Stable Hopping of a Muscle-Actuated Leg System
Using Positive Force Feedback

by

Chanikarn Wongviriyawong

Submitted to the Department of Mechanical Engineering
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Abstract

In control of movement, two key components, which are pure mechanical response of the system and response due to sensory feedback, must be thoroughly understood. Recent studies suggest not only the existence of positive force feedback in vivo, but also the emergent property of positive force feedback in having a stabilizing effect on a dynamical system in the presence of disturbances. In this thesis, simulated environment of simple one-dimensional point mass hopping model with positive force feedback as well as experimental results of the same dynamical system are compared and studied in detail. Three important hypotheses are investigated. The first hypothesis involves positive force feedback and its stabilization property despite disturbances in the system. A system with positive force feedback control attains cyclic motion while system energy is being added or removed without changing its steady state system energy. Secondly, overall mechanical behavior of the leg becomes elastic in the existence of positive force feedback. In locomotion, elastic leg behavior is desired for a pertinent adaptation to physical properties of the environment and utilization of the locomotory performances. The last hypothesis investigated is the effect of feedback control parameters on closed loop system behavior, i.e. frequency of hopping, steady state hopping height, etc. Simulation and pilot experimental data are compared both qualitatively and quantitatively concerning all three hypotheses.

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0.1 Acknowledgments

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Tetanic Fusion Frequency

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Chapter 1

Introduction

1.1 Motivation

In locomotion, the utilization of sensory information is seemingly inevitable. Classical findings of Sherrington [30] and Denny-Brown [8] showed that force from hind limbs extensors is an important control signal for transition between swing and stance phase. Many studies also suggested that the force signal actually reinforces muscle activity, implying the positive sign of feedback controller. There is a considerable number of studies suggesting the existence of positive force feedback control in humans [33], cats [3, 19, 25, 27] or even invertebrates [4, 7].

In a positive force feedback system, afferents input information about ground reaction force does not only reinforce extensor activity in a stable manner, but also allows controller robustness to disturbances in presence of unexpectedly long delay and high gain in normal walking cats [13, 21]. In locomotion of spinal and decelerated cats, reflexes mediated by golgi tendon organs are excitatory [15, 20, 26]. Moreover, experiments in human subjects performing load-bearing tasks showed stable load compensation under a wide range of gain [1].

A positive feedback system is counterintuitive in classical control theory. However, it was suggested that stability of closed loop system with positive feedback can be
achieved as a result of automatic gain reduction due to nonlinearity in muscle properties and the structure of the feedback loop [1]. The main focus of this thesis is to study the stability of a positive force feedback system and to verify certain hypotheses based on modeling and experimental results. The experiments are conducted in vitro on actual muscle tissue, namely Plantaris Longus, from leopard frogs (Rana Pipiens).

In the thesis three key hypotheses are evaluated. First, we hypothesize that a one-dimensional hopping leg model with a single knee extensor muscle will exhibit a spring-like leg response when a positive force feedback muscle activation is applied. This elastic leg behavior is highly desirable in locomotion [23]. Leg stiffness is one of the most important mechanical control parameters affecting stability in the control of legged movement. We also hypothesize that such a positive force feedback system will converge to a fixed steady state energy level independent of the initial mechanical energy state of the system. Thirdly, we hypothesize that leg stiffness can be modulated by varying positive force feedback loop gain.

1.2 Thesis Organization

The purpose of this study is to understand the closed loop system behavior of a one-dimensional hopping leg model with a single knee extensor muscle that is controlled using a positive force feedback reflex. The thesis is divided into two major sections. The first section focuses on a development of a numerical simulation of the legged system. Simulation involves interaction between neural control via positive force feedback and muscle mechanics in a simple one dimensional hopping model. The model used here contains a point mass with a two-segmented leg [16]. Muscle mechanics is modeled using a Hill type model. Positive force feedback is modeled as a gain feedback with constant offset representing activation level previously in the muscle. Comprehensive information on modeling muscle mechanics, neural control and system dynamics can be found in chapter 2. The second part of the thesis involves experimentation to verify or dispute hypotheses based on previous findings about positive force feedback on an actual extensor muscle. Detailed descriptions of simulation and
experimental protocols are found in chapter 3. Chapter 4 contains plots as a comparison between theoretical predictions and experimental results. Lastly, how and why experimental results support or refute the three hypotheses are discussed in chapter 5. Appendix A shows detailed derivations of system equations of motion. While, appendix B describes a model for excitation-contraction coupling. Appendix C contains two sets of system parameters used for computer simulation and experimental trials. Lastly, appendix D has additional plots that are excluded from chapter 4.
Chapter 2

