

Electromechanical Analogs of Human Reflexes^a

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Are spinal-level reflexes important to skeletal movement? Surprisingly this question has not been answered satisfactorily since the concept of reflex action was introduced by Sherrington almost a century ago. Although much is known about individual reflex arcs in man, including minute details about the sensory and motor structures and the neuroanatomic and electrophysiologic organization of control networks, very little is understood about system integration and motor-related computation. In a recent article in *Trends in Neural Science* entitled, "Hard Lessons in Motor Control from the Mammalian Spinal Cord," motor physiologist Gerald Loeb urged researchers to adopt top-down strategies for understanding motor control, because reductionist approaches had failed to explain the basic organizational principles of movement.¹

An example of the confusion that persists in the study of motor control is the lack of understanding of the simplest reflex in man, the stretch (or myotatic) reflex. Some motor physiologists argue that stretch reflex response is too weak to have a significant impact on movement,² whereas others suggest that strong reflex response elicited by tendon tapping or other means demonstrates that these reflexes cannot be dismissed as trivial or unimportant.³

Researchers have suggested that the multilevel hierarchy of reflexes, from the stretch reflex on up, is a distributed control network that serves to facilitate motion as well as to speed and optimize responses to perturbations such as changing loads.⁴ The notion that simple neural networks in the spinal cord contribute significantly to motor action and sensing is intriguing to many investigators, particularly those in-

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dividuals who are using biology to guide development of computers, graphic processors, robots, and the like.

At present it is not practical to apply biologic solutions to the control of engineering systems because the fundamentals of biologic control are not well enough understood. Despite this, interesting biologically inspired approaches to system organization and control have been proposed and implemented. In the work of James Albus of the National Bureau of Standards, for example, a model of hierarchical goal-directed behavior is used to define control structures.⁵ The biologic accuracy of Albus's models is open to debate; however, there is no doubt as to the success of his approaches in dealing with complex control problems in a variety of areas including robotics.

To gain deeper insight into the function of reflexes relating to control and sensation of movement, we are building electromechanical systems that mimic, to some low level of approximation, the skeletal, muscular, and neurologic systems in man. By implementing and evaluating mechanical and electrical analogs of human reflexive systems, we can learn much about motor control organization and computation.

Thus far, models of two low level reflex arcs, the stretch (myotatic) reflex and the tendon (inverse myotatic) reflex, have been implemented in the control of two artificial muscles arranged as an agonist/antagonist pair about a hinge joint. The electromechanical model suggests the function of these two reflex arcs by demonstrating that: (1) the stretch reflex alone can make artificial muscles springlike; (2) the tendon reflex alone can give artificial muscles a baseline tone; and (3) stretch and tendon reflexes working in tandem allow for the integration and control of spring and tonal properties.

The stretch reflex is believed to be the simplest and quickest of all reflexes in the motor control hierarchy, involving only one excitatory synapse between two neurons (a dorsal root ganglion cell and an anterior horn cell—the alpha motoneuron) (FIG. 1). The action of the reflex is a neurologically mediated graded opposition to muscle stretch. The stretch reflex sensor is the muscle spindle organ that is sensitive primarily to muscle length and velocity, and it is in parallel with the muscle. The greater and quicker the stretch of this encapsulated structure from its equilibrium position, the greater its firing rate of action potentials. Because the synapse with the alpha motoneuron is excitatory, the increased firing rate of the spindle organ results in increased muscle contraction.^{6,7} The net action of this reflex is thus analogous to the action of a viscously damped spring, which is characterized by a resistance to external stretches that scales in linear proportion to both the distance and the speed of displacement from an equilibrium length.

