

Neural Control of Locomotion

How do all the data fit together?

Gerald E. Loeb

Locomotion is a particularly richly studied but frustrating aspect of biology. Much is known about individual components of the process, but there is no general hypothesis for how it works.

Vertebrate locomotion is distinguished from invertebrate locomotion by the conjunction of two features. The skeletal movements are complex but they are usually regular, repeating sequences (see Hildebrand page 766 this issue), and most of the organisms are massive enough that the amount of energy and force invested in moving the whole animal constitutes a significant percentage of its metabolic economy and is a significant determinant of the details of its body plan (see Biewener page 776 this issue).

At the phenomenological level, we have good descriptions of the movements and even the mechanics involved in walking, swimming, and flying. At reductionistic and comparative levels, we have catalogued and differentiated the properties of skeletons (see Gordon page 784 this issue), muscles (see Weeks page 791 this issue), and sensory and motor neurons. We even know that the critical neural circuits for generating locomotor patterns are, for the most part, confined to a relatively small and primitive part of the central nervous system, the spinal cord.

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No machines successfully walk, swim, or fly using the principles animals employ

We might be able to convince ourselves that all of these descriptions, in toto, constitute an understanding of animal locomotion, except for one telling failure: there have been no successful machines that walk, swim, or fly using any of the principles that have served animals so well. It is not for lack of trying. Although boats with fins and planes with flapping wings are now reminiscent of Jules Verne, walking machines with legs remain an active interest of industries such as forestry, mining, and defense.

Modern materials and electronics can provide mechanical assemblies, actuators, and sensors that are stronger, lighter, faster, and simpler than the corresponding bones, muscles, and receptors of living organisms. Yet even a child knows that robots "walk funny," characterized by graceless and inefficient gaits on regular terrain and prone to disaster in the face of unexpected obstacles.

The problem is that we do not really have a theory of control for locomotion. This article considers the relationship between the epiphenomena known as reflexes and the moment-to-moment regulation of individual muscles. It describes the

properties of the entirely conceptual entities called central pattern generators, which are thought to control the sequential recruitment of these muscles. And it confronts the almost bewildering complexity of the few neural circuits that have been identified to date, circuits that presumably contribute to both levels of control.

The amount of data available is impressive and the neurophysiological research involved in obtaining it has been arduous, even heroic. But the general biologist will likely and rightfully be uneasy with how it all might fit together. The last sections of this article look to comparative, developmental, and evolutionary biology for a perspective that is often missing in the current rush to reductionism.

Reflexive control of muscles

Each muscle is endowed with its own ensemble of sense organs that report on mechanical conditions within the muscle, including its length and velocity of stretch and contraction (muscle spindles) and its force output (Golgi tendon organs). In most limb muscles, the neurons from these sense organs carry signals to other neurons, called alpha motoneurons, which activate the muscle fibers. This connection may be direct (via a single synapse) or through neurons within the spinal cord (called interneurons). These pathways, or projections, give rise to reflexes such as the clinical tendon jerk, in which a sudden stretch of the muscle spindles results in a rapid contraction from reflexively activated

muscle fibers (reviewed by McCrea 1986). In addition, each mammalian spindle contains several different types of intrafusal muscle fibers under the independent control of special gamma motoneurons. The output of the spindle afferents represents a complex sum of extrinsic muscle length changes and intrinsic contractions of the intrafusal muscle fibers (reviewed by Loeb 1984).

These arrangements of proprioceptive sensory feedback are reminiscent of the feedback loops used in the servocontrol of industrial torque motors. Thus it is not surprising that servocontrol loops have figured prominently in hypotheses for motor control. Figure 1 shows a historical progression of such schemes that were designed to account for patterns of connection inferred from large numbers of animal and human experiments.

More recently, interest has shifted from the control of individual muscles to the control of individual joints through the balance of forces in antagonistic muscle pairs. By specifying an operating point for the stiffness of each muscle, the central nervous system effectively could specify a target position at which the system is in equilibrium (Berkinblit et al. 1986, Bizzi et al. 1982). Servocontrol for such a push-pull system requires reciprocal connections, in which the effects of feedback signals from any given source are opposite for the two antagonistic muscles. Figure 2 shows known projections of reciprocal interneurons that could subserve such control (Baldissera et al. 1981).