Background

This chapter contains background knowledge on muscle physiology, relevant muscle models and dynamics of a one-dimensional hopping model. Moreover, the interaction between nervous system and the mechanics will be discussed. The first section of this chapter outlines muscle physiology. Secondly, section 2.2 explains the type of muscle model used in the simulation study. Section 2.3, explains how central nervous system uses reflex pathways to communicate with peripheral mechanics. It also talks about how reflex pathways is modeled mathematically in this study, leading to the concept of this project, namely positive force feedback control. The final section switches from background in muscle mechanics to details in the modeling of this particular mechanical system, or a hopping leg model.

2.1 Muscle Physiology

A muscle belly is composed of muscle fibers, nerves, blood vessels, connective tissues, etc. Skeletal muscle fibers contain filaments of myofibrils (myofilaments), which are the main component for force production, mitochondria, which provide energy, and sarcoplasmic reticulum which are used for the muscle activation process. Myofibrils consist of highly organized and repetitive subunits called sarcomeres. A sarcomere is the main functional unit for muscle contraction. Each sarcomere contains two main types of filaments, namely thick and thin filaments. Through cyclic movement of
these two filaments sliding across each other, muscle contraction is produced.

2.1.1 Sarcomere Shortening

A sarcomere contains contractile proteins, thick and thin filaments confined by Z disks to which thin filaments are attached. The overlap between thick and thin filaments gives skeletal muscle a striated appearance.

Thin filaments are composed of polymerized actin, troponin and tropomyosin. Troponin and tropomyosin prevent interaction between actin and myosin in the absence of calcium. The area containing only thin filaments is called the “I-band”. At the z-line, actin filament is fixed to each other at the middle of the I-band.

Thick filaments are composed of myosin molecules interlacing one another. Each myosin molecule’s double heads project outwards except in the “bare zone” in the middle of a sarcomere. Myosin filaments are joined at the “M-line”.

Another type of filament, which is thin and highly elastic, is called connectin. It connects both Z disks to the thick filament. Connectin is significant in contributing to passive stiffness in muscle fibers [10].

The sliding movement of thin filaments past thick filaments causes the myosin heads to interact with neighboring actin filaments, forming links between actin and myosin heads called “crossbridges” [17]. The amount of force production under isometric conditions and full activation depends on the number of available crossbridges and the average crossbridge force. By engaging and disengaging crossbridges, muscle contracts and relaxes. The cyclic attachment and detachment of myosin heads from actin filament result in mechanical work production in muscle.
Skeletal muscle contains bundles of fascicles, which are made up of muscle fiber bundles. Muscle fiber is composed of many myofibrils. Each myofibril has repeating longitudinal subunits called sarcomeres, the smallest subunits for muscle contraction. In each sarcomere, thick filaments, thin filaments and elastic filaments are found. Thin filaments are usually called actin filaments, whereas thick filaments are called myosin filaments. The cross sections at the bottom of this diagram show the extraordinary regular orientation of each filament with respect to one another.
2.2 Muscle Models

Modeling of muscle behavior has received much attention and is a well-studied field. Several approaches include Hill’s muscle model, Huxley’s Sliding Filament theory, Zahalak’s Bond Distribution Moment model, and many more. Due to computational intractability of Huxley’s and Zahalak’s models, Hill’s model is frequently used in biomechanical modeling.

Motivated by experimental evidence, A.V. Hill suggested that muscle could be modeled as an undamped spring in series with three parallel elements, namely a stiffness, viscosity and force generator.

