Emergence of tri-phasic muscle activation from the nonlinear interactions of central and spinal neural network circuits

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Abstract


The origin of the tri-phasic burst pattern, observed in the EMGs of opponent muscles during rapid self-terminated movements, has been controversial. Here we show by computer simulation that the pattern emerges from interactions between a central neural trajectory controller (VITE circuit) and a peripheral neuromuscular force controller (FITE circuit). Both neural models have been derived from simple functional constraints that have led to principled explanations of a wide variety of behavioral and neurobiological data, including, as shown here, the generation of tri-phasic bursts.

Disputed origins of the tri-phasic EMG burst pattern

When humans make rapid, self-terminating limb movements, measurements of muscle activity during movement reveal a characteristic tri-phasic burst pattern. For example, in movements that require rapid elbow flexions, the EMG reveals an initial burst in the biceps (B1) followed by a burst of activity in the triceps (T1), followed by a smaller burst in the biceps (B2). Because the biceps' and triceps' bursts

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generate muscle forces that oppose each other's effects on the forearm, this burst pattern can be given a simple functional interpretation (e.g. Lestienne 1979; Karst and Hasan 1987). The B1 burst is necessary to accelerate the forearm to the desired movement velocity. Then the T1 burst is necessary to decelerate the arm and thereby halt the movement. The smaller B2 burst, which appears less reliably, may sometimes prevent the arm from reversing direction in the event of too large a T1 burst.

Though the function of the burst pattern now appears clear, its origin has remained a matter of dispute. Many observers (e.g. Hallett et al. 1975) have viewed the pattern as a clear indication of the existence of a central, pre-formed, motor program, whereas others have argued that the pattern is an emergent property of interactions between central and peripheral components of a neuro-muscular dynamical system (e.g. Bullock and Grossberg 1988; Feldman 1986). In this paper, we present simulation results which show that the basic tri-phasic pattern can emerge from interactions between central and peripheral components of the neuro-muscular dynamical system. Because both the central and peripheral components of the model are well-grounded in physiological, anatomical, and psychophysical data, the emergence of the tri-phasic burst can now be analysed as a consequence of interactions between components of a dynamical system rather than as a pattern that must be imposed at the periphery by the higher brain. After reviewing how the phenomenon emerges within the model, we will return to a brief discussion of the possibility that burst patterns may eventually be learned by the higher brain after initial genesis at the periphery.

The VITE and FLETE modules: Trajectory formation and force generation for trajectory realization

The VITE circuit is a neural network model constructed to explain a wide range of data on the kinematics and neurophysiology of planned, point-to-point reaching movements (Bullock and Grossberg 1988, 1989, 1991; Gaudiano and Grossberg this issue). For present purposes, the key properties of the model are: (1) that it is a central pattern generator capable of operating without sensory feedback; (2) that its output stage sends gradually changing reciprocal commands to
the two opponent muscles whose length changes (one increasing, one decreasing, whence 'reciprocal commands') are required to produce movement; and (3) that the duration and amplitude of the specified length changes are controlled by inputs to the VITE central pattern generator. In summary, as the first of two components of our composite model, we require a central circuit capable of generating a ramp-like change of prescribed amplitude and slope. This ramp-like change will specify the desired time course and amplitude of a decrement in agonist muscle length and a corresponding increment in antagonist length.

The second component of the composite model is the FLETE model of the peripheral neuro-muscular system, shown in fig. 1 (Bullock and Grossberg 1989, 1991). The ramp-like reciprocal com-
mands generated by the the VITE circuit appear as opponent inputs $A_1$ and $A_2$ at the top of the diagram. The FLETE circuit generates the forces needed to move the limb in obedience to VITE-controlled ramp-like change in the inputs $A_1$ and $A_2$.

Elsewhere, we have presented analyses and simulations in support of the thesis that the circuitry shown in fig. 1 enables separable control of muscle lengths (by descending signals $A_1$ and $A_2$) and co-contractive tension (by descending signal $P$). The need for such separable control in limbs with tunable compliance gave the model its name, which stands for Factorization of LEngh and TEnsion.

