

From Equilibrium Point to Optimal Control

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In the mid sixties, Feldman (1966) reported on experiments on feedback control processes that stabilize the human arm. In a typical test, the elbow flexors (or extensors) would be loaded and the subject was asked to maintain a predetermined elbow angle. After a sudden decrease of the load, the forearm would settle at a new position. The experiments were repeated for the same initial elbow position and load, but each time the change in the load was different. The set of points that resulted consisted of two variables: elbow angle θ and force f . These points formed an exponential-like curve

$$f = a \left(\exp \left[b(\theta - \theta_\lambda) - 1 \right] \right) \quad (1)$$

In the above expression, θ_λ is a threshold length beyond which the muscle will produce force. Feldman's thesis was that the signals sent from the brain to the various motoneurons did not produce force, but rather produced a recruitment threshold θ_λ by depolarizing α -motoneurons. This idea was a refinement of an earlier hypothesis put forth by Merton (1972) in which he had suggested that the descending system initially activated γ -motoneurons to drive the movement. However, as Feldman (1986) noted: "Both models nevertheless proceed[ed] from the basic idea that the nervous system uses the stretch reflex to control movements." That is, the descending commands could not control force directly, but only control the set point of a local feedback circuit.

To test this idea, Feldman and Orlovsky (Feldman and Orlovsky, 1972) stimulated a motor center in the brainstem of cats, attempting to artificially produce activity that resembled a voluntary movement. They found that their stimulation did not produce force changes, but rather changes in the force-length relationship of the muscle.

The model in Eq. (1) sits in sharp contrast to muscles models commonly used for description of movement (Stern, Jr., 1974; Hof and Van den, 1981; Zheng et al., 1984; Hogan, 1984; Winters and Stark, 1985). All of these "other" models belong to a single class in which the nervous system specifies an input that generates force in the muscle by engaging a non-linear elastic element with an adjustable stiffness. How do these muscle models relate to the work of Feldman?

Using data from Hoffer and Andreassen (1981) in which muscles were analyzed in both isolated and reflexive conditions (i.e., connected via Ia afferents to the spinal cord), Shadmehr and Arbib (1992) argued that whereas an isolated muscle indeed

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behaved as a non-linear spring with an adjustable stiffness, under the control of spinal reflex circuitry it acted as a non-linear spring with an adjustable threshold length. We can summarize the argument as follows: Hoffer and Andreassen (Hoffer and Andreassen, 1981) had measured the rate of change in stiffness of muscles with respect to force. They had found that the relationship between force and stiffness in the intact muscle-reflex system was independent of muscle length

$$\frac{df}{dx} = k(1 - \exp[-\alpha f]) \quad (2)$$

We note that the solution to the above differential equation is of the form

$$f = \frac{1}{\alpha} \ln(\exp[\alpha k(x - \lambda)] + 1) \quad (3)$$

Therefore, the intact muscle with its reflex system has a static behavior that resembles a non-linear spring with an adjustable threshold.

Feldman's idea was that brain could not control muscles independent of this reflex system, that is, the brain could not specify a set EMG pattern because that pattern was inherently dependent on the state of the sensory system that measured muscle length. This reflected a fundamental departure from the way robots are typically controlled, and a departure from models that used robotic inspirations to describe control of biological movements. To illustrate this difference, let us consider a simple example. For an inertial system like the human arm, the dynamics can be written as

$$\ddot{\theta} = I^{-1}(f_c(\theta, \dot{\theta}, u) - f_m(\theta, \dot{\theta})) \quad (4)$$

In Eq. (4), I is the position dependent inertia matrix of the system, f_c is the active forces imposed on the system due to the motor commands u to the muscles, and f_m is the passive forces produced by the motion of the inertial coordinate frame (Coriolis and centripetal). To move such a system along a desired trajectory specified by $\theta_d(t)$, a typical approach would be to use an "inverse model" (called a computed torque controller in the robotics literature) to cancel the passive dynamics

$$f_c = \hat{f}_m + \hat{I}\ddot{\theta}_d \quad (5)$$

In Eq. (5), \hat{f}_m is an internal model of f_m . Note that when we put Eq. (5) in Eq. (4), we get the desired acceleration (if our internal model is accurate). To ensure stability, we add a simple feedback control law

$$f_c = \hat{f}_m + \hat{I}\ddot{\theta}_d - B(\theta - \theta_d) - K(\dot{\theta} - \dot{\theta}_d) \quad (6)$$

In Eq. (6), the terms $\hat{f}_m + \hat{I}\ddot{\theta}_d$ imply that the controller can specify force independent of state of the system (the last two terms). Such models have been extensively used in describing biological motor control in the last two decades (Shadmehr et al., 1995; Berniker and Kording, 2008).

Feldman's argument was that these models are flawed because they assume that the brain has the capability to specify muscle force independent of sensory feedback. Instead, the following scenario was described. In Eq. (3), muscle force is a result of a feedback control system that could be controlled through *state* variables (e.g., muscle length χ), and a *gain* function that depends on parameter λ . When this parameter is set for the various antagonist muscles of a limb, the result is an *equilibrium point*, that is, a position for which the various antagonist forces cancel. Movement results from a shift of this equilibrium position (Flash 1987).

A major assumption of the equilibrium point hypothesis was that state variables are described through spindle sensory afferents. That is, the EMG that one measured during voluntary movements came about not because of descending commands that could directly specify them, but because of interaction with sensory feedback from the muscles. Therefore, someone with sensory neuropathy affecting the muscle spindles should not be able to make normal movements. For example, the 3-burst EMG that one observes during rapid elbow movements should not be present without sensory feedback. In a crucial set of experiments, these predictions were shown to be false (Rothwell et al., 1982; Berardelli et al., 1996).