Hill originally proposed a two-component model comprising a series elastic component representing the tendon and a contractile component representing the muscle belly. The contractile element is composed of parallel stiffness and viscosity as well as force generator due to muscle activation. The series elastic component is in series with the contractile component as shown in the figure below.

Figure 2-2: Muscle Tendon Complex. Muscle belly, modeled as parallel stiffness, parallel viscosity and force generator, is referred to as contractile component. Tendon is modeled using a series stiffness, which is referred to series elastic component.
Note that the force borne by both components is identical, while the length change of muscle is the sum of deformations from both the contractile component and the series elastic component due to this model’s architecture.

### 2.2.1 Series Elastic Element

In simulation, the series elastic element is assumed to have a nonlinear stiffness characterized by the following equation [29]. Apart from the parabolic relationship between strain ($\epsilon$) and force generated by series elastic element ($F_{SE}$), it is also typical to assume exponential relationship.

$$F_{SE} = \begin{cases} 
  k_{se}\epsilon^2 & \text{if } \epsilon > 0 \\
  0 & \text{otherwise}
\end{cases} \quad (2.1)$$

Where $\epsilon(t)$ is described by $\frac{L_{SE}(t) - L_{SE,0}}{L_{SE,0}}$

$L_{SE}(t)$ represents the series elastic element’s length.

$L_{SE,0}$ represents the series elastic element’s rest length.

### 2.2.2 Contractile Element

The contractile element includes parallel stiffness and viscous elements. The critical assumption is that force output of contractile element is a product of four terms, namely muscle activation level, maximum isometric force, force in response to length and force in response to velocity. In other words, activation level, force due to length and force due to velocity are assumed to be independent of one another. This assumption is valid for a steady state force response and is commonly used in muscle modeling [9].

$$F_{CE}(t) = ACT(t) \cdot F_{max} \cdot F_{L}(L_{CE}) \cdot F_{V}(V_{CE}) \quad (2.2)$$

Where $F_{CE}(t)$ is the contractile element’s length.

$ACT(t)$ is the muscle activation level.
$F_{\text{max}}$ is the maximum isometric force.

$F_{L}(L_{CE})$ is force generated according to the force-length relationship.

$F_{V}(V_{CE})$ is force generated according to the force-velocity relationship.

$L_{CE}$ is the contractile element’s length.

$V_{CE}$ is the contractile element’s velocity.

**Force-Velocity Relationship**

Hill’s characteristic equation describes the force-velocity relationship during contraction as a rectangular hyperbola [28]. Force during muscle shortening at low velocity is higher than force at high velocity. Originally, Hill suspected that parameters in his equation would be found from trials of experiments. However, when trying to fit Hill’s equation to experimental data such that the parabola is constrained to pass through the observed maximum isometric force, deviation from Hill’s prediction for high force of interest is below the actual force. Therefore, two separate equations should be used to capture the force-velocity relationship during shortening and lengthening of muscle.

During shortening, equation (2.3) is used. However, the observed force is higher than that predicted by continuation of Hill’s equation for muscle shortening and the force-velocity relationship during lengthening is modeled using equation (2.4) [16].

$$\frac{F_{V}}{F_{\text{max}}} = \frac{1 - V_{CE}/V_{\text{max}}}{1 + (1/K)(V_{CE}/V_{\text{max}})} \quad (2.3)$$

Where $F_{V}$ is the force from force-velocity relationship.

$F_{\text{max}}$ is the maximum isometric force.

$V_{CE}$ is the contractile element’s velocity.

$V_{\text{max}}$ is the maximum shortening velocity.

$K$ is the curvature constant

$$\frac{F}{F_{\text{max}}} = 1.8 - 0.8 \left(1 + \frac{V}{V_{\text{max}}}\right) \quad (2.4)$$
Figure 2-3: Force Velocity Relationship taken from [22]. During shortening of muscle, force decreases as the magnitude of velocity increases. For muscle lengthening, muscle force increases much more rapidly than in the case of muscle shortening. Equation (2.3) and equation (2.4) are good approximations for this force-velocity relationship.
Parameters that are native to each muscle type are maximum isometric force, maximum shortening velocity and muscle lengths. Maximum shortening velocity depends on many factors, for example, different body masses [2], different temperature at which the muscles are studied, etc. Maximum isometric force usually varies according to muscle cross sectional area.

