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2002-02

Motorneuron Recruitment

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February, 2002

Technical Report CAS/CNS-02-001

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To appear in *The Handbook of Brain Theory and Neural Networks*, Second edition,
(M.A. Arbib, Ed.), Cambridge, MA: The MIT Press, 2002.

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INTRODUCTION

Motoneurons are neurons that directly innervate muscle fibers. When motoneuron discharges cause muscle fibers to contract, the resultant forces oppose static loads, and produce active accelerations and decelerations of limb segments. Moreover, co-contractions of opposing muscles allow us to stiffen joints and thereby maintain desired postures despite perturbations of unexpected magnitude and direction. Because of the direct anatomical link between motoneurons and contractile fibers, there is a close relationship between motoneuron activity and force production.

A motoneuron together with the contractile fibers that it innervates constitute a motor unit. The range of forces producible by one motor unit is small. To make it possible to generate large forces, motor units must be combined into larger aggregates, and the results of such aggregation are the muscles. Immediately associated with each muscle is a population or pool of motoneurons. Muscles are therefore composite structures, and their force-generating components, the motor units, are typically heterogeneous. For example, muscle fibers differ systematically in fatiguability and the associated motoneurons differ systematically in their size. How are these heterogeneous aggregates of force-generating elements recruited in the service of reflexes, voluntary movement and posture? Such task-dependent recruitment is achieved by a combination of motor unit and neural network specializations.

Consider the simple question of control of force magnitude. If any excitatory input were sufficient to cause simultaneous excitation of all motor units, then the minimum force produced by the aggregate would be much too large for most purposes. To produce accurate movements, forces must be finely graded in response to the input to the motoneuron pool. The fine grading of forces required for accuracy favors a design that allows both partial activation of the motoneuron/fiber pool and finely graded changes, up or down, from preexisting states of activation.

Such force grading by a cells/fibers aggregate provides a functional context for understanding the size principle of motoneuron recruitment proposed in 1965 by Henneman, Somjen and Carpenter (see Burke, 1998, for a review). The size principle encompasses many aspects of the design of motoneuron pools and their embedding within the sensory-motor system. In this design, an excitatory input often reaches all elements of the motoneuron pool at the same time. However, elements of the motoneuron pool differ in their activation thresholds. Because there exists a distribution of threshold values from small to large, the larger the excitatory input to the pool, the more elements become active. This enables a continuously varying input signal to produce a graded force response from the muscle. As the excitatory input to the pool grows, motoneurons are recruited in order by size from smallest to largest, because motoneurons with larger somatic volumes also have higher thresholds. As excitatory input declines, or inhibitory input increases, motoneurons are de-recruited in order by size, from largest to smallest.

The grading of force by recruitment, which is necessarily quantal, is supplemented by finer grading through firing rate modulation of individual cells, because each cell's firing rate is sensitive to input fluctuations in its supra-threshold range. This design affords

finely graded increments and decrements in force over the entire range of muscle force capability.

It might appear that the size principle serves to make each spino-muscular force generator a fixed-gain, near-linear, amplifier of excitatory inputs. However, many factors complicate the situation. First, the gain is not fixed because muscle force can become decoupled from motoneuron pool activation if a contraction-opposing load causes muscle yielding, or if the muscle fatigues. Second, the amplification function is often faster-than-linear because motoneurons with larger cell bodies, and thus higher recruitment thresholds, typically project by larger, faster-conducting axons to more muscle fibers, each of which exhibits shorter twitch contraction times. Third, twitch contractions of muscle fibers are slow relative to rapid fluctuations of excitatory inputs to motoneurons. Fourth, muscle obeys a force-velocity law: force output from a muscle decreases as its shortening velocity increases. Fifth, the conventional delimitation of a motor unit, although minimal, is somewhat arbitrary. Several other closely linked neural and sensory constituents appear in most mammalian muscle control systems as part of the apparatus for force generation (cf. Burke, 1998). For example, before exiting the spinal cord, the axons of most alpha-motoneurons give off collaterals that excite Renshaw cells (RCs), which inhibit those same alpha-motoneurons. Sixth, the net torque developed at a joint depends upon both mechanical advantage and the balance of forces created by groups of muscles arranged into synergistically antagonistic sets. Each of these considerations reveals a need for network control of recruitment, to ensure that opponent muscle sets generate the right force balances through time.

