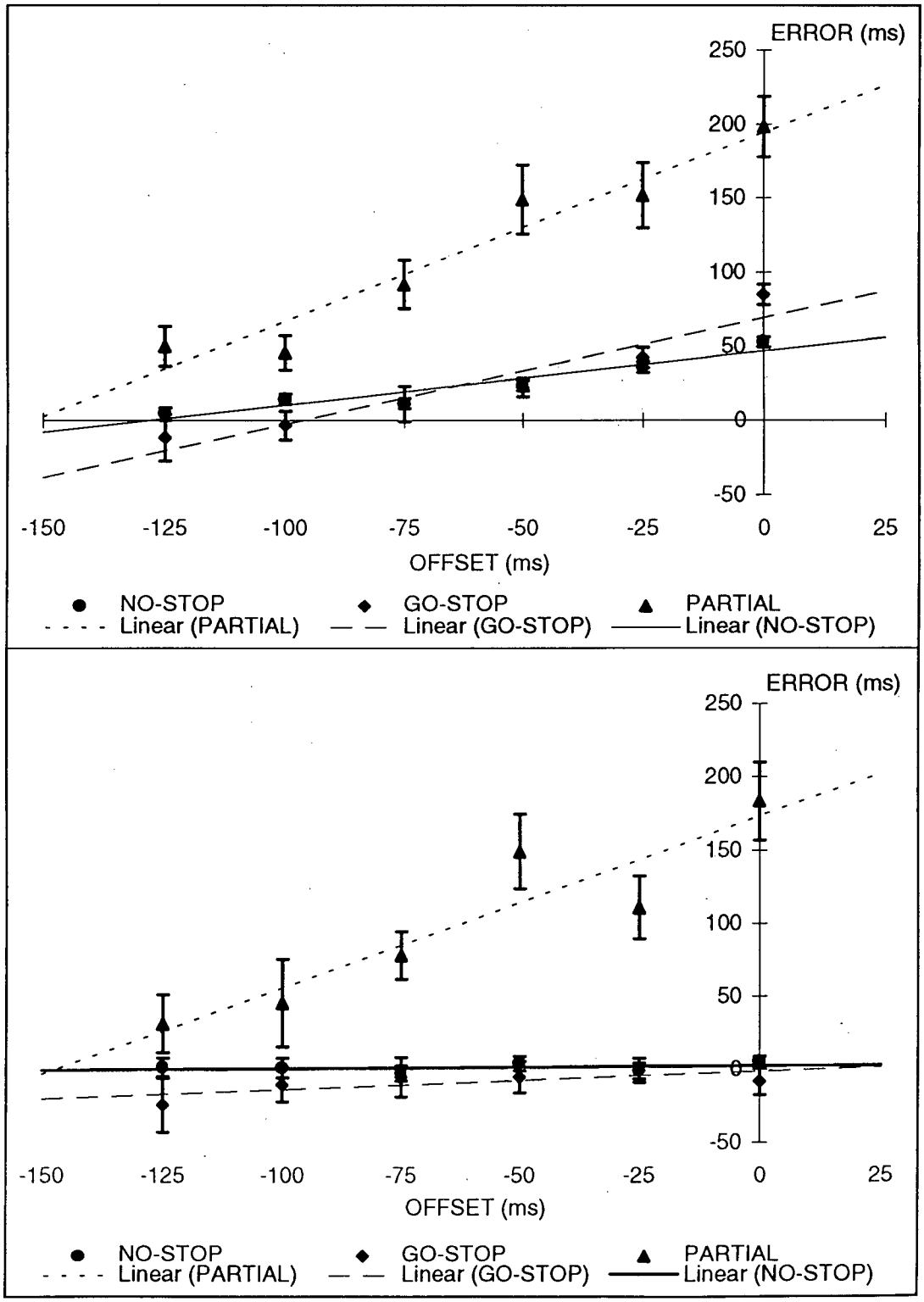


Figure 5.4 Latency error (mean and standard error) from the target for the no-stop, go-stop and partial responses as a function of the offset collapsed across participants (upper panel). Error from the target for the no-stop, go-stop and partial responses re-analyzed as a function of the offset, including those trials previously rejected on the basis of premature voluntary EMG onset, collapsed across participants (lower panel).

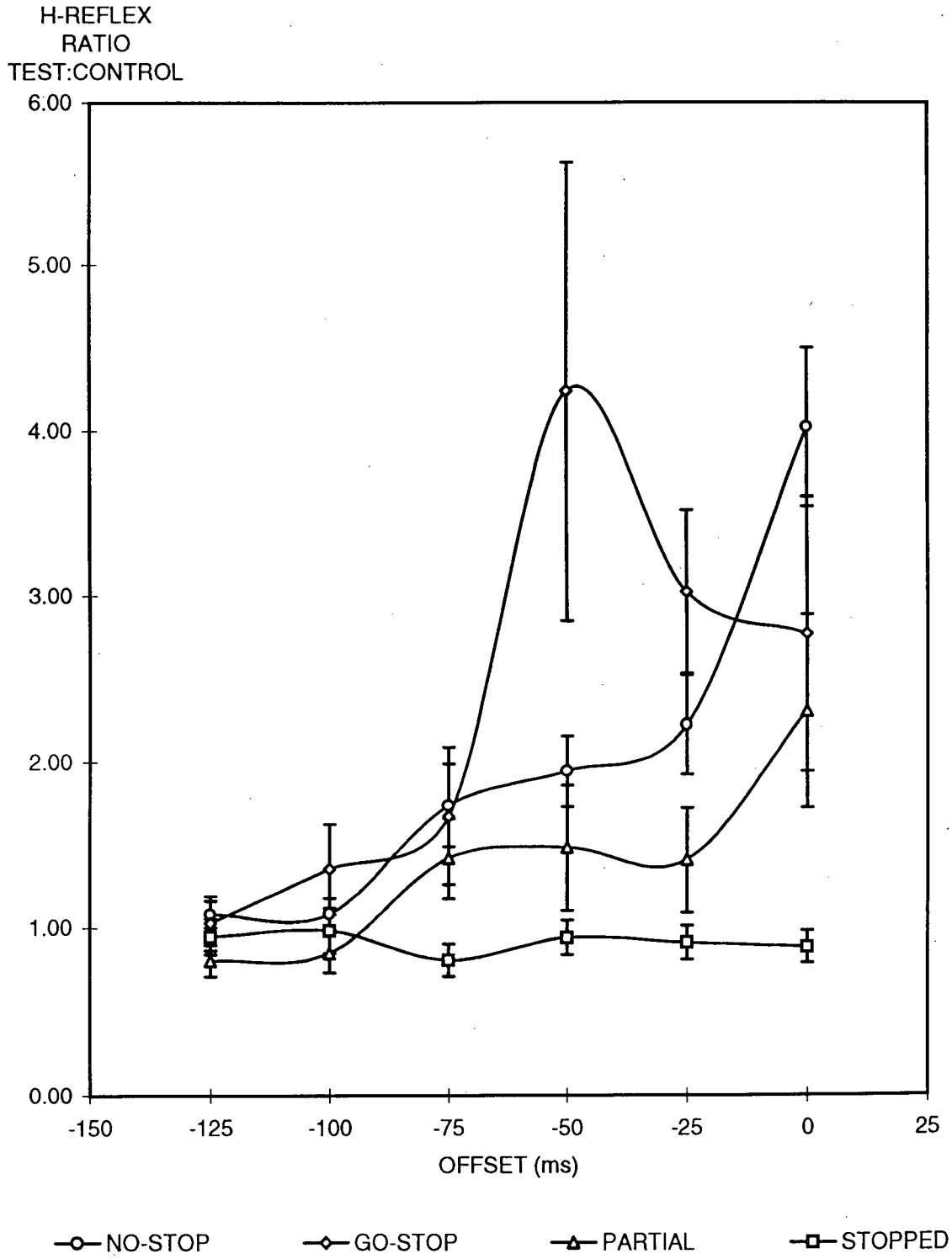


in the analysis (r = -.315, p > .05). Similar trends were evidenced for the go-stop responses (r = -.720, p < .01) and the partial responses (r = -.924, p < .001), although the effect remained significant in each instance, likely because these results were based on fewer observations (see Figure 5.4, lower panel).