**Force-Length Relationship**

Muscle force depends on sarcomere length and muscle fiber architecture, i.e. pennation angle. If the fibers are parallel to the longitudinal axis of the muscle, then the pennation angle is zero. Normally pennate muscles generate larger amounts of force [22].

The force-length relationship describes the steady state force produced by crossbridges under an isometric condition (when muscle length is fixed). At lengths smaller than the muscle rest length ($L_0$), the number of crossbridges is reduced. Hence, force produced is lower. As muscle length increases, the number of crossbridges increases, leading to a larger production of force. Thin filaments slide over thick filaments before they reach the bare zone, where the maximum force $F_0$ is observed. Further straining of muscle would lead to the reduction of force generation because the overlapping area between the thin and thick filaments then decreases as explained in the earlier section 2.1.1.

![Figure 2-4: Length Relationships in Sarcomere](image-url)

Figure 2-4: Length Relationships in Sarcomere taken from [34]. $I_{act}$ is the length of actin filament. $I_z$ is z-disk length. $I_{bz}$ is the length of bare zone. $I_{myo}$ is the length of myosin filament. $I_{sar}$ represents the overall sarcomere length.
A plausible model for the active (developed) force-length relationship is a bell-shape curve with subtle rising and falling edges. The force-length curve can be described using the following equation.

\[ F_{L(L_{CE})} = e^{c \left( \frac{L_{CE} - L_{opt}}{L_{opt} - L_{rest}} \right)^3} \]  

(2.5)

Where \( L_{CE} \) is the contractile element's length.

\( F_L \) is force extracted from the force-length relationship.

\( L_{opt} \) is the contractile element's rest length.

\( c, w \) describe the shape of the bell curve.

Figure 2-5: Force Length Relationship from [22]. These two graphs show active (developed) force-length relationships for two different types of muscles. \( t_0 \) represents the rest length of muscle’s contractile element. The passive force-length curve results from the parallel stiffness and viscosity in muscle.
2.3 Neural Reflex Pathway

The central nervous system (CNS) receives information from muscle via nerve fibers. The nerve fibers are mainly from two organs, namely muscle spindles and golgi tendon organs. Muscle spindles send out information about length and length change via Ia and II afferent fibers. Other nerve endings found in muscle spindles are called γ motor neurons. By activating γ motor neurons, the CNS can adjust the sensitivity of muscle spindles to length and/or length change. This enables muscle spindles to provide accurate information at any operating lengths. Golgi tendon organs send information about the load acting on a muscle unit to the CNS via Ib afferent fibers.

The CNS processes the retrieved information and sends commands down via α motor neurons which innervate muscle fibers, initiating muscle contraction. The question lies in how CNS computes such commands. Many hypotheses and conjectures have been made to answer such question. More details on possible models for command generation are present in the next two sections.

2.3.1 Muscle Spindles

Muscle spindles are found within the muscle belly. They lie in parallel to extrafusal muscle fibers (main muscle fibers). Muscle spindles are encapsulated sensory receptors, composed of two types of intrafusal muscle fibers, nuclear bag and nuclear chain. The nuclear bag fibers are divided into two groups, static and dynamic.

Sensory fibers wrap around and end on the central part of intrafusal muscle fibers. They consist of two main receptors: primary (Ia afferents) and secondary (II afferents). The Ia afferents entwine all types of intrafusal fibers in the spindle and are sensitive to length and length change, while the length-sensitive II afferents spirals around static nuclear bag and nuclear chain fibers.