Properties of the muscle spindle organ, including sensitivity and length set-point are influenced by a separate system known as either the fusimotor or the gamma motor system. (This system is distinct from the alpha motor system, described in the paragraph above, which is responsible for direct activation of the force-generating extrafusal fibers of the muscle.) Action potentials from gamma motoneurons activate muscle fibers that are internal to the encapsulated muscle spindle organ. These internal or so-called intrafusal fibers act to modify properties of the spindle sensor such as the sensitivity to stretch. The gamma system is complex: there are static and dynamic types of intrafusal innervation that have different influences on the muscle spindle organ sensors. The static system seems to be involved with the setting of the muscle spindle length set-point, whereas the dynamic system seems to affect the muscle spindle organ sensitivity to movement.⁸

In addition to the stretch reflex, muscles are influenced by the tendon reflex (FIG. 2). The tendon reflex arc begins with a tension detector, the Golgi tendon organ, that lies at the boundary between muscles and fascia, such as tendons, and therefore is in series with the muscle. Action potentials from this sensor excite an interneuron, which

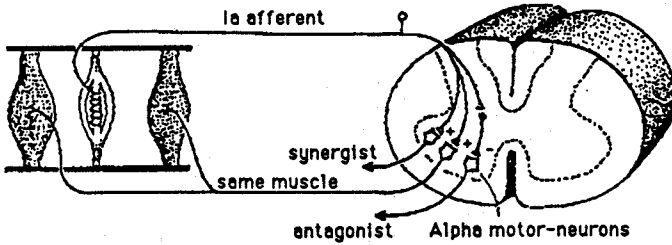


FIGURE 1. After Ghez. Schematic of human stretch (myotatic) reflex arc.

subsequently inhibits the alpha motoneuron. This reflex functions as a tension servo that acts to maintain constant muscle tone.⁷

There are many other aspects of the motor control system that may or may not be important to modeling human motor control. Features that may be significant include: organization of muscle fibers into varying sized groups known as motor units; ordered recruitment of increasingly sized motor units as force is developed (the Henneman size principle); organization of multiple muscles into task groups; variation in temporal response and fatiguability of muscle fibers; nonlinear viscoelastic properties of muscle fiber; the varying prevalence and location of sensors within muscle tissue as well as the existence of other sensors and their influence on reflexes; varying conduction times of action potentials along nerve fibers; the variety of types and speeds of synaptic inhibition and facilitation; and biochemical aspects of control including neurotransmitters, ATP generation, and lactic acid absorption.⁸ Which of these, or perhaps other, factors are important to modeling human motor control is yet to be determined.

Even though individual reflex arcs are reasonably well understood, the combined actions of multiple reflex arcs are not. In the combined action of stretch and tendon reflexes, Houk⁹ suggested that jointly these reflexes might serve to maintain constant muscle stiffness.⁹

MODEL STRETCH REFLEX

The physical model of the stretch reflex consists of an artificial muscle, an artificial muscle spindle organ, and an artificial spinal-level neural network.

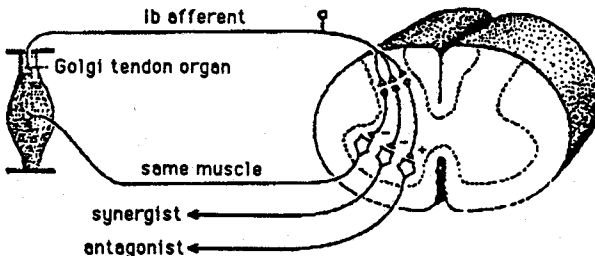


FIGURE 2. After Ghez. Schematic of human tendon (inverse myotatic) reflex arc.

The model muscle shown in FIGURE 3 is a plastic chain that is threaded through several free-wheeling pinion gears, much like sprocketed film is threaded through a movie projector. By rotation of the platform that supports the pinion gears, the path length through the assembly is varied, thereby allowing the straight-line distance between the ends of the chain to be altered.