Figure 5.5 details summary data of the H-reflex ratio (test to control, see Figure 5.1) for each response type as a function of the offset collapsed across participants. The no-stop responses (circles) yielded the typical H-reflex facilitation in the short time before EMG onset. (The onset of the H-reflex precedes the onset of the EMG by the offset plus the signed error from the target.) The go-stop responses (diamonds) also showed the typical H-reflex facilitation albeit at earlier offsets before its attenuation as a result of active stopping. The partial responses (triangles) yielded subdued H-reflex facilitation and the stopped responses (squares) showed no H-reflex facilitation whatsoever.

That the H-reflex facilitation precedes and seemingly necessitates voluntary EMG onset supports a point of no return in the control of a voluntary action. Evidence for a final ballistic process might be suggested from the absence of a stopping effect on the H-reflex that precedes the EMG onsets that typify the partial responses. Evidence against a final ballistic process might be suggested from the presence of a stopping effect on the H-reflex that precedes the EMG onsets that typify the go-stop responses. If the H-reflex facilitation for the partial responses and the H-reflex facilitation-inhibition for the go-stop responses are interpreted as speaking for and against a final ballistic process respectively, then it becomes necessary to square the level to which a final process is ballistic as an inverse function of the level of input that the final process receives. We see no reason as to why a process of control immediately before EMG onset should be ballistic at low levels of input, as for a partial response, but, at the same time, should not be ballistic at high levels of input, as for a go-stop response. Instead, it is more likely that the lack of inhibition in the partial H-reflex facilitation results from a lack of power that stems from; (a) few data (n = 69), (b) variance in the H-reflex, and (c) a small effect size (i.e., the small facilitation in the H-reflex from baseline necessarily limits any subsequent effect of stopping.)

Figure 5.5 The H-reflex (mean and standard error) expressed as a ratio of the test peak-to-peak amplitude to the control peak-to-peak amplitude (see Figure 5.1) for the no-stop, go-stop, partial and stopped responses as a function of offset collapsed across participants.



That the H-reflex for the go-stop responses is facilitated earlier in time than is the H-reflex for the no-stop responses is perplexing. In keeping with the horse race, only the faster go processes (from the go distribution) escape the stop process, so yielding earlier onset times in the presence of a stop signal (Logan & Cowan, 1984; Logan, Cowan & Davis, 1984). In this study, faster go-stop latencies are indicated from an increased negative error (Figure 5.4). This increased negative error, which indicates an early flexion response in relation to the target, would result in earlier H-reflex facilitation as a function of offset. The possibility of the stop signal (tone), presented 80 ms to 240 ms before the target, somehow facilitating the early onset of the H-reflex before its later attenuation, while speculative should also be considered.

In sum, the facilitation of the H-reflex that seemingly compels voluntary EMG onset might be taken to mark a point of no return in the control of a voluntary action. We make two observations in this regard. First, the point of no return is phantom if, as it seems, the onset of the H-reflex facilitation necessitates an action yet at the same time the H-reflex facilitation remains subject to further effects of stopping. Second, the point of no return is fluid. If the onset of the H-reflex facilitation marks the point of no return, then it locates at about 75 ms before EMG onset for the no-stop responses and the go-stop responses and it locates at about 175 ms before EMG onset for the partial responses. These values were derived from the offset at which the H-reflex is first facilitated for that response (Figure 5.5) plus its signed error at that offset (Figure 5.4).

5.3.2 On a mechanism for stopping a voluntary action

The pre-synaptic inhibition of Ia terminals would seem a candidate mechanism as to how the typical H-reflex facilitation before EMG onset might be reduced as a result of stopping. For example, increased pre-synaptic inhibition (from sensorimotor cortex and red nucleus) and decreased pre-synaptic inhibition (from cortico-spinal and rubro-spinal fibres) allows for the following suggested functional properties (Baldissera, Hultborn & Illert, 1981). On the one hand, increased pre-synaptic inhibition would provide negative feedback control of the motor neurons and so serve to limit rogue motor discharges before the arrival of the impending efferent drive. On the other hand, decreased pre-synaptic inhibition, or disinhibition, would provide positive feedback

control of the motor neurons and so serve to promote the effect of the efferent drive at the motor pools. This negative-positive control gain on motor discharge is thought to be regulated from supra-spinal centres and relayed through spinal mechanisms (i.e., inter-neuron relays) that act to increase or decrease pre-synaptic inhibition (Baldissera et al., 1981; Hultborn, Meunier, Pierrot-Deseilligny & Shindo, 1987; Meunier & Pierrot-Desilligny, 1998; Riedo & Ruegg, 1988; Schieppati & Crenna, 1985).

Thus, pre-synaptic disinhibition and pre-synaptic inhibition causes facilitation and suppression of the H-reflex respectively. The hypothesised stopping effects on the H-reflex facilitation before EMG onset observed in this study is consistent with a role for pre-synaptic inhibition in the stopping of a voluntary action. Not only does pre-synaptic inhibition serve to contain the motor pool before its disinhibition on the arrival of the efferent drive, but its facilitation (following disinhibition) serves to help stopping by reversing the aforesaid process. This mechanism is supported in Schiepatti and Crenna's (1984) report of the H-reflex being suppressed immediately after the onset of voluntary relaxation in the soleus muscle as a supposed result of pre-synaptic inhibition. This finding obligates a mechanism of stopping that acts on the voluntary descending control right up to the motor pool.

Lastly, the results from this study raise the possibility that the H-reflex is tied to the level of the descending voluntary drive to the motor pools. Just as the descending efferent volleys to the motor pools are reduced by the stop process, resulting in the various graded EMG onsets reported elsewhere (McGarry & Franks, 1997, in review-a, in review-b), so might the H-reflex be reduced. If the level of descending control to an agonist acts on its inter-neurons and its motor neurons in like quantity, then it follows that reduced effects on the inter-neurons (that disinhibit pre-synaptic inhibition) would be matched in reduced effects in motor discharge. Thus, an inverse linkage between the level of pre-synaptic inhibition and the level of efferent drive might be expected. This suggestion, however, is seemingly inconsistent with the dissociation of the H-reflex and the EMG onset as reported by Hammond and Choo (1994). Further research would be needed to address this issue.

5.4 Summary

The data from this study demonstrate that the descending voluntary control that reaches the motor pool in the short time interval before the arrival of the efferent drive is open to the effects of stopping. The varying patterns of the H-reflex facilitation that delineate each type of response - no-stop, go-stop, partial and stopped - affords two related observations in this regard. First, the onset of the H-reflex facilitation seemingly obligated the onset of voluntary EMG at some later time. These data speak for a point of no return in the control of an action. Second, the H-reflex was subject to stopping effects both before and after the onset of its facilitation and, importantly, these effects occurred before voluntary EMG onset. These data speak against a final ballistic process, as defined, in the control of an action. Taken together, these findings provides strong corroborative evidence for a phantom point of no return in the on-line control of an action that is subject to stopping effects at all times up to motor discharge (McGarry & Franks, 1997).