Apart from sensory nerve endings, muscle spindles also receives input from CNS via γ motor neurons. The static γ motor endings innervate static nuclear bag and nuclear chain fibers. The dynamic γ motor endings innervate dynamic nuclear bag fibers. By activating the dynamic γ motor neurons, sensitivity of Ia will increase,
whereas sensitivity of II afferents remain unchanged. In other words, by activating dynamic $\gamma$ motor neurons, sensitivity to length will remain the same, while sensitivity to length change will increase. Muscle spindles can be modeled as simple as a single pole disregarding the dynamics of $\alpha-\gamma$ coactivation.

Figure 2-6: Muscle Spindle from http://www.abcbodybuilding.com. This figure shows the architecture of muscle spindles and how they are innervated.

2.3.2 Golgi Tendon Organ

Golgi tendon organs are composed of collagen strands, situated at the insertion of muscle fibers and tendons. Each organ sends information to a single type Ib afferent axon. Because golgi tendon organs are in series with muscle fibers, they can measure the amount of force borne by muscle fibers. As muscle contraction occurs, the collagen strands lengthen causing the deformation of the afferent axon terminals. This leads to the depolarization of the membrane and the transmission of a nerve impulse. The force signal is then sent to the spinal cord. Empirically, the discharge rate of golgi tendon organs is approximately linear with muscle force [24].
Another important function of a golgi tendon organ is to damp out excessive amounts of force imposed on muscle to prevent any damage to muscle fibers [10].

Figure 2-7: Golgi Tendon Organ from http://www.unmc.edu/Physiology. Golgi tendon organ situates between muscle fibers and tendon. When muscle is loaded, collagen fibers will stretch and thus cause mechanical stimulation of Ib afferents.

In simulation, simple gain feedback is used for representing behavior of golgi tendon organs. The stimulation signal represents centralized computed command coming from CNS. This command computation is modeled based on delayed force signal sensed by golgi tendon organs. Additionally, the base command that represents stimulation signal at zero sensory signal is taken into account.

\[
stim(t) = G F_{\text{muscle}}(t - t_{\text{delay}}) + \text{stim}_{\text{offset}}
\]  

(2.6)

Where \(stim(t)\) is the computed command from CNS based on sensory information sent from golgi tendon organ.

G is the feedback gain.
$F_{\text{muscle}}$ is force sensed by the golgi tendon organ.

t$_{\text{delay}}$ is time delay due to synapses and signal transportation typically ranges from 30 to 50 ms.

stim$_{\text{offset}}$ or stim$_{\text{bias}}$ represents stimulation level previously existed in muscle.

### 2.3.3 Spinal Cord

The spinal cord is a part of the CNS that extends from the brain stem and ends at the filum terminale [10]. In the spinal cord, there are sensory neurons, interneurons and motor neurons. Sensory neurons' cell bodies reside in the dorsal root ganglion. Their axons extend into the grey matter in the spinal cord. See figure 2-8.

The signals are transferred through interneurons residing in the grey matter. In the grey matter, there also exist motor neurons whose axons extend into the ventral root. The nerve carrying nerve impulses from sensory organs into the spinal cord is called afferent nerve. The nerve carrying nerve impulses away from the spinal cord is called efferent nerve. The white matter which surrounds the grey matter contains groups of nerve fibers which carry information to/from the brain, and up/down the spinal cord [31].

### 2.3.4 Excitation Contraction Coupling

Muscles are activated through nerves. Various types of innervations are found in different species. In order to achieve muscle contraction, commands sent from CNS must encode the correct information. This command is called action potential (AP). CNS sends out action potential via efferent nerves. AP travels down one or more motor neurons. Since axon of motor neuron is not directly connected to the muscle fibers, in order to transfer information, the induction of potential due to AP must take place. At the neuromuscular junction (gap between the axon of motor neuron and muscle fibers) AP causes the release of neurotransmitters such as acetylcholine. It is released at the end of axon during a very short period equal to approximately 2 ms for every action potential. On the muscle side, there are neurotransmitter receptors. These
receptors change shape after binding with neurotransmitters, leading to the change in membrane potential (EPSP-excitation post-synaptic potential) on the sarcolemma of muscle fiber. When EPSP exceeds the threshold, an action potential is generated. Enzyme called acetylcholinesterase then promptly destroys acetylcholine.