The pinion gear "contractile" assembly is driven by a DC torque-motor. By choice, this motor is excited only by the polarity of current that leads to contraction of the plastic chain; thus, like real muscle, there is no capacity for active extension. (There

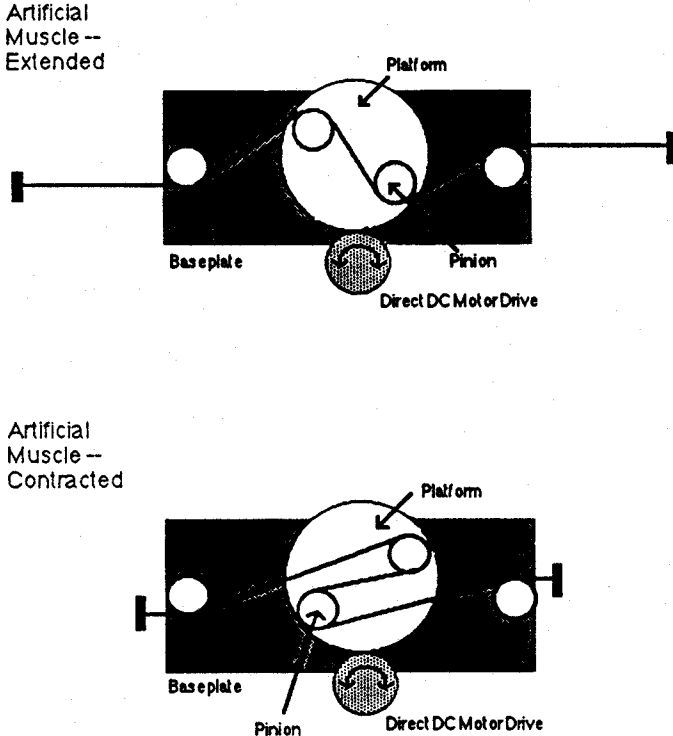


FIGURE 3. Artificial muscle.

is, however, the capacity for active control during lengthening, which is a property of biologic systems.) Also similar to natural muscle, the model muscle offers little resistance to externally imposed forces acting to extend it, provided that there is no active contractile drive. The low resistance is due to minimal gear reduction and the use of low friction components throughout. (All mechanical components and the DC motor are from LEGO Systems, Inc., Enfield, CT 06082.)

The model of the muscle spindle organ is a multi-turn potentiometer that measures the effective length of the plastic muscle. The linear motion of the muscle is converted to rotation by the use of a nonslip sprocket pulley. The geometry is carefully chosen to ensure that the potentiometer is in parallel with the contractile assembly, as with real muscle spindle organs with respect to real muscles.

The model neurons that are ultimately wired together into neural networks are

multiple-input, unipolar, clamped, linear amplifiers. The single polarity of the output assures that any individual neuron is either excitatory or inhibitory, but not both. The clamped aspect of the amplifier means that the output never exceeds a set amount determined by the selection of amplifier power supply voltage. (Different model neurons may have different saturation values by the use of different power supply voltages.) The gains of the linear amplifiers are set to realistic values as derived from electrophysiologic studies. No attempt has been made to model the pulsed aspect of action potentials; rather, signals between elements are represented as analog voltages.

The model system corresponding to the stretch reflex is shown in FIGURE 4. Because the DC torque-motor produces a force proportional to current and not voltage as with real muscle, an additional circuit has been added to achieve the proper voltage-force proportionality. In addition, a voltage follower circuit has been incorporated into the muscle spindle potentiometer to minimize distortion of its linearity by circuit loading.