6 General Discussion

We have posited that the control of voluntary action is effected through the confluence of excitatory and inhibitory influences that act on the motor pools. The consequent effect is temporal-spatial motor discharge that contracts skeletal muscle that leads to movement. That control is effected through the timely discharge of motor neurons is only consistent with known physiology. Our account of excitatory-inhibitory interaction suggests one way in which the control processes might act on the motor pools when an earlier intended action is to later be stopped. We offer some brief considerations on control that follow from this account of excitatory-inhibitory interaction.

6.1 On the control of movement by reducing the degrees of freedom

One expected function of the intermediary role that motor neurons assume (Sherrington's final common path) between cortical centres and skeletal muscle is a reduction in the degrees of freedom available to the control mechanism. For instance, that higher order neurons effect control through their summed action on a fixed constituency of motor neurons would be expected to reduce variance in the final outcome. (Note. This reasoning does not seek to explain how the motor neurons might further be reduced to lower dimensions which, along with peripheral indeterminacy, was Bernstein's original problem of motor control.) In effect, variance in the outcome of earlier neural discharges that bombard the motor neurons is reduced by skeletal muscle acting as a low pass filter (Ghez, 1991), given the physical limits of rate discharge of the finite motor neurons.

The net result of motor discharge might thus be considered the basic unit through which control is effected. In this light, Gottlieb et al. (1989a, b) posited control to proceed in accordance with one of two neural strategies, which they opted to call speed sensitive and speed insensitive. Here, the amplitude (speed sensitive) or duration (speed insensitive) of the neural burst is modulated as a presumed consequence of the task demands. Pfann, Hoffmann, Gottlieb, Strick & Corcos (1998) later modified this proposal to suggest a single set of rules of control with the earlier EMG differences (Gottlieb et al., 1989a, b) being accounted for through biomechanical constraints at the joint in question.

Ulrich & Wing (1991) proposed a similar strategy of control in which units of force of constant amplitude are thought to be controlled by varying their number and duration (see also Ulrich, Wing & Rinkenauer, 1995). In fact, this account is incorrect from a physiological standpoint for, as identified by Bernstein (1935), the force amplitude from a given motor neuron would change as a function of various factors, most notably the length of the muscle. Nevertheless, the control of force might be effected at the level of an executive and, indeed, it is effected at the level of skeletal muscle, through motor discharge patterns that somehow translate to force amplitude. The results on stopping from the studies presented here speak neither for nor against these theories of control, though they share the same medium of motor discharge through which such control strategies must be effected.

6.2 On latency distributions

The binary hierarchy reported earlier (Figure 4.3) provides for increasing neural recruitment up to motor discharge. The binary power increase of motor neurons (2^{g-1}) from their relay neurons (2^{g-2}) might then suggest an expected increase in variance of motor discharge, rather than a decrease in variance as suggested above. In fact, this is not the case, at least insofar as it relates to the timing of motor discharge. The inter-connecting network - recall that each parent projects to each child - allows that each relay neuron (2^{g-2}) projects to each motor neuron. Thus, increasing values of g serves to reduce variance in the timing between sibling motor discharges. This is because, for increasing g, each motor neuron receives an increasing bombardment from higher order relay neurons (2^{g-2}), thus prompting each motor neuron to reach threshold in shorter (i.e., minimal) time. Furthermore, the time window in which this barrage occurs across the motor pool reduces as some function of g.

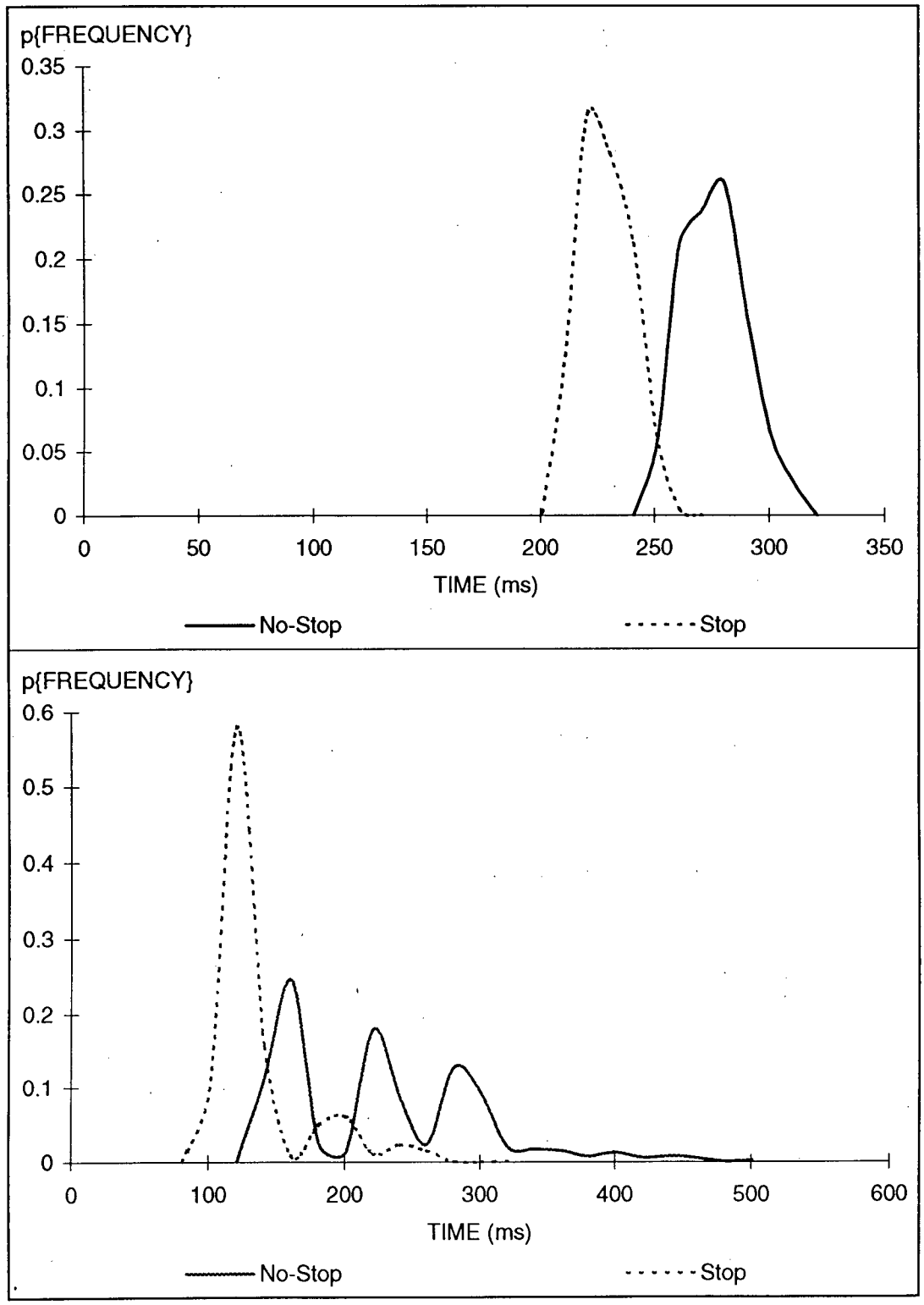
The binary architecture provides for a run-away effect as more neurons propagate the hierarchy much like a nuclear chain reaction. The run-away effect results from each lower level neuron reaching its threshold faster as it receives increasing bombardment from higher level neurons. In addition, since increasing like neurons (i.e., siblings) discharge closer in time to each other, the result is an ever closer approximation to the lower limits of time-to-discharge. In other words, the

stochastic nature of the time-to-discharge reduces as the lower limits are increasingly approximated. The asymmetric effect tending to symmetry of the time-to-discharge that results from the increased inter-connectivity between parents and children for each generation provides a positive skewed latency distribution for both the go process and the stop process. This is observed mildly from a frequency histogram analysis (Figure 6.1, upper panel), when each process in the first model (McGarry & Franks, in review-a) was allowed to traverse the network in the absence of the other process. (Note. In the first model, threshold was presumed to be reached on receipt of the first pulse from a parent.) That latency data were also positively skewed provides good support for this type of architecture in which increasing neurons are recruited in the generation of an action.