Fig. 1 graphically illustrates the hypothesis that the reciprocal commands $A_1$ and $A_2$ that control opponent muscle lengths are generated from different central sites than the co-contractive command $P$ that controls joint compliance. The hypothesis of separate central sites of origin for these two classes of control signals receives direct empirical support from studies of distinctive cell populations in motor cortex by Humphrey and Reed (1983). Another major aspect of the model is the branching of the $A_i$ signals to alpha-motoneurons, gamma-motoneurons, and \textit{Ia} interneurons. Such a branching was recently discussed by Baldissera et al. (1981), who wrote that 'The hypothesis of "$\alpha$-$\gamma$-linkage in reciprocal inhibition" postulates that neuronal systems acting in $\alpha$-$\gamma$-linked movements excite in parallel not only $\alpha$- and $\gamma$-motoneurons to agonists but also \textit{Ia} inhibitory interneurons to antagonists' (p. 529). They also cited data confirming this hypothesis in neuronal systems subserving both cat stepping movements and human voluntary limb movements. Readers interested in additional functional considerations underlying the model, or in further documentation of the biological reality of depicted cell types and connectivities, should consult our earlier papers (Bullock and Grossberg 1989, 1991).

In this paper we focus on a qualitative description of simulation results and their basis in model interactions. All aspects of the model, from the neurons with their membrane dynamics, to the mobile forearm segment with its geometry and mass, were specified mathematically via a system of algebraic and ordinary differential equations in Bullock and Grossberg (1989, 1991). In those reports, the operation of this system was studied by computer simulation, and additional computer simulations are reported below. Unless otherwise noted, subsequent references to the activity of muscles, neuron pools, etc.
should be understood as references to the behavior of our mathematical representations of these entities.

Emergence of the tri-phasic burst in response to a fast ramp change in descending reciprocal commands

Fig. 2 shows plots of all important FLETE model variables during the simulation of a single rapid movement. Because the EMG measures muscle activations caused by and proportionate to immediately prior activations of the alpha-motoneuron pools, the appropriate place to look for a tri-phasic burst is in fig. 2C, where agonist channel alpha-motoneuron activity appears as a solid curve and antagonist channel activity appears as a dashed curve. A clear tri-phasic burst pattern, similar in peak timing, relative peak magnitude, and degree of temporal overlap to those often observed in vivo (e.g., Lestienne 1979), can be seen. Yet the time-varying inputs to the model, shown as the lower two curves in fig. 2I, have the form of simple ramps (which approximate VITE circuit output signals): a steep increment in the agonist channel's input, $A_1$ (solid line), and a steep decrement in the antagonist channel's input, $A_2$ (dashed line).

In our simulation, descending input $P$ to the alpha-motoneurons was constant, and thus inputs $A_1$ and $A_2$ to the alpha-motoneurons, gamma-motoneurons and Ia interneurons were the sole time-varying descending inputs (see fig. 1). Therefore the much different temporal pattern of alpha-motoneuron activities visible in 2C — that is, the triphasic burst — must be arising as a consequence of peripheral interactions generated in response to the ramp inputs.

In the simulation shown, the burst pattern emerges as follows. As fig. 1 makes clear, the same ramp changes in alpha-motoneuron inputs co-occur in gamma-motoneuron inputs. Just as the alpha-motoneurons drive contraction of the large agonist extrafusal muscles in an attempt to move the forearm, the gamma-motoneurons drive contraction of the small agonist intrafusal muscles. However the latter muscles, to which spindle stretch receptors are attached, are free to contract without significant delay imposed by the forearm load. Thus it soon occurs that the agonist intrafusal muscle is contracting more quickly than the agonist extrafusal muscle. This discrepancy causes a response in the spindle organ similar to what would be observed during a high-velocity muscle stretch imposed on the arm by an external force.
**TRI-PHASIC EMG GENESIS**