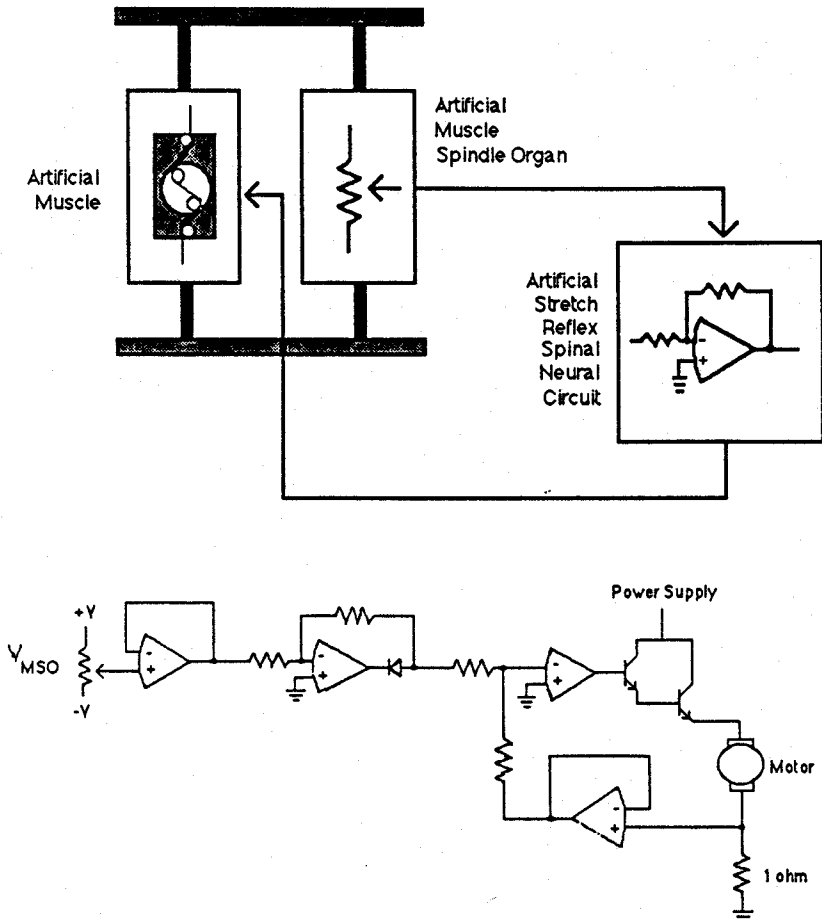


FIGURE 4. Schematic of artificial stretch reflex arc.

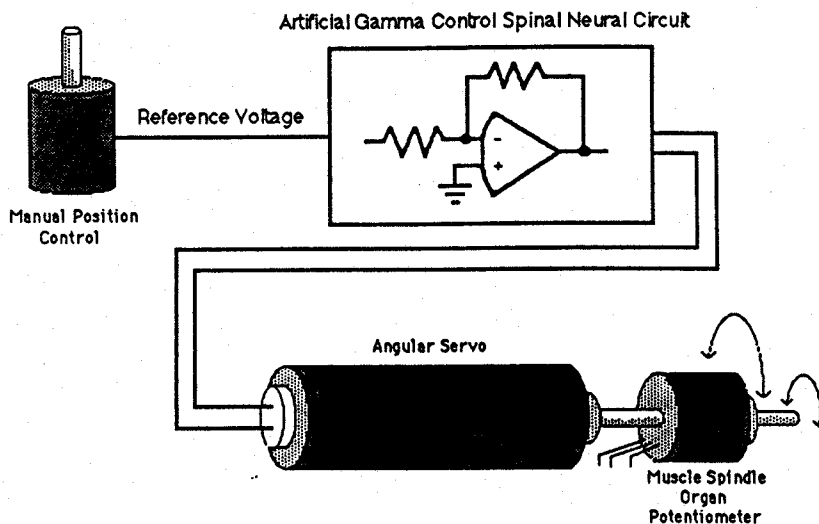


FIGURE 5. Schematic of artificial gamma static control.

As expected, once all of the elements of the synthetic reflex arc are interconnected, the plastic chain becomes springlike for stretches extending its length beyond a length set-point. The length set-point is determined by the location of the zero voltage setting on the muscle spindle potentiometer.

To improve the model stretch reflex, we have also modeled the static portion of the gamma motor system. This addition allows for adjustment of the length set-point by providing a motorized positioner that rotates the muscle spindle potentiometer body. The equivalent to gamma static motorneuron drive is thus the reference voltage to the positioner servo, as shown in FIGURE 5.

MODEL TENDON REFLEX

The physical model of the tendon reflex is similar to that of the stretch reflex and consists of synthetic muscle, synthetic spinal-level neural network, and a synthetic Golgi tendon organ.

The artificial muscle used in the tendon reflex model is somewhat similar to that described previously, with the significant difference that the DC torque-motor drives the contractile assembly through a gear train with a mechanical advantage of 900:1. This gear reduction allows the motor to develop much greater forces than before, but at the expense of speed and compliance. Compliance or "give" in response to external loading for this model is provided by the give in the artificial Golgi tendon organ. The motor drive is bipolar, so that unlike real muscle, active lengthening is possible.