Figure 6.1 (lower panel) provides the latency distribution from the second model (McGarry & Franks, in review-b). This model produces a multi-modal (tri-modal) latency distribution, with each mode being ordered successively from high to low and from fast to slow (i.e., the highest mode is the fastest mode and so on). The reason for this, in no particular order, is a combined result of; (a) the random time of discharge from parent to child, (b) the size of discharge pulse from parent to child and (c) the decay of the child after receipt of discharge. These effects are plainly differential as indicated from the different distributions between the go process and the stop process.

The following account of self-propagation (c.f., Wickens, Hyland & Anson, 1994) explains how multi-modal distributions were observed in the absence of the other process. On presentation of its signal, the first parent fired repetitively at its discharge frequency (as would be expected on receipt of a strong excitatory stimulus). (Note. For any SOA, E-excitatory discharge in E : 1 - 1 was inhibited on the first receipt of a strong I-inhibitory signal from its like cousin I : 1 - 1.) On occasion, the first excitatory discharge and, less frequently, the second excitatory discharge failed to promote sufficient excitatory weight for the process to self-propagate through to the motor pool. The third excitatory discharge always managed to follow through to the motor pool by virtue of the pre-existing excitation in the hierarchy. Insufficient excitatory weight was observed as rogue excitatory discharges in the hierarchy (mostly in the small neurons, sometimes in the intermediate neurons, and rarely, if ever, in the large neurons) within various generations. This failure of the excitatory

Figure 6.1 Frequency histograms of the go latencies (solid line) and the stop latencies (dashed line) for the first model (Figure 3.4, Experiment II) (upper panel) and the second model (Figure 4.3, Experiment III) (lower panel). Note. Units of time are arbitrary.



process to self-propagate on occasion was offset somewhat by assigning the first parent in the hierarchy as large. In addition, I-excitation was more likely to self-propagate than E-excitation on the first discharge (see Figure 6.1) because of lower time-to-discharge values.

The result of the sometimes insufficient activity to carry the process through to motor discharge is three latency distributions (i.e., one for each pulse generated by the first parent). These three distributions super-impose to form one latency distribution for each process. This finding would seem to run counter to empirical data. Surprisingly, however, the TEMG latency distributions for each participant ($\underline{n}$ = 20), analysed in the same way as the generated data, also yielded multi-modal distributions in most cases (see Figure 6.2).[13] These data are unexpected and are likely a product of the task, that is, the requirement to stop on presentation of a stop signal. That said, the finding that the generated latency distribution of the go process is supported somewhat from our empirical data offers reasonable support for this type of architecture, as well as the basic underlying principles on which the model is predicated (see Figures 6.1 and 6.2).

The noted difference between the generated data and the empirical data is that the former consists of distinct distributions (one for each parent pulse) while the latter consists, seemingly, of overlapping distributions. This is expected to pose no problem for the model which would presumably achieve overlapping distributions from shortening the time interval between the pulses from the first parent. This might be achieved through increasing the firing frequency of the first parent or, alternatively, through introducing a sibling parent, or parents. It is intriguing that trains of single pulses that are used to kick-start the model in this study results in the multi-modal distributions that might underlie the process of control insofar as it relates to stopping. These considerations warrant exploration in further study.

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[13] It is interesting to note that saccadic latencies have been analysed statistically (from 963 data sets, 170 participants and 90,927 reaction latencies) as a multi-modal distribution consisting of three separate overlapping distributions; (a) express saccades (90 ms - 120 ms), fast-regular saccades (135 ms - 170 ms) and slow-regular saccades (200 ms - 220 ms) (Gezeck, Fischer & Timmer, 1997).

Figure 6.2 Frequency histograms of go latencies to onset of triceps EMG (TEMG) observed for each participant (1 to 20) from Experiment III.