Limb mechanics

Unfortunately, all of the above are basically static control schemes. In principle, continuous movements such as locomotion could be represented by a dynamic trajectory of equilibrium positions. However, for most animals the goal of locomotion is not a sloth-like gliding through a series of stable postures.¹ Rather it is to maintain a dynamic equilibrium in which postural stability and energy

¹This scheme has been the strategy of most legged robots, but see Raibert (1986) for some surprisingly simple and effective strategies for achieving dynamic equilibrium in high-speed movements of hopping machines.

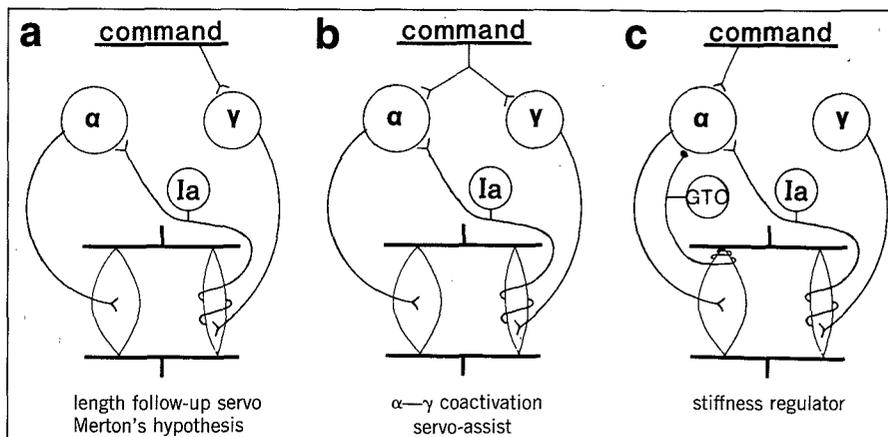


Figure 1. Previously proposed servocontrol systems. The muscle spindle afferents (Ia) can be driven by intrinsic activity of gamma motoneurons (γ) as well as extrinsic length changes; their synaptic projections excite the alpha motoneurons (α ; γ -shaped ending) controlling the extrafusal muscle fibers. a. Merton (1953) originally proposed that the length of a muscle could be commanded by activating the gamma motoneurons alone, with consequent alpha motoneuronal excitation via the Ia pathway, until the muscle shortened sufficiently to off-load the spindles. b. Recordings of spindle activity during voluntary contraction in humans (Vallbo 1974a, b) led Granit (1975) to propose that the alpha and gamma motoneurons were coactivated to form a servoassisted system under direct descending control. c. Houk (1979) incorporated the negative feedback (ball-shaped ending) from the Golgi tendon organs in proposing that the spinal cord regulated the muscle's stiffness (the ratio of force and length changes in response to a perturbation). (Adapted from Loeb 1984.)

efficiency depend on the anticipation of limb trajectories, which are determined at least as much by inertia as by muscular effort.

For example, the motion of the shank and foot during the swing phase of the cat hind limb depends on translational accelerations of the pelvis that propagate through the pendant limb like the oscillations of a marionette (Hoy and Zernicke 1985). This mechanical coupling saves energy, because these rapid movements would be inefficient to produce by contraction of the individual muscles operating across each joint. If the motion is perturbed (for example, by contact with an obstructing object), the response is not an attempt to return to the desired course, but rather a complex "stumbling corrective reaction" (Forssberg 1979) designed to get around the unseen object. Both of these strategies appear to reflect a control scheme that is based not on individual muscles or joints but rather on the mechanics of the whole limb.

The dynamic mechanical properties of a multisegmented limb are not a simple combination of the properties of the individual joints for at least

three reasons:

- Movement at any particular joint has an effect on foot position, for example, that depends on the orientation of the proximal segment defining that joint, which in turn depends on the angles of all the joints proximal to it.
- Intersegmental coupling causes motion at any joint to generate torques at all joints (consider the marionette). These effects can be surprisingly large and even counterintuitive (Zajac and Gordon 1989).
- Many muscles cross more than one joint. Their torques at each joint cannot be set independently by single-joint controllers. The force output of such muscles in response to the commands of their motoneurons depends on their net length and velocity, which is influenced by motion at all joints that they cross.