The model Golgi tendon organ is a spring-loaded potentiometer, as shown in FIGURE 6. The equilibrium position of the spring (i.e., position corresponding to zero restoring torque) is set to correspond to the zero voltage position of the potentiometer

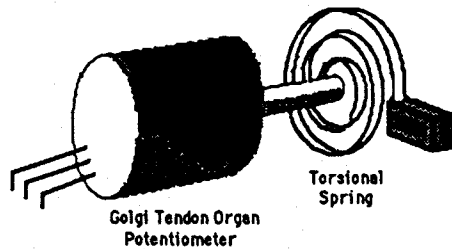


FIGURE 6. Artificial Golgi tendon organ.

wiper. Because the spring tension is equivalent to the tension (i.e., tone) of the artificial muscle, the combination of mechanical spring and potentiometer attached to the muscle chain by a nonslip sprocket provides a voltage output that is proportional to chain tension. Unfortunately, however, the chain unwinds from the sprocket so that the effective muscle length varies with tension. To minimize this problem the sprocket chosen is as small as possible. In future models, load cells will be used as artificial Golgi tendon organs because they are capable of measuring tension without altering chain length.

The model neural network for the tendon reflex is shown in FIGURE 7. The network here includes two model neurons, namely, an alpha motorneuron and an inhibitory interneuron. For this circuit to be active a constant input current must be fed to the alpha motorneuron. The amount of current sets the tension in the muscle and thus is labeled as the tension reference in FIGURE 7. In a real organism such a current might come from a descending pathway or from other local circuits, if indeed it has an analog.

The synthetic tendon reflex arc functions as a tension servo. Here the static tension in the chain is disturbed by stretching or shortening the muscle, and the circuit acts to restore tension to a value close to the desired (i.e., reference) one, differing only by a small amount identified as the servo error.

COMBINED STRETCH AND TENDON REFLEXES

The stretch reflex makes the electromechanical muscle springlike, and the tendon reflex provides it with tone. What happens when both reflexes are active?

In the biologic case Houk suggested that the purpose of the combined action of the stretch and tendon reflexes is to maintain a constant muscle stiffness.⁹ Although there is some evidence of this effect, the suggestion is controversial owing to the many possible factors that can contribute to stiffness of biologic muscle. In the artificial case, in contrast, all operative parameters are known and the actions of the stretch and tendon reflexes are unambiguous.

When the synthetic spindle organs are unloaded, the tendon reflex circuit acts to hold a constant tone irrespective of muscle length and does so with a small servo error that is proportional to the tension set-point value.

If the synthetic spindle organ is active (i.e., stretched beyond its length set-point), then the combined stretch and tendon circuits act to hold the model muscle at both the tension set-point and the length set-point. Because these circuits allow for a finite

servo error, external stretching of the muscle beyond the length set-point gives rise to an error signal that grows linearly with the length of the muscle. This error signal is responsible for making the model muscle springlike, and it leads to a spring constant that depends on the gains of the alpha motorneuron, the inhibitory interneuron, and the contractile assembly as well as the sensitivities of the muscle spindle organ and the Golgi tendon organ.

The effective spring constant for the combined stretch and tendon reflex case is lower than that if only the stretch reflex were active, because the tendon reflex is inhibitory to the alpha motorneuron. From a simple analysis, given in the appendix, this opposing strategy does not maintain a constant stiffness. For example, if the contractile assembly gain were reduced (simulating fatigue), then the net muscle stiffness would be reduced. What the two reflexes do accomplish, however, is the maintenance of both the tone and springlike properties of the muscle. They also solve