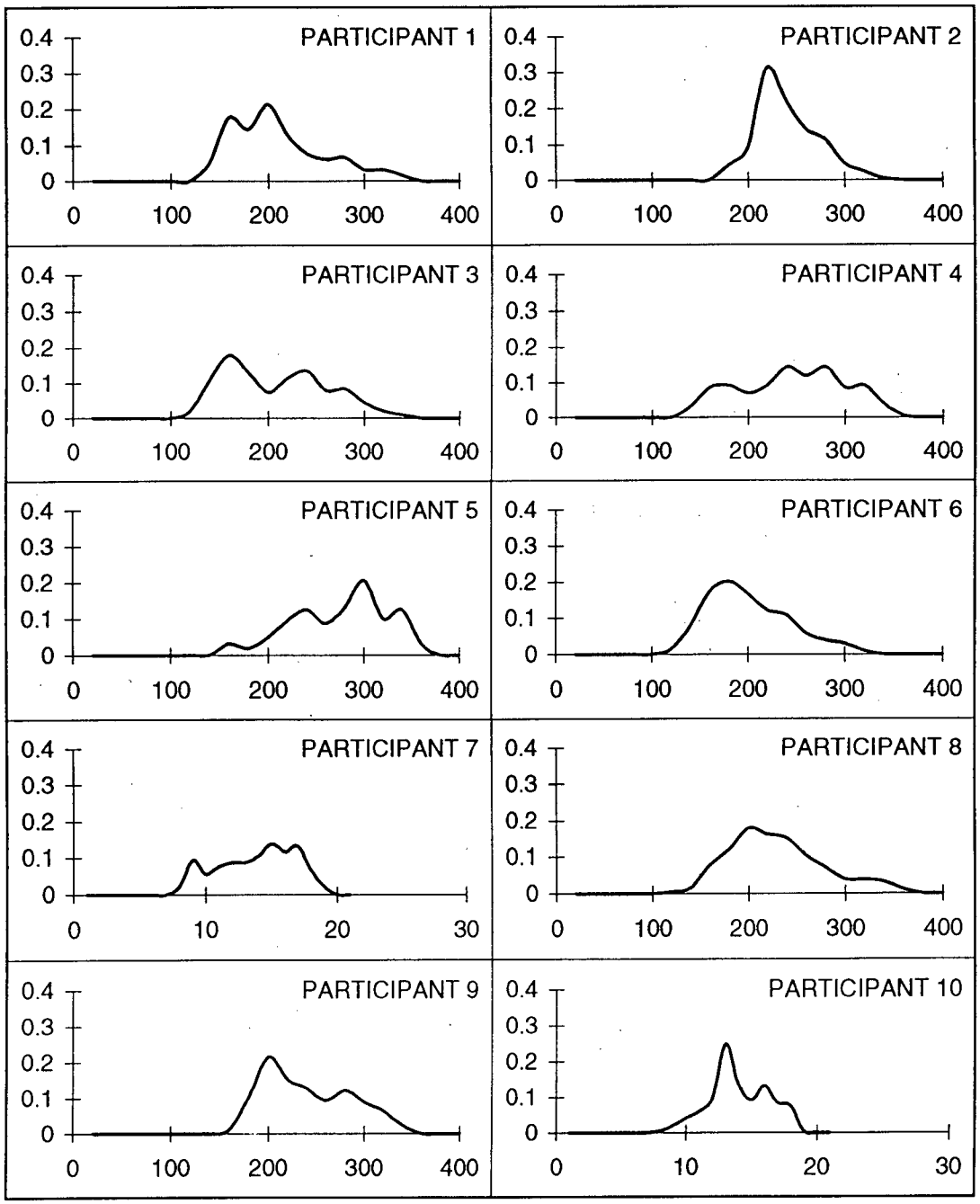
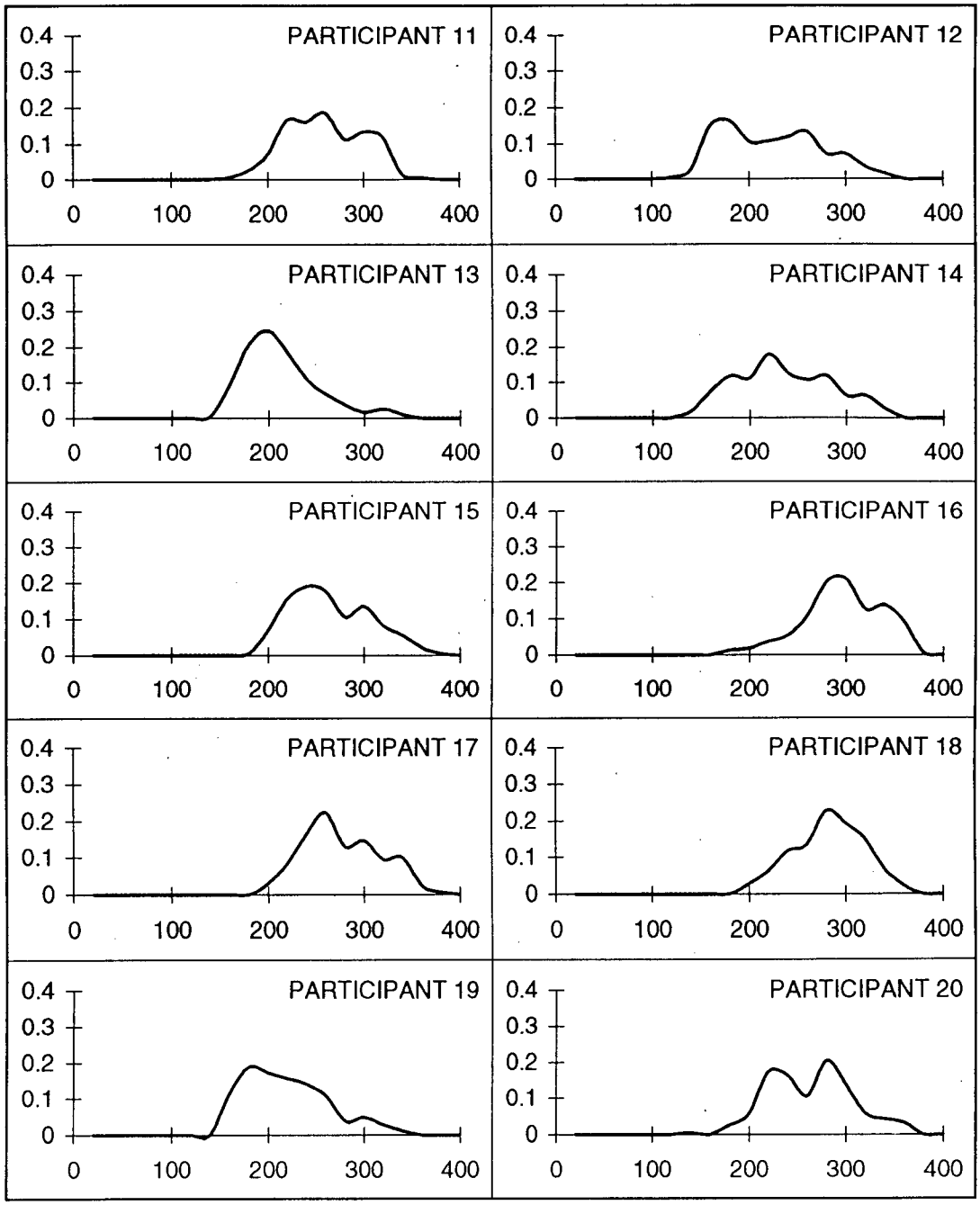


Figure 6.2 continued.



6.3 On the organisation of a motor pool

For convenience, we assumed three sizes of neurons - small, intermediate and large - to be evenly distributed throughout the architecture, including the motor pool. We do not know the likelihood of small, intermediate and large relay neurons nor, therefore, do we know their distribution, but we do know the respective details for motor neurons. The intrinsic properties of motor neurons show positively skewed graded thresholds that range from minimum (i.e., smallest) to maximum (i.e., largest). Thus, small motor neurons are distributed frequently and large motor neurons are distributed infrequently throughout the motor pool (Henneman & Mendell, 1981).

There are two ways through which force can be exerted through motor discharge. Increasing the level of synaptic drive increases the number of recruited motor neurons and also increases the firing frequencies of those motor neurons that are already recruited. Small motor neurons recruit first and begin to saturate at their maximum discharge rates as some of the large motor neurons are only beginning to be recruited. Heckman and Binder (1993a) suggested that such rate limiting effects, which are not explained from the intrinsic properties of motor neurons, might be explained from non-uniform weighted inputs to the motor pool, in other words, the preferential weighting of small and large motor neurons.

Heckman and Binder (1993a) reproduced the effects of rate limiting properties using computer simulation. The synaptic drive to the motor pool was weighted using empirical data taken from four synaptic inputs to the motor pool of a cat's hindlimb; (a) monosynaptic Ia afferent input (excitation), (b) oligo-synaptic rubrospinal (excitation), (c) reciprocal Ia inhibition and (d) recurrent (Renshaw) inhibition. The two excitatory inputs provide non-linear weightings to the motor pool. The two inhibitory inputs provide approximate linear weightings to the motor pool and were therefore rejected by Heckman and Binder (1993a) from further consideration.

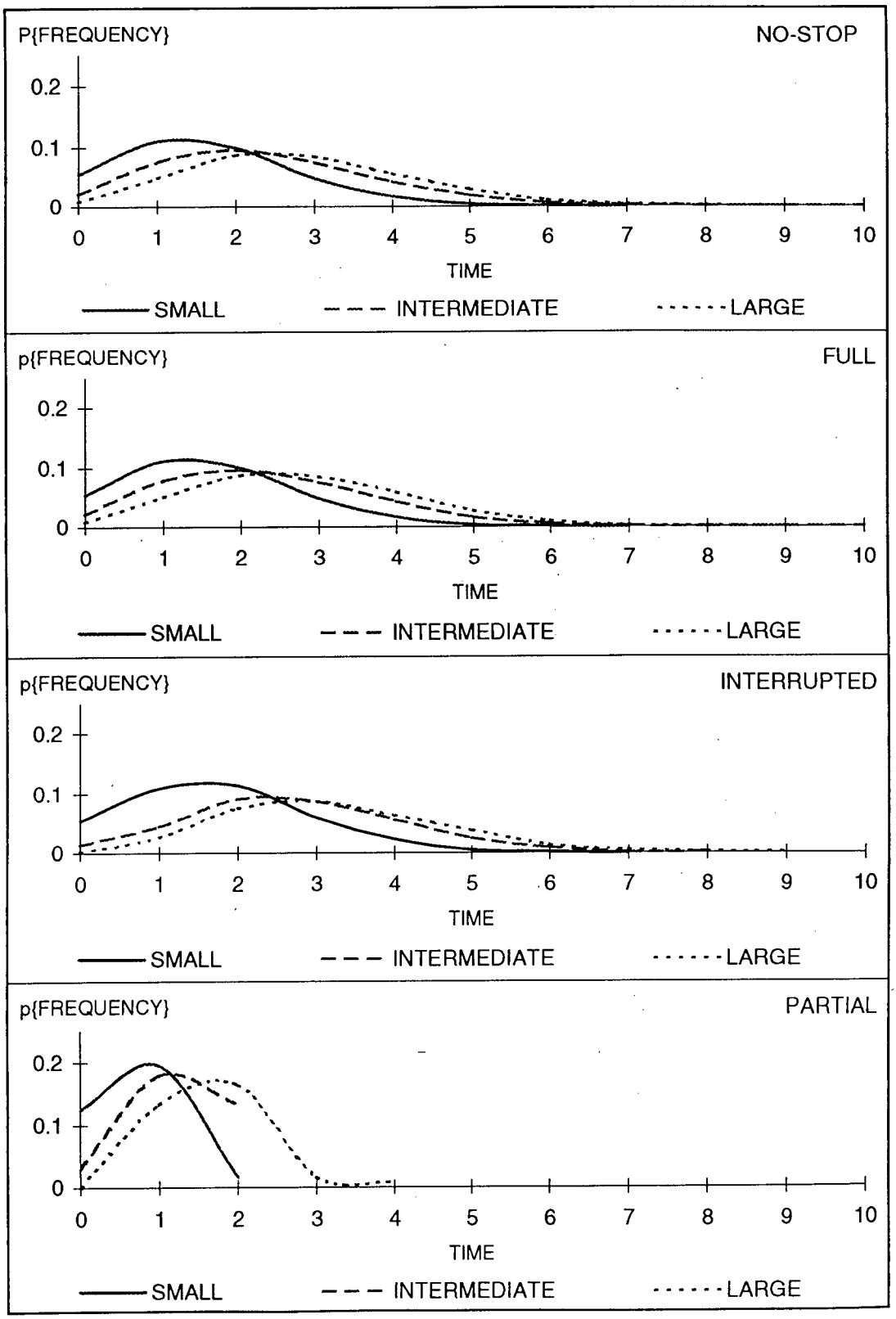
The non-linear weightings are unequal. The Ia afferent input biases weighting in favour of the small motor neurons at the expense of the large motor neurons, while the oligo-synaptic rubrospinal input biases weighting in the reverse way to stronger effect. The result of non-linear weightings to the motor pool is to expand or to contract the threshold range of the motor pool in the order of two