Spinal cord circuitry

If the control of any single muscle depends on postural and dynamic conditions throughout the limb, then we might expect rather more complex control circuits than those shown in

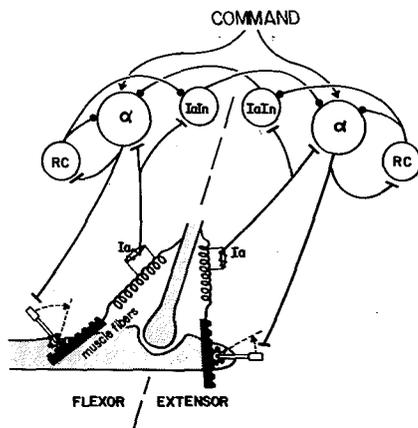


Figure 2. Reciprocal control of antagonist muscle pair. A joint tends to reach an equilibrium position in which equal torques are contributed by the antagonistic muscles acting on that joint. Because of the length-dependency of force output of muscles (arising from both intrinsic properties of their myofilaments and extrinsic organization of stretch reflexes), they may be modeled as springs whose stiffness is established by the motor commands (Bizzi et al. 1982; illustration adapted from Kandel and Schwartz 1984). Here the alpha motoneurons (α) are shown connected to pull on ratchet mechanisms in series with springs. Sensory feedback in such systems appears to be reciprocally organized. The spindle afferents of one muscle (Ia) directly excite the local alpha motoneurons as well as inhibitory interneurons (IaIn) that act to reduce the output of alpha motoneurons of the antagonist muscle. These inhibitory interneurons also turn the self-inhibition of motoneurons via the Renshaw cells (RC) into net excitation (actually disinhibition) of the antagonist motoneurons.

Figures 1 and 2, with motoneurons receiving sensory feedback not only from their own muscles, but also from many other muscles and joints. In fact, that is what neurophysiologists have discovered when such circuits have been traced through the spinal cord using electrophysiological and histological techniques.

Figure 3 shows a more realistic picture of the input-output connectivity of one neuron, called the Ib-inhibitory interneuron (Harrison and Jankowska 1985), which appears only as a ball-shaped ending signifying a logical inverter gate in Figure 1. This neuron converts excitatory synapses from Golgi tendon organs into inhibitory synapses onto the motoneurons supplying the muscle fibers

whose force is sensed by the tendon organ. Many other equally complex interneurons undoubtedly remain to be discovered. Methodological problems have generally precluded systematic identification of interneurons that synapse on other interneurons instead of projecting directly to identifiable motoneurons.

Central pattern generators

Although all of this sensory feedback is undoubtedly important, much of the initiation and timing of muscle action seems to be the work of a self-oscillating network of interneurons in the spinal cord that produces a cyclic pattern of signals. The most direct demonstration comes from patterns of electrical activity recorded from muscle nerves in cats that have had the brain severed from the spinal cord surgically and the muscle contractions blocked pharmacologically. Despite the complete absence of movement to produce sensory feedback, the spinal cord can produce sequences of motoneuronal activity that are similar to those recorded during normal walking (Perret 1983).

Recently, it has been shown that isolated sections of the spinal cord as short as two or three vertebral segments produce cyclical outputs (Grillner 1985), suggesting that locomotor patterns are generated by a simple, localized, and presumably primitive circuit in the spinal cord, the so-called central pattern generator (CPG). However, there is little agreement on the nature of this oscillator or the identity of the interneurons from which it is formed.

At first, it seemed that gaits such as walking and trotting could be divided into just two reciprocating phases of muscle action in each limb (Engberg and Lundberg 1969), corresponding to the stance phase (requiring mostly extensor, load-bearing muscles) and the swing phase (requiring mostly flexor, foot-lifting muscles). This model suggested that the CPG worked like a flip-flop (Lundberg 1980), a common circuit used to control timing in computers and one easily realized by reciprocally connected interneurons.

However, more recent EMG studies of a wider range of muscles have noted that the amplitude and timing

of each muscle's natural activity may be controlled individually (Grillner 1986). Furthermore, the reflex pathways projecting from sensory to motor neurons may be turned on and off (gated) at different points in the step cycle so as to change the behavioral response to a given mechanical perturbation (Abraham et al. 1985). This evidence suggests that the CPG must have more than two internal states. Alternative models to the simple flip-flop include ring-cycle oscillators and systems of loosely coupled but independent oscillators, such as may control the segmented axial muscles used by fish for swimming (Grillner 1985).