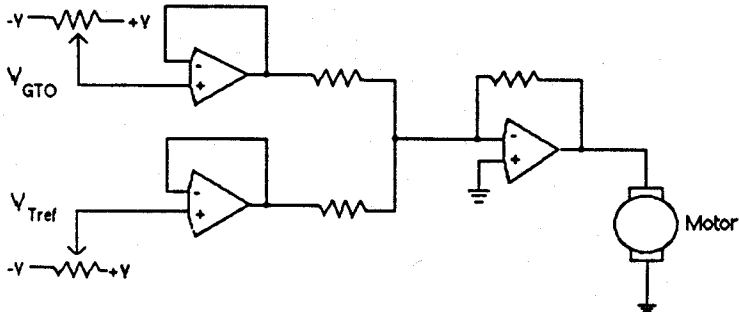
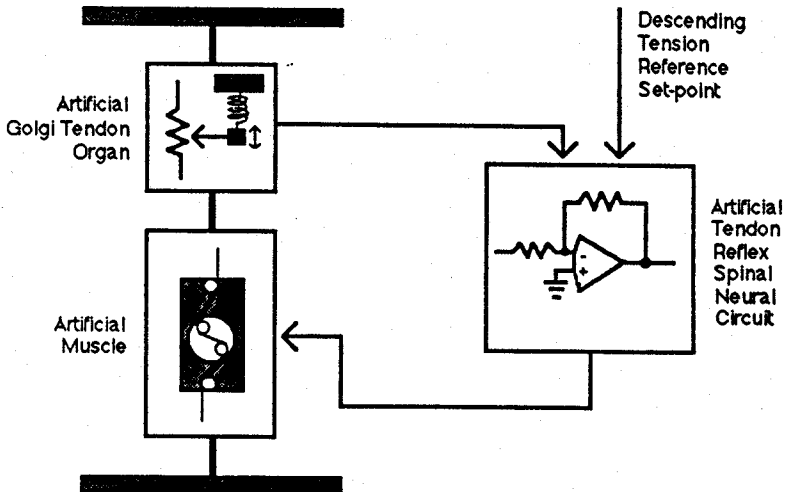


FIGURE 7. Schematic of artificial tendon reflex arc.

the nontrivial problem of integrating both length and tension control into a single embodiment.

AGONIST/ANTAGONIST MUSCLE PAIR

In addition to building a model of a single reflexive muscle, we have also combined two muscles as an agonist/antagonist controller of a simple hinge joint. As might be expected, if both muscles are equipped with only stretch reflexes with the length set-points aligned at some joint posture, then the joint will display bidirectional torsional springlike behavior. Likewise, if both muscles are equipped with only tendon reflexes, then the joint displays a resistance to externally imposed motion by an amount that depends on the values of the tension set-points of the muscles and is independent of the joint angle.

An interesting property of the agonist/antagonist pair when only stretch reflexes are present is that when the joint is externally rotated to a position away from its rest posture so that the restoring torque is generated only by the stretched muscle, the opposing muscle slackens and droops. If we add a tendon reflex to the control strategy of the agonist/antagonist pair, this undesirable aspect (muscle slackening) is eliminated.

SUMMARY

The conclusion to be drawn from our modeling is that the combined stretch and tendon reflexes alone can endow artificial muscle with a springlike feel as well as give it a baseline tone. In response to questions that motor physiologists often ask as to what variables the system controls,¹⁰ the answer here is clear: the stretch and tendon reflexes act together to maintain both a tension set-point and a length set-point, but in so doing they also give the system a springlike feel because of the existence of a servo error.

The main goal of our studies is to understand the integration of reflexes, and thus far we have only begun to explore the two lowest-level spinal reflexes. We are in the process of expanding this work by developing a much more refined arm explicitly modeled after the human arm. This new arm is to be activated by a minimum of 10 muscles, each of which is reflexively driven, and it will allow us to explore the integration of higher-level reflex action such as automatic inhibition of antagonists and facilitation of synergists.

REFERENCES

1. LOEB, G. E. 1987. Hard lessons in motor control from the mammalian spinal cord. *Trends Neur. Sci.* 10: 108.
2. RACK, P. M. H. 1981. Critique *in* *Muscle Receptors and Movements*. Macmillan, London.
3. STEPHENS, J. A. 1981. Critique *in* *Muscle Receptors and Movements*. Macmillan, London.