fold to ten fold. Interestingly, Heckman and Binder (1993a) reported that the best combination to approximate the empirical data on rate limiting effects results from a cross-over effect in which the Ia input provided the sole weighting at low levels of synaptic drive and plateaus early thereafter, while rubrospinal input provided increased weighting as the synaptic drive increased.

In a related study, Heckman & Binder (1993b) showed that the presence of both types of input were important in determining the degree to which the size principle was preserved. Intuitively, Heckman and Binder's (1993b) finding that order was better preserved at higher threshold ranges in the motor pool in the presence of large random variance amongst motor unit thresholds would be expected. (This is because variable inputs would be expected to reverse order more easily at lower threshold ranges because of the closer proximity of threshold between the motor neurons.) In our study (McGarry & Franks, in review-b), we opted to reduce the complexity of the analysis to three cell sizes (small, intermediate, large) of non-biased weightings, rather than to try and account for the effects of size distributions and or varying weighted inputs on the question at large, namely, that of how a to-be-generated action might be stopped at various times to varying degrees.

The motor neurons increase in the size of their recruitment as the synaptic drive increases. We therefore analysed the recruitment order of the motor neurons to discharge in our model (Model C). The distributions of the small, intermediate and large motor neurons are detailed as a function of time from onset of the first motor neuron to discharge for the no-stop, full, interrupted and partial responses (Figure 6.3). These data were obtained from analyzing the discharge patterns in the absence of a stop signal for the no-stop responses ($\underline{n}$ = 1000), and in the presence of a stop signal (SOA 100), for the full ($\underline{n}$ = 471), interrupted ($\underline{n}$ = 38) and partial ($\underline{n}$ = 8) responses. The results confirm the expectations that, in general, small motor neurons discharge first and large motor neurons last. Importantly, these distributions overlap which indicates that, on occasion, some large motor neurons discharge before some small motor neurons. Thus, while the order is specified in accord with physiology (i.e., size), the order is not specified in a deterministic way, but instead it results from the distributed action of the synaptic activity that is spread across the motor pools.

Figure 6.3 Frequency histograms of the first motor discharges as a function of size for the no-stop, full, interrupted and partial responses from model C (see Section 4.3.3 On a theory of control for stopping for further detail).



The process detailed here allows for the occasional reversal of recruitment order in some cases as a result of stochastic properties. This is because the synaptic (excitatory) input acts non-preferentially on the motor pool yielding the following properties. Low synaptic input would recruit preferentially the small motor neurons at low levels of excitatory input since these levels would be insufficient to allow the large motor neurons to reach threshold. While the decay function of the discharge pulse from parent to child means that small motor neurons would still be favoured over large motor neurons at all times, this bias is reduced increasingly at higher levels of excitatory input.

Table 6.1 details the size of the first motor neuron(s) to discharge as a function of response type from the data presented in Figure 6.1. In many instances, multiple motor neurons discharge at onset (i.e., time zero) in which case only one motor neuron from each size was counted. For example, if the neurons to discharge at time zero were of size 1, 1, 1 and 1, then this would count as a single instance of size 1. If the neurons to discharge at time zero were of size 1, 1, 2, 1, 2, 3, 2 and 1, then this would count as a single instance of size 1, 2 and 3 respectively. Each observed size combination is presented as a proportion of the total number of observations for that response type.

Table 6.1 shows that proportionally more small neurons discharge for the interrupted responses and for the partial responses than for the no-stop responses. This is consistent with the reasoning advanced above of preferential recruitment of small motor neurons at low excitatory input, that is when the descending excitatory drive is reduced by the stopping process. In contrast, unrestrained excitatory input will increase the probability of combinatorial orders and so decrease the probability that only small motor neurons will discharge. The infrequent observations of intermediate motor discharge, as well as the small-large combination in which case the intermediate motor neurons are by-passed, shows that the size order is subject to variation as a result of stochastic influences (see Table 6.1). This suggestion fits with Henneman, Somjen and Carpenter's (1965a, p. 561) observation that "... some exceptions to the size principle might be expected to occur due to slight variations in the mixture of excitatory and inhibitory

Table 6.1 Combination probabilities of the size of motor discharges at onset of first discharge from Model C (for further detail, see Section 4.3.3 On a theory of control for stopping a voluntary action).

		Combination				
Response Type	n	S	S-I	S-L	S-I-L	I
No-Stop	1000	.489	.231	.011	.267	.002
Full	471	.456	.242	.013	.287	.002
Interrupted	38	.684	.237	-	.079	-
Partial	8	.625	.250	-	-	.125

Note. n = number of responses. S = Small. I = Intermediate. L = Large. S-I = Small and Intermediate. S-L = Small and Large. S-I-L = Small, Intermediate and Large.

impulses impinging on different cells in a pool". That the mixture might be stochastic offers one explanation as to how stray reversals would occasionally arise. This possibility is reinforced by Henneman (1985, p.111) who pondered as to how "... a highly deterministic output may emerge from a set of probabilistic connections". Our results are further supported in Heckman and Binder's (1993b) report that the interaction of three factors; (a) threshold, (b) amplitude of the receiving input (or pulse) and (c) inherent randomness in either the motor neuron threshold, or its share of the synaptic input - resulted in a stable order given a non-biased weighting of input to the motor pool. The predominant feature of our results, congruent with those of Heckman and Binder (1993b), is that order is preserved for the most part. This result is consistent with most findings in the literature under reasonable (i.e., normal) physiological conditions.