An action potential then reaches the sarcoplasmic reticulum (SR) where $Ca^{2+}$ are stored and then enters muscle fibers at every t-tubule. On the SR membrane, there are two special proteins, namely $Ca^{2+}$ gate and $Ca^{2+}$ ATPase. As the action potential travels along t-tubule, the $Ca^{2+}$ gate opens allowing sudden release of $Ca^{2+}$ into the sarcoplasm. Troponin in the sarcoplasm then binds to $Ca^{2+}$ causing a change
of shape. This results in the formation, rotation and breakdown of crossbridges. As long as $Ca^{2+}$ concentration in the sarcoplasm is high, crossbridge cycling persists. When the $Ca^{2+}$ gate closes, the $Ca^{2+}$ ATPase starts the breakdown of ATP. In this process, $Ca^{2+}$ is used. This process is much slower than the release of $Ca^{2+}$. When $Ca^{2+}$ concentration is low enough, troponin starts losing its $Ca^{2+}$, crossbridge cycling stops and this ends muscle contraction [5].

Figure 2-9: Simple diagram representing $Ca^{2+}$ gate and $Ca^{2+}$ pump at sarcoplasmic reticulum membrane taken from [5].

The time difference from when the muscle activation takes place to when muscle contraction occurs is approximately three times the excitation-contraction coupling time constant. In Rana Pipiens, this time delay is approximately 20-30 ms in fast twitch fiber and 50-80 ms in slow twitch fiber. This excitation contraction coupling dynamic is modeled as a first-order system with a time constant of roughly 10-25 ms [11, 32].

In the simulation, excitation contraction coupling is modeled as a first order system with one real pole. Transfer function relating central commands (stimulation signal) to muscle activation is shown below.
Where \( \text{ACT}(s) \) represents a laplace transform of activation signal in which muscle perceives.
\( \text{STIM}(s) \) represents the centralized command from CNS.

### 2.4 Dynamical Model

The purpose of this project is to investigate the properties of positive force feedback in a hopping model using both simulation studies and experimental evidences. Thus, a hopping model must be developed. This simple model for hopping has a point mass which represents the overall body mass (\( m \)) concentrated at a single point. The upper and lower legs with equal lengths (\( L_s \)) connect at the knee joint. Both segments are assumed to have negligible masses and inertias. The muscle generates torque (\( \tau_m \)) around the knee joint that has a constant moment arm (\( r \)). The system can be captured using two states, which are either \((y, \dot{y})\) or \((\theta, \dot{\theta})\).

When the body is in the air, the governing equation follows the law of gravity. Once the lower limb hits the ground, the muscle is activated. Clearly, the center of mass (CM) height will determine a switch between these two phases, namely the swing phase (body is in the air) and the stance phase (the muscle is active).

The equation of motion during the swing phase is simply a free fall equation. However, a slightly more complicated governing equation during the stance phase is due to kinematics of the segmented leg. Please refer to appendix A for a derivation using the Lagrangian approach. Below is the equation of motion during the stance phase.

\[
\ddot{y} = \frac{\tau_m}{mL_s \cos(\theta/2)} - g
\]
Figure 2-10: 1-D Hopping Model. The model has a point mass and a two-segmented leg with negligible masses and inertias. The muscle is attached to the knee joint and another location between the hip joint and the knee joint. Muscle generates torque around the knee joint with a constant moment arm \((r)\). \(\theta\) represents the knee angle, which can be directly computed from the position variable, \(y(t)\).

where,

\[
y = 2L_s \sin(\theta/2)
\]  
\(\text{(2.9)}\)

\[
\tau_m = r F_m
\]  
\(\text{(2.10)}\)

Where \(\tau_m\) is the only input and represents the torque generated by the muscle.

\(r\) is a constant moment arm of the muscle about the knee joint.

\(F_m\) is described by equation (2.2).

According to the assumption that muscle tendon complex (MTC) consists of the contractile element in series with the series elastic element, two following equations can be deduced.
\[ F_m = F_{CE} = F_{SE} \quad (2.11) \]

\( F_m \) represents force produced by the muscle.