COMPENSATIONS FOR FATIGUE AND YIELDING

Muscle fatigue and yielding make the functional relation between pool activation and force inherently variable, and network interactions provide compensations that reduce the variability in this linkage. Nichols and Houk (1973) argued that two feedbacks from muscle receptors to spinal motoneuron pools cooperate to reduce variability in muscle stiffness, the ratio of muscle force changes to muscle length changes. Muscle yielding events reduce stiffness while also increasing the activity of stretch-sensitive receptors, the spindles, and decreasing the activity of tension-sensitive receptors, the Golgi-tendon organs (GTOs). Because spindle feedback directly excites alpha-motoneurons via type Ia sensory fibers, whereas GTOs can inhibit motoneurons via Ib interneurons, both feedbacks are compensatory. Often noted is that GTO feedback also has appropriate characteristics to compensate for muscle fatigue. Bullock and Grossberg (1989) argued that the covariation of motor unit sizes and contraction rates is also compensatory for yielding.

LINEARIZATION OR EQUALIZATION OF POOL RESPONSES

By itself, the covariation of recruitment threshold, number of fibers contacted, and fiber contraction rates with motoneuron size can produce a faster-than-linear relationship between excitatory input to the motoneuron pool and the force output of the muscle, at least under isometric conditions when the system is not approaching saturation. Akazawa and Kato (1990) and Bullock and Grossberg (1989) independently proposed that Renshaw

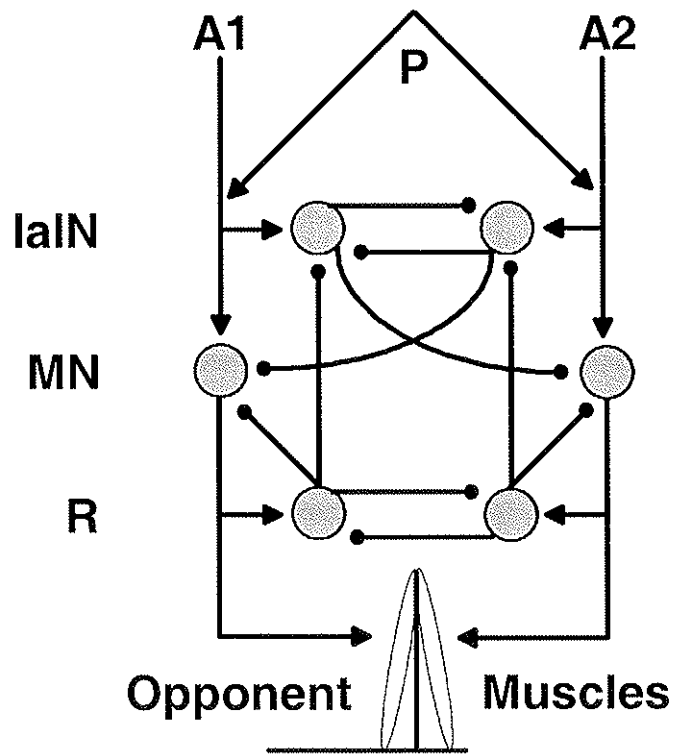


Figure 1. Partial connectivity of the FLETE model for independent control of joint angle and joint stiffness. To set desired joint angle, the higher brain reciprocally adjusts descending signals A1 and A2 directed to two opposing alpha motoneuron (MN) pools that project to opposing muscles. Descending signal P to both motoneuron pools adjusts joint stiffness without modifying joint angle if increments in P lead to equal increments in the force outputs of the two opposing muscles. Renshaw (R) cell feedbacks, among others, compensate for non-linearities in the motoneuron response function and thereby help assure equal force increments in the two muscles affected by P. Renshaw feedback disinhibits opponent MNs via the Ia interneurons (IaIN). Arrow and dot line-endings respectively indicate excitatory and inhibitory synapses.

feedback improves this transduction. The Akazawa and Kato analysis treated a single motor unit pool, and showed that inhibitory Renshaw feedback may be able to linearize the relationship between excitatory inputs and force outputs. Bullock and Grossberg

sought to explain how spinal circuitry enabled the higher brain to achieve independent control of joint angle and joint stiffness. Accordingly, these authors analyzed the "FLETE" circuit (Figure 1), which encompassed a lumped pair of motor unit pools associated with biomechanically opposed muscles. By factoring the Length and Tension properties of muscle, the FLETE network allows a descending co-contraction signal to stiffen and thereby stabilize the joint at any desired angle. Available data (Humphrey and Reed, 1983) indicate that voluntary stiffness adjustments are achieved by varying an excitatory signal relayed to both opponent motoneuron pools. Bullock and Grossberg showed that in the absence of Renshaw feedback, a descending co-contraction signal would generally be unequally amplified by recruitment events within opposing motoneuron pools. Such unequal amplification would lead to an undesired joint rotation as well as to a change in joint stiffness. They then showed that Renshaw-mediated feedback could help guarantee independent control of joint stiffness and joint angle by equalizing the two pools' amplifications of the co-contraction signal. This equalization, which need not involve global linearization of recruitment, is achieved by a local circuit that incorporates mutual inhibition between opponent Renshaw pools and between Ia reciprocal inhibitory interneurons, which, like alpha-motoneurons, are inhibited by Renshaw cells (RCs).