Scaling of motor control

Comparative biologists have a longstanding interest in the scaling (allometry) of musculoskeletal structure in animals of different sizes. Consider the strength-to-weight ratio of two similar structures, one of which has all of its linear dimensions ten times greater than the other's. Because strength is related to cross-sectional area (x^2) whereas mass depends on volume (x^3), the larger structure has a strength-to-weight ratio that is only 10% that of the smaller structure. Therefore, large animals appear to be built much more sturdily than scaled-up versions of small animals (McMahon and Bonner 1983).

Dimensional changes affect neurophysiological as well as mechanical function. One of the limiting factors in achieving stable control in any motor system (robotic or biological) is delay in the feedback circuits. If corrective responses are initiated on the basis of outdated information, the system will tend to overcorrect; if the strength (gain) of the feedback circuit is high enough, the system may oscillate wildly. In the neuromuscular system, significant delays occur at every stage, including the transmission of impulses along sensory axons to the spinal cord, processing of sensory information through interneuronal circuits, transmission back to the muscle along motor axons, spread of action potentials along muscle fibers, and release and diffusion of calcium ions to catalyze the formation of force-producing cross-bridges. In animals of different sizes, the various delays scale in quite different ways, suggest-

ing that the animals must use rather different control strategies.

At a simple level, consider just the control of large versus small muscles having simple architecture (parallel fibers). The larger girth is achieved with a larger number of muscle fibers, with relatively little increase in individual fiber diameter. Electromechanical properties of the fibers also tend to remain fairly constant, including the velocity of action potential conduction along the fiber and the rate at which tension can be developed.

However, this simple constancy poses a threat of instability. If muscle fibers are more than a few centimeters long, the time taken for action potentials to spread from the neuromuscular synapse (usually near the midpoint of the fiber) to the ends of the fiber tends to be greater than the delay in the onset of contractile force in the cross-bridges. Therefore, the middle of the fiber would be able to contract at the expense of lengthening the still-passive ends. Once lengthened past their optimal length, these distal sarcomeres could not generate enough force to pull themselves back into homogeneity with the rest of the muscle when the action potential finally reached the ends.

This problem has been overcome by a complex (and generally overlooked) architectural specialization. In the long muscles of larger animals, muscle fascicles are composed of several short muscle fibers in series (Loeb et al. 1987). The motor axons serving such fascicles conduct action potentials much faster than the muscle fibers. Branches of these motor axons innervate short muscle fibers distributed over the length of the fascicle, which can thereby be activated almost synchronously.

Differential scaling problems such as this must abound in the neural control of large limbs. Conduction delay in peripheral nerves depends on axon length divided by its conduction velocity. Because axon diameters are relatively similar among animals, this delay tends to rise proportionately with the length of the limb. Fortunately, larger animals may need to update their motor programs much less frequently than small animals because of the stabilizing effect of their relatively much larger rotational inertias, which rise as the fourth