The size order is reversed under certain physiological conditions. For instance, the size order was reversed in a ramp isometric contraction task using background continual cutaneous stimulation (Stephens, Garnett and Butler, 1978) and fast (large) motor units were preferentially activated in the eccentric phase of rhythmical concentric-eccentric actions (Howell, Fugelvand, Walsh & Bigland-Ritchie, 1995; Nardone, Romano & Schieppati, 1989). Heckman and Binder (1993a, b) suggested weighted inputs to the motor pools from specialised neural circuits in order to explain how the recruitment order of motor neurons, as well as the persistent disorder in some cases, might be achieved. These special distributed inputs were hypothesised to change the weighting of inputs that motor neurons of varying sizes receive, effected in one of two ways - either through weighting the thresholds of the individual neurons or, alternatively, through weighting the pulses that these individual neurons receive (Heckman & Binder, 1993b). We suggest that varying the discharge is preferable to varying the threshold in keeping with Hanes and Schall's (1996) empirical findings (see p. 96). In fact, the varying of discharge pulses is already provided for in our model through the random allocation of time-to-discharge which yields different pulse discharges by virtue of the linear decay function.

In sum, the theory of control that we have presented by way of a computer model is consistent with the extant literature and offers a promising way to analyse further the properties of control of a

voluntary action. Immediate advances of this model include the introduction of; (a) an agonist-antagonist pair, possibly controlled through the mechanism of reciprocal inhibition, (b) extending the frequency of cell sizes to more accurately reflect the distribution of motor neurons within a motor pool, and (c) introducing specialised neural circuits that act to bias the synaptic weights of the motor neurons (c.f., pre-synaptic inhibition), possibly through the inclusion of other cell assemblies. Interestingly, this latter suggestion of changing synaptic weights offers at least two possibilities; of priming the structure to bias the cells towards excitation (positive priming) or inhibition (negative priming), as well as extending the model of control to include Hebbian learning effects. The results from our model, in conjunction with the preceding comments, lead us to suggest that the systematic development of a computer model that incorporates physiological principles now constitutes a pressing demand, if a complete theory of control and action is to be realised (see also Ramos & Stark, 1988). Indeed, this technique is already being used in some quarters to good effect (Fortier, 1994; Fugelvand, Winter & Patla, 1993; Heckman & Binder, 1993a, b; Ramos & Stark, 1987).

7 Conclusion

The series of studies detailed above provide for the following comments. First, the point of no return that marks the onset of a final ballistic process, as defined by Osman et al. (1986, 1990), is phantom. Sub-maximal EMG records were observed in some cases when trying to stop a maximal speeded action at various times. These data betray a go process that is reduced as a result of stopping seemingly at all times up to motor discharge, thus speaking against a final ballistic process. This assertion was fortified from instances of single EMG spike trains which tend to support the case for a competitive mechanism of control at the level of the individual neuron. Lastly, the reversal of the typical H-reflex facilitation that precedes EMG onset as a result of stopping affirmed, to the best of our ability thus far, that the point of no return is phantom. This inference was derived from the reasoned expectation that the presence of a final ballistic process leading into EMG onset would otherwise act to preserve the aforesaid H-reflex facilitation. These results weigh heavily against a final ballistic process, as defined, in the control of voluntary action.

Second, the race model well describes the latency relations of the go process, the stop process, the SOA and the outcome probability. These relations were not contested in the series of studies that form this thesis. Instead, we analysed the amplitude (from EMG onset) rather than the latency of an initiated action and observed, in some cases, graded EMG onsets as a result of stopping at various times. In the parlance of the race model, sub-maximal EMGs are analogous to the go process winning the race but crossing the finish line at much reduced velocities. This is an unexpected observation that cannot be explained from the race model. Since a theory of control must account for all of the empirical data, we presented, by way of a computer model, an account of excitatory-inhibitory interaction that can reproduce the empirical data to a reasonable degree. Furthermore, since this account is grounded in basic physiology, this theory provides a first step towards meshing the principles of control, as understood thus far, from cognitive science and neuro-science. Further steps should now be taken towards this objective.

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Appendix

The following example details the state of the architecture for each time step listed, as well as a brief companion description of each cell update, for model A (see Section 4.3.3 On a theory of control for stopping a voluntary action, for further detail). We present an abridged version in which only the active cells, indexed G - S, are detailed. Some time steps are omitted for reasons of brevity.

We use the following notation. G = generation, S = sibling order, C = cell size, ET = excitatory threshold, EST = excitation state, ERS = excitation refractory period, $T_E[1..n]$ = time of excitation pulse, E[1..n] = excitation pulse, IT = inhibition threshold, IST = inhibition state, IRS = inhibition refractory period, $T_I[1..n]$ = time of inhibition pulse, I[1..n] = inhibition pulse. The present status and future status of each cell, indexed by G - S, is updated on each time step.

The initial conditions are: Number of generations = 4; Time to excite on discharge = 15 ± 5; Time to inhibit on discharge = 10 ± 5; SOA = 10; Excitatory Threshold for cells 1, 2 and 3 are 50, 100 and 150 respectively; Excitatory Refractory State for cells 1, 2 and 3 are 100 ± 10, 50 ± 5 and 33 ± 3 respectively; Inhibitory Threshold for cells 1, 2 and 3 are -50, -100 and -150 respectively; Inhibitory Refractory State for cells 1, 2 and 3 are 100 ± 10, 50 ± 5 and 33 ± 3 respectively. Time begins at -1.

TIME	G-S	C	ET	EST	ERS	$T_E[1]$	E[1]	IT	IST	IRS	$T_I[1]$	I[1]
-1	1-1	3	150	0	999	**0**	150	-150	0	-999	10	-150

-1 1-1 EST = 0. $T_E[1]$ = 0. E[1] = constant = 150.
IST = 0. $T_I[1] = T_I[1]$ + SOA = 10. I[1] = constant = -150.
Next t=0.

TIME	G-S	C	ET	EST	ERS	$T_E[1]$	E[1]	IT	IST	IRS	$T_I[1]$	I[1]
0	1-1	3	150	999	31	31	150	-150	0	-999	**10**	-150
	2-1	1	50	0	-999	11	150	-50	0	-999		
	2-2	2	100	0	-999	13	150	-100	0	-999		

0 1-1 EST = 0 + E[1] = 0 + 150 = 150. ET = 150. EST ≥ ET, EST = 999, ERS = 31. Thus, an excitation pulse is generated and refractory state is entered.
Next excitation pulse, E[1] = constant = 150, to be received at $T_E[1]$ = 31.
2-1 E[1] = 150. $T_E[1]$ = 11
2-2 E[1] = 150. $T_E[1]$ = 13
Next t = 10.

TIME	G-S	C	ET	EST	ERS	$T_E[1]$	E[1]	IT	IST	IRS	$T_I[1]$	I[1]
10	1-1	3	150	999	999	999	150	-150	-999	44	44	-150
	2-1	1	50	0	-999	**11**	140	-50	0	-999	24	-150
	2-2	2	100	0	-999	13	140	-100	0	-999	23	-150

10 1-1 IST = 0 + IT = 0 - 150 = -150. IT = -150. IST ≤ IT, IST = -999, IRS = 44. Thus, an inhibition pulse is generated and refractory state is entered.
Next inhibition pulse, I[1] = constant = -150, to be received at $T_I[1] = 44$.
Note. ERS = 999 so all subsequent excitatory pulses from 1:1 are inhibited.
2-1 I[1] = -150. $T_E[1] = 24$.
2-1:2 I[1] = -150. $T_E[1] = 23$.
Next t = 11.