This view of the role of RCs is consistent with data that contradict alternative views. Pratt and Jacobs (1987) showed that RCs fired in phase with alpha-motoneurons during fictive locomotion, but that they were not needed for generation of the locomotor cycle. This disconfirmed the hypothesis that they were an integral part of the spinal locomotor generator. Lindsay and Binder (1991) observed that although steady-state Renshaw inhibition caused similar synaptic currents in alpha-motoneurons of different sizes, IPSP amplitudes did correlate with cell size. They concluded that "the biggest impact of [RC] inhibition will be on the force output of motoneurons firing on the steep part of their force-frequency curve" (p. 176).

A subsequent extension of the FLETE model showed that the triphasic EMG bursts characteristic of rapid self-terminated joint rotations emerge within an arm-controlling network activated by monophasic descending control signals, if the network incorporates velocity-sensitive muscle spindles. Contreras-Vidal et al. (1997) showed that the FLETE model is applicable to multi-joint arm movement control using both mono- and bi-articular muscles, and that the independent control property is enhanced by the incorporation of sensory feedbacks from spindle (Ia), GTO, and joint receptors. Moreover, van Heijst et al. (1998) showed that connection weights consistent with the independent-control property will self-organize in the circuit of Figure 1 if local synapses are adjusted by a Hebbian learning process while the circuit is stimulated by a rhythmic input. Their developmental simulation modeled how such spinal circuits self-tune during prenatal episodes of rhythmic activity in avian and mammalian embryos.

ADAPTIVE CENTRAL CONTROL OF MOTONEURON GAIN

Renshaw cells also mediate descending modulation of the motoneuron recruitment process. Stimulation in nucleus interpositus (NIP) of the cerebellum, or in its target, the Red Nucleus (RN), which projects to spinal pools via the rubro-spinal pathway, enhances