\( F_{CE} \) represents force produced by the contractile element.

\( F_{SE} \) represents force produced by the series elastic element.

\[ L_m = L_{CE} + L_{SE} \quad (2.12) \]

\( L_m \) represents muscle’s length.

\( L_{CE} \) represents the contractile element’s length.

\( L_{SE} \) represents the series elastic element’s length.

### 2.4.1 Dimensional Analysis

The key objective of this thesis is to investigate hypotheses related to a positive force feedback control. The dynamic model representing the hopping motion is then introduced in order to characterize the dynamics of the system. In reality, since many muscles act in accordance and/or opposition in order to obtain such periodic motion, to use a single frog muscle in experiment to test such hypothesis, parameter scaling is unavoidable.

Frog muscles have been studied extensively in the literature [12]. A lower limb muscle, Plantaris Longus, from male leopard frog (Rana Pipiens) was used during all experimentation. Experimental methods are described in chapter 3.

Due to variability in muscle parameters, i.e. maximum isometric force, dimensional analysis is introduced as a general guideline to promote comparability across experiment trials. There are four dimensionless parameters for the leg model; each of which represents rescaled length, velocity, stiffness and energy.

Rescaled length:

\[ \pi_1 = \frac{y}{L_s} \quad (2.13) \]
Rescaled velocity:

\[ \pi_2 = \frac{v}{\sqrt{gL_s}} \]  \hspace{1cm} (2.14)

Rescaled equivalent stiffness:

\[ \pi_3 = \frac{kL_s}{mg} \]  \hspace{1cm} (2.15)

Rescaled energy:

\[ \pi_4 = \frac{E_s}{mgL_s} \]  \hspace{1cm} (2.16)

Where \( k \) is the equivalent stiffness of the muscle.

\( E_s \) represents system energy.

\( L_s \) represents the leg length.

These rescaled parameters remain constant in all systems. For example, from equation (2.15) using the assumption that maximum isometric force \( (F_{iso}) \) is proportional to \( kL_s \), one can obtain the following expression,

\[ \frac{F_{iso,1}}{m_1} = \frac{F_{iso,2}}{m_2} \]  \hspace{1cm} (2.17)

Where \( F_{iso} \) refers the maximum isometric force.

Similarly, from equation (2.14)

\[ \frac{v_{max,1}}{\sqrt{L_{s,1}}} = \frac{v_{max,2}}{\sqrt{L_{s,2}}} \]  \hspace{1cm} (2.18)

Where \( v_{max} \) refers to the maximum shortening velocity.
Chapter 3

Methods and Procedures

This chapter explains how the virtual neural control signal is interpreted and implemented with the current hardware setup. It also covers an overview of software implementation and experimental procedures. The first section describes simulation studies as well as system performance indices for the closed loop behavior. The second section covers general information about the experimental apparatus. Then details on the interplay between virtual environment and muscle tissue as well as the approach used for muscle stimulation are provided in section 3.3. Lastly, the experimental protocol is explained in the last section of the chapter.

3.1 Simulation Study

Numerical simulations of the hopping leg model were conducted to study the closed loop system behavior. As previously described in chapter 2, a dynamic model together with a positive force feedback control is implemented using simulink provided by MATLAB v7.0. The forward dynamic simulation is performed using fixed step solver, ode5, with step size of 0.5 ms (or sampling rate of 2 kHz).

The apparatus used for experiments runs on MATLAB’s real time window workshop, or specifically xpc target. The real time workshop only allows certain types of solvers. The most complex solver allowed by the current setup is ode 5 (Dormand-Prince Formula). Details on apparatus can be found in section 3.2.
When modeling the leg dynamics, two critical assumptions were made. The first assumption was that the extensor muscle acted about a fixed moment arm. This is not always realistic in biology. For example, in the elbow joint, the moment arm of the biceps brachii depends on the amount of load acting on it. The second important assumption is that the muscle force at any given time is a product of muscle activation level, force due to length and force due to velocity, and not the cross product of any two of those terms. This assumption only applies to steady state force response. However, when modeling using a Hill type model, this assumption is widely used.