Fig. 2. FLETE model simulation of the generation of a tri-phasic muscle activation pattern. Key: In all panels except panel A, solid lines plot variables in the agonist channel and dashed lines plot variables in the antagonist channel. (A) Forearm angular position (solid trace) and angular velocity (dashed trace). (B) Agonist and antagonist contractile states. (C) Agonist and antagonist alpha motoneuron activities. (D) Agonist and antagonist muscle forces. (E) Agonist and antagonist composite spindle feedback signals. (F) Agonist and antagonist gamma motoneuron activities. (G) Agonist and antagonist Ia interneuron activities. (H) Agonist and antagonist Renshaw cell activities. (I) Agonist and antagonist channel descending inputs $A_1$ and $A_2$ (lower pair of traces) and intrafusal muscle contractions (upper pair of traces). A tri-phasic burst pattern appears in the alpha-motoneuron activity plot (panel C) despite a mono-phasic ramp change in descending inputs (panel I).
As shown in fig. 1, the responding agonist spindle organ sends an excitatory feedback pathway directly to the agonist alpha-motoneuron, where it can generate a large burst. This is so because in the simulation, as in nature, the stretch velocity component of the spindle feedback signal has a high gain. Indeed the large spindle-based feedback signal generated in response to the rapidly growing discrepancy between intrafusal and extrafusal contractions can be seen in fig. 2E to coincide with the onset of the first agonist burst in fig. 2C. This agonist alpha-motoneuron burst generates an agonist contraction (fig. 2B) which in turn generates the large force (fig. 2D) needed to propel the model forearm mass at a high rate, as shown by the dashed line in fig. 2A. When the agonist extrafusal contraction (fig. 2B) exceeds the rate of intrafusal contraction (upper, solid curve in fig. 2I), stretch velocity becomes negative and the high gain component of the agonist's spindle signal collapses (fig. 2E). Because of the rapid agonist shortening, the antagonist extrafusal muscle is soon beginning to lengthen more quickly than the antagonist intrafusal muscle, so the antagonist spindle registers a high-velocity stretch (fig. 2E, dashed line). This causes a burst of activity in the antagonist alpha-motoneuron pool (fig. 2C, dashed line), an antagonist extrafusal muscle contraction (fig. 2B), and an antagonist braking force (fig. 2D) which does indeed decelerate the moving limb (fig. 2A).

In our simulated model, the second agonist burst does not arise in the same way as the first. As can be seen in fig. 2C, it begins prior to the small second burst of agonist spindle activity shown in fig. 2E. Instead of being initiated by spindle activity, the second agonist burst is driven by a temporary collapse (fig. 2G) of inhibition from the antagonist channel's Ia interneuron (IaIN). This collapse is in turn driven by the antagonist channel Renshaw burst (fig. 2H) associated with the antagonist alpha-motoneuron burst. Or, now working forward in time, we see that the antagonist alpha-motoneuron burst drives an antagonist Renshaw cell burst, which inhibits the antagonist IaIN, thereby transiently releasing the agonist alpha-motoneuron from antagonist IaIN inhibition. The same Renshaw inhibition of the antagonist IaIN also releases the agonist IaIN from inhibition and thereby produces the marked burst seen in fig. 2G (large deflection of the solid curve). Finally, after being initiated as just described, the second agonist burst is sustained by the late agonist spindle activity shown in fig. 2E. This spindle activity is due to the stretch associated with the reversal of direction visible in fig. 2A.
Our earlier papers on the FLETE model (Bullock and Grossberg 1989, 1991) gave a full treatment of the behavioral function of the unique interneuronal connectivity pattern responsible for the complex dynamics just described. In particular, we showed that the size principle of alpha-motoneuron recruitment could begin to achieve a wide force range at any muscle length, but also could cause distortions in the lengths commanded by the descending signals ($A_1$, $A_2$). It was also shown, however, that the opponently organized Renshaw-Ia system could automatically compensate for this positional distortion. Thus the model rationalized a formerly mysterious interneuronal connectivity pattern, which was already known to exist in vivo (Baldissera et al. 1981; Pompeiano 1984; Renshaw 1946; Ryall 1970). The present results show some of the dynamics associated with this same connectivity scheme during rapid movement.