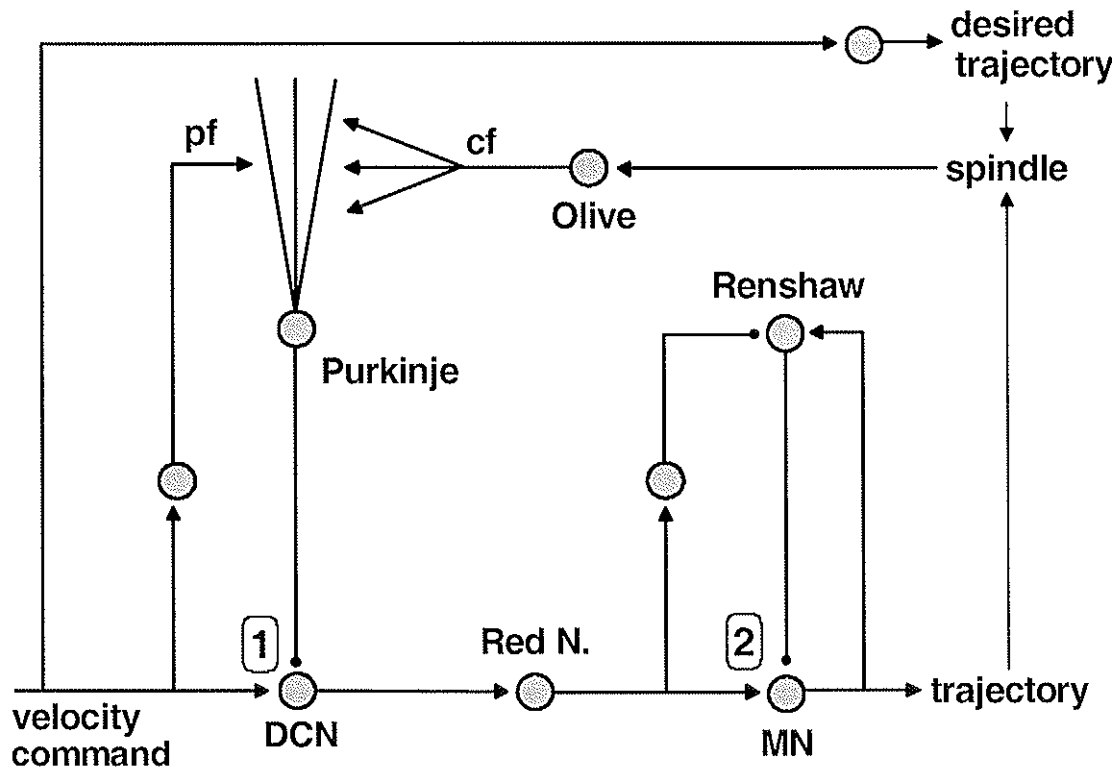


Figure 2. Network model incorporating two sites for controlling motoneuron excitation by release from inhibition. Prior to learning, a velocity control signal directed toward a muscle via the deep cerebellar nuclear (DCN) pathway will have a negligible effect due to Purkinje (P) cell inhibition of DCN sites and Renshaw cell inhibition of alpha motoneurons (MN). However, trajectory errors detected by muscle spindles activate the inferior olive, whose climbing fibers (cf) reach the dendrites of Purkinje cells. Climbing fiber activity causes long term depression of coactive parallel fiber (pf) synapses that excite Purkinje cells. Depression of Purkinje excitation causes disinhibition of DCN sites. This “opens the gate” for the velocity control signal to activate the Red Nucleus. The Red N. both excites alpha motoneurons and inhibits Renshaw cells.

the gain of the monosynaptic stretch reflex by inhibiting RCs, thereby releasing alpha-motoneurons from recurrent inhibition. The NIP or RN stimulation also excites motoneurons. Bullock and Grossberg (1989) proposed that the implied bivalent rubral projection to RCs and alpha-motoneurons afforded adaptive, i.e., learning-based, control of the “gain” of movement commands directed to motoneuron pools. Contreras-Vidal et al. (1997) introduced a neural network comprising a central trajectory generator, an extended FLETE model, and a model cerebellar network capable of learning to modulate motoneuron recruitment via a bivalent output to RCs and alpha-motoneurons. Simulations of the circuit (Figure 2) showed that if the cerebellum received both a desired velocity signal and an error feedback routed from spindles to cerebellum via the inferior olive, then a learning-adjusted cerebellar output substantially enhanced the dynamic

tracking characteristics of the limb by transiently exciting, and removing inhibition from, the agonist motoneuron pool (Figure 2). This model is consistent with recent biophysics-based models of cerebellar adaptive timing (e.g., Fiala et al. 1996), and with common observations of phasic RN and interpositus activity during learned movements. A closely related modeling treatment, encompassing cerebellar modulation of the Figure 1 circuit in the context of realistic sensory lags, has recently appeared (Spoelstra et al. 2000).

ROLES OF MOTOR CORTEX IN MOTONEURON RECRUITMENT

Many cells in the primary motor cortex (M1) of primates excite motoneurons via mono- or short poly-synaptic pathways, and the pathway for the long-loop stretch reflex traverses M1. Moreover, cooling of the dentate nucleus of the cerebellum, which affects M1 via the thalamus, eliminates anticipatory, force-related, components of normal M1 activity. Many studies have strongly implicated M1 in load compensation achieved by direct recruitment of motoneurons, although a subset of M1 cells are relatively load insensitive (Kalaska et al. 1989). Yet other studies have appeared to implicate M1 in a high-level representation of the direction of movement in Cartesian space. Recently, two models have begun to address the dilemma posed by these observations. The extended Vector Integration To Endpoint (VITE) model of Bullock et al. (1998) proposed a circuit involving 6 electrophysiologically identified cell types in M1 and parietal area 5 to explain the distinct computational roles of load-sensitive and load-insensitive cells in both arm trajectory generation *and* load compensation. This model's relatively load-insensitive cells have poly-synaptic links to alpha-motoneurons, whereas the most load-sensitive cells have mono-synaptic links. Todorov (2000) proposed a model (pertinent primarily to load-sensitive cells) based on the assumption that M1 recruitment compensates for the negative effects of the force-velocity law on the ability of muscle to sustain force when shortening at a significant velocity.

If some M1 cells directly control motoneuron recruitment, and thus force generation, then theories of sensory-motor transformations (e.g., Barreca and Guenther, 2001) predict that the preferred spatial directions of such M1 cells must be strongly posture-dependent – and they are. Several recent simulations based on this premise have succeeded in predicting posture- and trajectory-dependent tuning properties of M1 cells and the muscles to which they project (Ajemian et al. 2001; Scott and Kalaska, 1997).

DISCUSSION

Neural network analyses have begun to clarify how local spinal circuits cooperate with central adaptive circuits for task-dependent control of motoneuron recruitment, but many basic questions remain to be addressed. Too little is known about the pathways for descending control of gamma- versus alpha-motoneurons. Also, the behavioral functions of many known aspects of the recruitment system, such as motoneuronal plateau potentials, remain to be elucidated by computational analyses. Models must also be elaborated to accommodate the unique connectivities that govern recruitment in different species, which differ dramatically in biomechanical, behavioral, and neuronal specializations.

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