However, the inverse model approach (Eq. 6) was equally flawed because it imagined descending commands to be driven by a desired trajectory that activated the muscles and produced force independent of their state. This kind of approach would make sense if our muscles were position independent direct-drive motors that one finds in robots. It made little sense in terms of actuators and the associated reflex system that we are born with.

In summary, by relying on physiological data suggesting that an intact muscle with its spinal reflex system behaved as a non-linear spring with an adjustable threshold length, equilibrium point hypothesis (EPH) built a model of motor control in which descending commands modulated spinal reflexes. A failing of this model was that it could not explain the ability of people with proprioceptive loss to make voluntary movements. Computational approaches that relied on inverse models could explain these data, but they were equally flawed because they ignored the state-dependent force characteristics of muscles.

An alternative idea was set forth in the framework of forward models. In this framework, the state upon which the nervous system acted was not simply due to the sensory system's measurements, but also due to internal predictions made by the brain. For example, Ghez and Sainburg (1995) noted that the first time a deafferented patient was asked to make a rapid arm movement, the hand trajectory had significant errors. However, upon repeating the movements a few times with visual feedback, it became comparable to healthy controls and could be generalized to nearby movement directions. They argued that the reason why deafferented patients could make normal movements was because motor commands that acted on the muscles could be driven by internal predictions of state. These internal predictions, driven by forward models, improved with practice and visual feedback, forming a more accurate estimate of state. Muscles were state dependent force generators, but these states could be predicted even when proprioception was missing.

To expand this idea, consider that in the equilibrium point formulation of motor control, there are two key quantities: state, and gain. Motor commands arise from a feedback control law that applies a gain to the state, in which state is measured

by proprioceptive sensors. The forward model framework replaces the notion of sensory state with the *estimate* of that state, i.e., an estimate in which sensory information is combined with prior predictions (Kording and Wolpert, 2004). The prior predictions are the expected states that are produced by forward models. When we make a movement, the state of our arm can be measured by proprioceptive and visual sensors. If a person has no proprioception, movements can still be made because forward models can continue to predict visual and proprioceptive state of the limb, and produce motor commands based on the integration of these predictions with whatever sensory feedback is available.

The next major step forward came when Todorov and Jordan (2002) used optimal feedback control (OFC) theory to link the concept of a gain with a feedback control law that depended on the properties of the task. In OFC, the gain of the feedback loop depends on a cost function that specifies why the task is being performed (i.e., what were the relevant states that produce reward), and what are the relevant costs (for a review, see Shadmehr and Krakauer, 2008). Therefore, in both equilibrium control and in OFC, one does not compute forces or motor commands. Rather, one computes the feedback gain. As a result, in both theories movements and reactions to perturbations arise from interactions of state with this feedback gain. The crucial differences, however, are that in OFC, the gain is task dependent, and the state depends on a forward model, i.e., the state is estimated through a combination of measurements and predictions.

The idea that the nervous system controls states that are relevant to the task is also called an *uncontrolled manifold* (Scholz and Shoner, 1999). The term “uncontrolled” refers to the gain function’s reduced response to state variables that have little or no relevance to the goal of the task. The idea has been used to account for complex movements that offer clear redundancies, like bimanual pointing (Domkin et al., 2005) and Frisbee throwing (Yang and Scholz, 2005). In OFC, the gain of states that are irrelevant to the goal is lower than states that are relevant. OFC states in mathematics the idea of an uncontrolled manifold, and has no fundamental differences with it in this respect.

In the current formulation of OFC, there is no specific constraint due to spinal reflexes. That is, the “low-level” control system of the spinal reflexes play no special role in the control system. As a result, when task properties change, the theory predicts that the brain should be able to alter the feedback response to optimize the task dependent cost function. In fact, there are numerous examples that this is not the case. For example, during maintenance of posture, the voluntary control of arm stiffness is possible but extremely limited (Selen et al., 2009). During voluntary movements, changes in response to perturbations are possible, but often much less than expected (Burdet et al., 2001; Diedrichsen, 2007). That is, when one closely examines the data, voluntary control of our body often appears less than globally optimal. The reason may be that low-level (spinal) control structures impose constraints on how the high-level processes can control behavior. As a result, a significant challenge will be to incorporate into OFC a more accurate description of low-level physiological constraints.

In summary, feedback control is at the heart of both equilibrium point and optimal feedback control theory. In any feedback control system, there are two crucial components, state variables and gain functions. In OFC, the concept of state is not limited to the proprioceptive information signaled by primary afferents,

but rather an estimate of the limb's state as produced by combining predictions of a forward model with sensory feedback. In OFC, the concept of feedback gain is task dependent and depends on a cost function that describes the rewarding states and nature of motor costs. Whereas in equilibrium point control the feedback controller was imagined at a spinal level acting on proprioceptive feedback, more recent developments of OFC envision a hierarchy of feedback controllers that act on estimates of sensory states at all levels of the central nervous system. Perhaps the greatest legacy of equilibrium point control will be its predictions regarding the apparent limitations in the central nervous system's ability to modulate feedback, that is, the less than optimal change in the feedback gains when task properties change. These limitations may be related to the relative inflexibility of low level (spinal) vs. high level (supra-spinal) feedback controllers.

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