**Parametric determinants of the burst pattern**

Though we have not yet completed our studies of parametric determinants of the burst pattern, simulations have already replicated the main trends in the data. In particular, and as expected given the reasoning in the third section, the phenomenon depends on the steepness of the ramp inputs (desired movement speed) and the mass of the forearm load (Lestienne 1979; Karst and Hasan 1987). The faster the desired movement, and the larger the load, the larger the initial discrepancy between agonist intrafusal and extrafusal contraction rates, and so forth.

It is also worth noting that it was not necessary to change any parameters of the FLETE model from those values chosen in our earlier simulations (Bullock and Grossberg 1989, 1991), which were wholly concerned with the equilibrium properties of the model. In fact, the only changes we made were to add a stretch velocity component to the spindle feedback signal and assign it a high multiplier or gain (25 in the simulation shown).

**Generality of the FLETE circuit and the tri-phasic EMG pattern**

In our theory, the biological design abstracted by the FLETE model equations can be conceptualized as a phylogenetic adaptation to the
problem of achieving separable control of limb position and joint stiffness (or its reciprocal, compliance) in systems where stiffness must be scaled over a large range. Thus one might expect such a design to control postural, load-bearing limb-segments. Consistent with this expectation, Pompeiano (1984), observed that correlations between different motoneuron species and the incidence of recurrent axon collaterals (the alpha-motoneuron to Renshaw projection) seemed to indicate 'that recurrent inhibition is primarily concerned with the control of proximal muscles (limb position) rather than of distal ones (movement of the digits)' (p. 467). This correlation has held through many subsequent studies (e.g. Hamm 1990). By comparison, we note that multi-phasic EMG bursts have been observed in single digit (thumb) movements (Marsden et al. 1983) as well as in movements about more proximal joints such as wrist and elbow. This is compatible with the anatomical data because the Renshaws are not necessary to generate the multi-phasic burst. In subsequent research, we hope to explain differences in the fine structure of EMG patterns by reference to evolutionary specializations of the FLETE design, including versions with no Renshaw subsystem.

Conclusions: Reactive vs. predictive control of position and force

Our results and their apparent basis in model dynamics generally support the heuristic derivation of the tri-phasic burst offered by Feldman (1986), who also emphasized the critical role of the spindle system's high gain response to stretch velocity (as opposed to its low gain response to stretch amplitude). Our new explanation of the second agonist burst, moreover, is consistent with the widespread observation that it often occurs too late to make a significant contribution to limb kinematics.

Two implications may be drawn from our discovery that the phenomenon emerged without need to adjust FLETE model parameters. First, the pattern appears to be a robust consequence of system components and geometry, given the addition of a stretch velocity feedback. Second, the FLETE model, despite its many simplifying assumptions and lumping of neural populations into single nodes, appears to have captured many of the most salient equilibrium and transient properties of the peripheral neuro-muscular system as it pertains to control of single joint movements.
The FLETE model hereby clarifies how evolution may have fashioned a spinal circuit capable of factoring position and force control in a reactive fashion. In addition, the FLETE model suggests how spinal components may create error signals to be used by the cerebellum to learn the parameters of predictive feedforward control (Bullock and Grossberg 1991). Feedforward control can achieve pre-emptive error compensation in high-performance skills where speed, accuracy, and predictable completion times are essential.

In the light of this distinction between reactive and predictive control, our demonstration that the tri-phasic burst can emerge in the manner described by no means rules out the possibility that some multi-phasic EMG bursts are eventually produced primarily as a result of feedforward commands. Indeed such a result can be expected from the general thesis (Ito 1984; Grossberg and Kuperstein 1989; Kawato et al. 1987), now widely held, that cerebellar learning frequently acts to pre-empt the occurrence of the kind of errors that played such a key role in the genesis of the phenomenon (see also Vilis and Hore 1986). However, because cerebellar learning requires the occurrence of trajectory errors to proceed, we believe that the present account will remain the correct explanation for the initial genesis of the tri-phasic burst.

References