TIME	G-S	C	ET	EST	ERS	$T_E[1]$	E[1]	IT	IST	IRS	$T_I[1]$	I[1]
11	1-1	3	150	999	999	999	150	-150	-999	44	44	-150
	2-1	1	50	999	119			-50	0	-999	24	-149
	2-2	2	100	0	-999	**13**	139	-100	0	-999	23	-149
	3-1	1	50	0	-999	27	50	-50	0	-999		
	3-2	2	100	0	-999	26	50	-100	0	-999		
	3-3	3	150	0	-999	28	50	-150	0	-999		
	3-4	1	50	0	-999	27	50	-50	0	-999		

11 2-1 EST = 0 + E[1] = 0 + 139 = 139. ET = 50. EST ≥ ET, EST = 999, ERS = 11 + 108 = 119.
(Note. E[1] = 139 as a consequence of decay i.e., E[1] = 150 - 11 = 139).
3-1 E[1] = 50. $T_E[1] = 27$.
3-2 E[1] = 50. $T_E[1] = 26$.
3-3 E[1] = 50. $T_E[1] = 28$.
3-4 E[1] = 50. $T_E[1] = 27$.
Next t = 13.

TIME	G:S	C	ET	EST	ERS	$T_E[1]$	E[1]	$T_E[2]$	E[2]	IT	IST	IRS	$T_I[1]$	I[1]
13	1-1	3	150	999	999	999	150			-150	0	44	44	-150
	2-1	1	50	999	119					-50	0	-999	24	-147
	2-2	2	100	999	67					-100	0	-999	**23**	-147
	3-1	1	50	0	-999	27	48	29	100	-50	0	-999		
	3-2	2	100	0	-999	26	48	30	100	-100	0	-999		
	3-3	3	150	0	-999	28	48	31	100	-150	0	-999		
	3-4	1	50	0	-999	27	48	28	100	-50	0	-999		

13 2-2 EST = 0 + E[1] = 0 + 137 = 137. ET = 100. EST ≥ ET, EST = 999, ERS = 13 + 54 = 67.
3-1 E[2] = 100. $T_E[2] = 29$.
3-2 E[2] = 100. $T_E[2] = 30$.
3-3 E[2] = 100. $T_E[2] = 31$.
3-4 E[2] = 100. $T_E[2] = 28$.
Next t = 23. We now jump ahead in time to t = 45.

TIME	G-S	C	ET	EST	ERS	T_E[1]	E[1]	T_E[2]	E[2]	T_E[3]	E[3]	IT	IST	IRS	T_I[1]	I[1]	T_I[2]	I[2]
45	1-1	3	150	999	999	999	150					-150	-999	77	77	-150		
	2-1	1	50	999	119							-50	-999	127	54	-149		
	2-2	2	100	999	67							-100	-999	70	52	-149		
	3-1	1	50	999	135							-50	-999	124				
	3-2	2	100	999	80							-100	-999	82				
	3-3	3	150	98	-999							-150	-118	-999				
	3-4	1	50	999	126							-50	-999	139				
	4-1	1	50	33	-999			47	34	48	85	-50	-999	141				
	4-2	2	100	68	-999					47	85	-100	-999	91			**46**	-38
	4-3	3	150	67	-999					**46**	85	-150	-128	-999			**46**	-38
	4-4	1	50	33	-999			**46**	34	**46**	85	-50	-999	137				
	4-5	2	100	33	-999			48	34	**46**	85	-100	-999	90				
	4-6	3	150	33	-999			48	34	49	85	-150	-129	-999			47	-38
	4-7	1	50	33	-999			48	34	**46**	85	-50	-999	141				
	4-8	2	100	33	-999			47	34	48	85	-100	-999	93			**46**	-38

45 Next t = 46.

TIME	G-S	C	ET	EST	ERS	T_E[1]	E[1]	T_E[2]	E[2]	T_E[3]	E[3]	IT	IST	IRS	T_I[1]	I[1]	T_I[2]	I[2]
46	1-1	3	150	999	999	999	150					-150	-999	77	77	-150		
	2-1	1	50	999	119							-50	-999	127	54	-148		
	2-2	2	100	999	67							-100	-999	70	52	-148		
	3-1	1	50	999	135							-50	-999	124				
	3-2	2	100	999	80							-100	-999	82				
	3-3	3	150	97	-999							-150	-117	-999				
	3-4	1	50	999	126							-50	-999	139				
	4-1	1	50	32	-999			**47**	33	48	84	-50	-999	141				
	4-2	2	100	67	-999					**47**	84	-100	-999	91				
	4-3	3	150	999	81							-150	-999	79				
	4-4	1	50	999	142							-50	-999	137				
	4-5	2	100	999	99			48	33			-100	-999	90				
	4-6	3	150	32	-999			48	33	49	84	-150	-128	-999			**47**	-37
	4-7	1	50	999	146			48	33	999	999	-50	-999	141				
	4-8	2	100	32	-999			**47**	33	48	84	-100	-999	93				

46 4-2 Since IST is in refractory state (IRS = 91), the inhibition pulse is forfeited.
4-3 EST = 66 + E[3] = 66 + 84 = 150. ET = 150. EST ≥ ET, EST = 999, ERS = 81.
4-3 IST = -127 + I[2] = -127 - 37 = -164. IT = -150. IST ≤ IT, IST = -999, IRS = 79.
4-4 EST = 32 + E[2] + E[3] = 32 + 33 + 84 = 149. ET = 50. EST ≥ ET, EST = 999, ERS = 142.
4-5 EST = 32 + E[3] = 32 + 84 = 116. ET = 100. EST ≥ ET, EST = 999, ERS = 99.
4-7 EST = 32 + E[3] = 32 = 84 = 116. ET = 50. EST ≥ ET, EST = 999, ERS = 146.
4-8 Since IST is in refractory state (IRS = 93), the inhibition pulse is forfeited.
Next t = 47.

TIME	G-S	C	ET	EST	ERS	T_E[1]	E[1]	T_E[2]	E[2]	T_E[3]	E[3]	IT	IST	IRS	T_I[1]	I[1]
47	1-1	3	150	999	999	999	150					-150	-999	77	77	-150
	2-1	1	50	999	119							-50	-999	127	54	-147
	2-2	2	100	999	67							-100	-999	70	52	-147
	3-1	1	50	999	135							-50	-999	124		
	3-2	2	100	999	80							-100	-999	82		
	3-3	3	150	96	-999							-150	-116	-999		
	3-4	1	50	999	126							-50	-999	139		
	4-1	1	50	999	144					48	83	-50	-999	141		
	4-2	2	100	999	93							-100	-999	91		
	4-3	3	150	999	81							-150	-999	79		
	4-4	1	50	999	142							-50	-999	137		
	4-5	2	100	999	99			48	32			-100	-999	90		
	4-6	3	150	31	-999			48	32	49	83	-150	-999	79		
	4-7	1	50	999	146			48	32			-50	-999	141		
	4-8	2	100	63	-999					48	83	-100	-999	93		

47 4-1 EST = 31 + E[2] = 31 + 32 = 63. ET = 50. EST ≥ ET, EST = 999, ERS = = 144.
4-2 EST = 66 + E[3] = 66 + 83 = 149. ET = 100. EST ≥ ET, EST = 999, ERS = = 93.
4-6 IST = -127 + I[2] = -160. IT = -150. IST ≤ IT, IST = -999, IRS = 79.
4-8 EST = 31 + E[2] = 31 + 32 = 63. ET = 100. EST < ET.
End of simulation. EST and or IST for 4:1 through 4:8 in refractory state at same instant.

Excitation History			Inhibition History		
Time	Cell	Size	Time	Cell	Size
			≤ 45	4-1	1
				4-2	2
				4-4	1
				4-5	2
				4-7	1
				4-8	2
46	4-3	3	46	4-3	3
	4-4	1			
	4-5	2			
	4-7	1			
47	4-1	1	47	4-6	3
	4-2	2			

Result: Stopped response. Stop-RT < 45. Go-RT = 46. No cell (4-1 through 4-8) yielded excitatory discharge before inhibitory discharge.