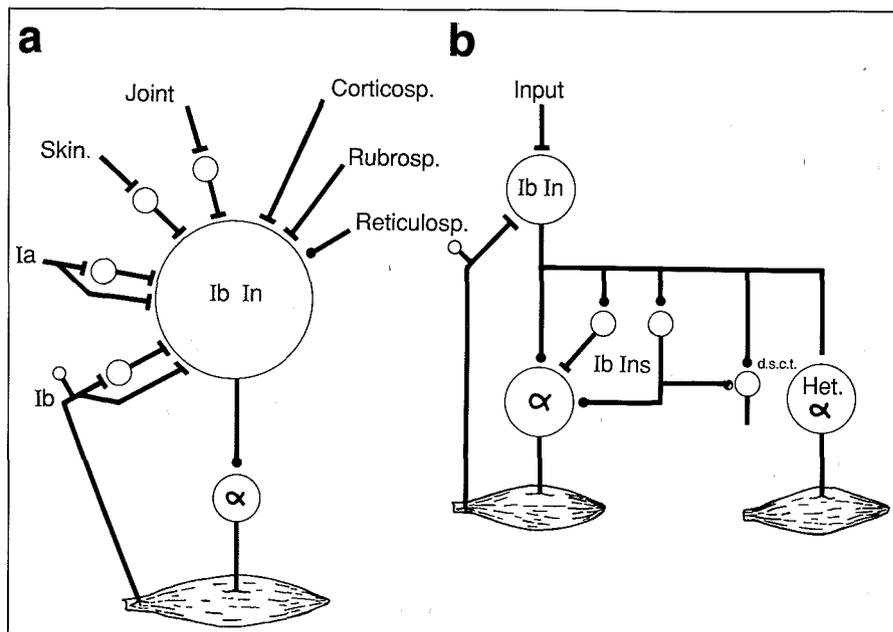


Figure 3. a. Negative feedback from force-sensing afferents in the Golgi tendon organs (Ib) arises through the action of inhibitory interneurons (Ib In), which project to the motoneurons (α) that control the muscle fibers that affect the tendon organs. However, these interneurons also receive direct and indirect input from a variety of other sensory modalities including muscle spindles (Ia) and from higher control levels (corticospinal, rubrospinal, and reticulospinal pathways). b. In addition to inhibiting the motoneurons that control their parent muscles, the Ib interneurons inhibit motoneurons controlling many other muscles (Het. α), and they send sensory information to higher control levels (e.g., via the dorsal spinocerebellar tract, d.s.c.t.). (Modified from Loeb 1987.)

power of linear dimension.

The synaptic propagation delays through the interneurons are probably quite similar in large and small animals. Therefore, interneuronal computation tends to represent a much lower percentage of total feedback delay in large animals. Alternatively, large animals might use more complex chains of interneurons to achieve responses that are more carefully computed rather than hastily composed. This strategy would seem practical given the much more drastic consequences of falls in large versus small animals.

Evolution of neuromuscular control

The solutions to scaling problems entail parallel changes in both musculoskeletal (mesenchymal) and neuronal (ectodermal) subsystems. Yet in all likelihood, most of the individual, random mutations involved in each evolutionary step have affected only one of the two subsystems. To survive, the modified organism must immediately have had a reasonably sat-

isfactory performance according to a mix of criteria, including energetic economy, peak strength, stability, and safety. Only then could it wait for further evolutionary refinement.

This problem suggests a general constraint on viable motor-control strategies for living organisms. A successful controller for a robot needs to deal only with a fixed mechanical apparatus; a neural controller must be capable of adapting gracefully to mechanical changes during the evolution of the species, plus the growth and development of individuals. These considerations may prove to be more instructive in developing and testing hypotheses for biological motor control than the more direct approach of circuit tracing in mature specimens, in which the details may obscure such general principles.

Recently, we have begun to apply engineering tools to design distributed controllers that improve performance of a given sensorimotor system (described mathematically as a computer model) for particular performance criteria (Loeb et al. in press). The underlying hypothesis is that dur-

ing locomotion, the spinal cord interneurons route proprioceptive feedback to obtain the desired stability of the limb as a whole, considering both the intrinsic mechanical properties of the musculoskeletal system and the range of perturbations that are expected on the basis of prior experience. Given the above-noted mechanical differences between species and during development, this approach should be able to generate testable predictions of differences in spinal cord circuitry. However, assembling and validating the necessary model of the musculoskeletal system for just the adult cat has been a major task, still in progress after seven years.

Conclusions

Both the musculoskeletal mechanics and the neural control circuitry are sufficiently complex that intuitive guesses about how locomotion works are unlikely to be useful. Quantitative analytical tools are needed just to compile all of the currently available information about each subsystem and to direct and interpret future experimentation. Beyond simply coping with this mountain of heterogeneous data, such tools offer the hope of developing formal links between the subsystems.

To accomplish this, the traditionally separate experimental disciplines of biomechanics and neurophysiology need to develop much closer ties so that motor control problems can be attacked simultaneously from both perspectives (Loeb 1987). Theoreticians in both areas must address not only the problem of motor control in a given organism, but also how their proposed solutions would fare in a world governed by ontogeny and phylogeny.

Locomotion is a particularly appropriate motor behavior for such a

multidisciplinary approach, because it is basic to the lives of almost all animals, assuring a highly constrained and orderly evolution.

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