4. EASTON, T. A. 1972. On the normal use of reflexes. *Am. Sci.* 60: 591.
- 5a. ALBUS, J. S. 1979. Mechanisms of planning and problem solving in the brain. *Math. Biosci.* 45: 247.
- 5b. ALBUS, J. S. 1981. *Brain, Behaviour and Robotics*. Byte Books, Peterborough, N.H.
6. GHEZ, C., in KANDEL, E. R. & J. H. SCHWARTZ. 1985. *Principles of Neural Science*. Elsevier/North Holland, New York.
7. GUYTON, A. C. 1989. *Textbook on Medical Physiology*. W. B. Saunders, Philadelphia.
8. BROOKS, V. B. 1986. *The Neural Basis of Motor Control*. Oxford University Press, New York.
9. HOUK, J. C. 1979. Regulation of stiffness by skeletomotor reflexes. *Ann. Rev. Physiol.* 41: 91.
10. STEIN, R. B. 1982. What muscle variable(s) does the nervous system control in limb movements. *Behav. Brain Sci.* 5: 535.

APPENDIX

In the following discussion, all constants, K , are assumed to be positive definite quantities. Refer to FIGURE 8 for a block diagram of the control system.

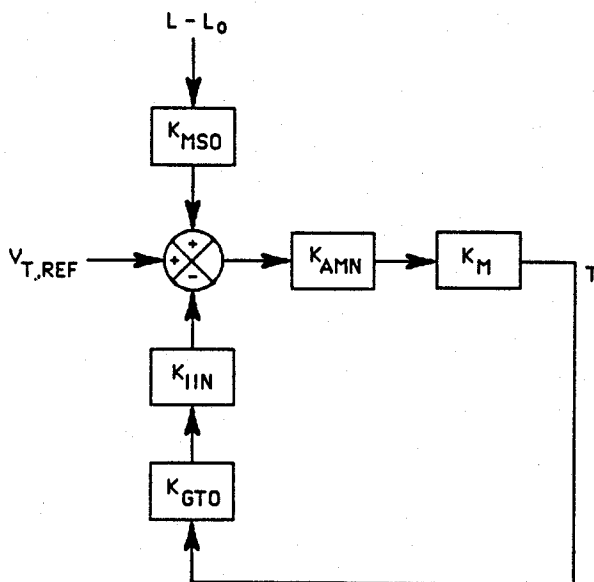


FIGURE 8. Control model of combined stretch and tendon reflex arcs. (MSO = muscle spindle organ; AMN = alpha motoneuron; M = muscle; GTO = Golgi tendon organ; IIN = inhibitory interneuron; T = tension; and L = length).

The governing relationship for the artificial muscle spindle organ (MSO) is:

$$\begin{aligned}
 V_{\text{MSO}} &= K_{\text{MSO}} (L - L_0), \text{ for } L > L_0 \\
 &= 0, \text{ for } L < L_0.
 \end{aligned}$$

where L is the muscle length and L_0 is the length set-point. L_0 is determined by the mechanical linkage and the location of the zero voltage position of the MSO potentiometer.

The governing equation for the artificial Golgi tendon organ (GTO) is:

$$V_{GTO} = K_{GTO} T,$$

where T is the muscle tension. The artificial inhibitory interneuron amplifies this signal with a gain of $-K_{IIN}$. A descending reference signal sets the desired tension to value, $T_{REF} = K_{IIN} K_{GTO} V_{T,REF}$. The gain of the artificial alpha motoneuron is K_{AMN} .

The equation describing the artificial muscle is:

$$\begin{aligned} T &= K_M V, \text{ for } V > 0 \\ &= 0, \text{ for } V < 0, \end{aligned}$$

where a positive voltage causes contraction of the muscle.

Combining all the elements gives a length-tension relationship:

$$\begin{aligned} T &= A(K_{MSO}(L-L_0) + K_{IIN} K_{GTO} T_{REF}), \text{ for } L > L_0 \\ &= A(K_{IIN} K_{GTO} T_{REF}), \text{ for } L < L_0, \end{aligned}$$

where

$$A = (K_M K_{AMN}) / (1 + K_M K_{AMN} K_{IIN} K_{GTO}).$$