In this section, three main issues regarding closed loop system behavior will be discussed and compared with previous studies on positive force feedback.

3.1.1 Stable Hopping Pattern

Parameters used in simulations are based on previous literature and experiences conducting experiments on Plantaris Longus muscle in leopard frogs (Rana Pipiens). The typical length of muscle is approximately 25 -35 mm. Maximum isometric force ranges from 0.5 to 3N under full activation. Maximum shortening velocity is typically 3-5 Lo/s, where Lo is the optimal muscle fiber length. Other system parameters are found in table C.

Simulation results show that stable periodic hopping can be achieved for a wide range of gain and time delays. Even with the signal transport delay from the modeled golgi tendon organ to “central nervous system” of up to 50 ms, the model is capable of regaining its stability. Note that typically total feedback delay, which already includes signal transport delay and calcium dynamic delay is typically 25-40 ms. In appendix D.1, the effects time delay on system performances are illustrated.

Another crucial property of positive force feedback is its ability to stabilize system mechanical energy in the presence of disturbances. Refer to figure 4-3.
3.1.2 Spring-Like Leg Behavior

Elastic leg behavior is observed in animal and human locomotion. Simulation results also show elastic leg behavior after the positive force feedback controller is enabled. For example, if constant muscle activation is applied, leg force as a function of leg compression is no longer spring-like in character. Examples of this behavior are shown in figure 4-1 and 4-2.

3.1.3 Effect of Change in Control Parameters

Feedback gain and stimulation offset in a positive force feedback controller are adjustable to achieve different system performances. In linear systems, the higher the feedback gain, the more likely the system will go unstable. However, it is not necessary the case for nonlinear systems.

In order to understand the effect of control parameter variations, feedback gain and stimulation offset were altered while observing the two performance indices, namely hopping height and hopping frequency. Plots are shown in section 4.2.1.

3.2 Experimental Apparatus

In order to transfer the dynamic environment of the point mass model to an experimental setting, the interaction between the actual mechanical system (muscle) and the simulated system must be merged as accurately as possible. In a mechanical system, the interaction port can be described by two parameters–force and velocity/displacement. During the experiment, force response will be monitored and utilized to generate appropriate electrical stimulation to the nerve while imposing displacement on the muscle.
This muscle testing apparatus is capable of simulating different mechanical boundary conditions and environments. It can impose force independent of its motion or displacement as well as electrical stimulation via square pulses of different stimulation parameters.

A high-bandwidth voice coil motor (VCM) is used for adjusting positions of the end effector. In short, current through the VCM results in a proportional force acting on the muscle end effector. A muscle is dissected along with the bone chips to minimize tendon damage and secured to reflex clips, which are firmly sutured to the end effector. Two main sensors involved are a non-contact magnetic encoder, which gives 1 μm resolution and a strain gage based load cell along with a wheatstone bridge amplifier, which gives out force readings. Muscle is stimulated via a contact electrode through the sciatic nerve. Electrical stimulation parameters such as frequency, pulse width, pulse amplitude and number of pulses per trigger can be programmed in real
The system was designed and built so that it is compatible with muscle with lengths between 10-40 mm. System bandwidth is approximately 150 Hz, which is much higher than that expected from muscle responses in vivo. The sampling rate is 2 kHz. Details on the muscle testing apparatus can be found in [12].

3.3 Real-Time Workshop Software

The real-time simulink model is compiled and transferred onto a target computer, which runs the visual C++ version of MATLAB simulink code. The target computer directly communicates with the apparatus. This connection however can be interrupted via command from the host computer. The graphical user interface software allows changes in virtual environment and stimulation parameters. After the experiment is completed, saved data files can be used for post-processing.

Three dynamic phases are modeled. The first phase is called the stance phase, where the interplay between the muscle and simulated mechanical system occurs. During this period, depending on force output as a response from imposed motion, stimulation parameters are adjusted. More details of how muscles are stimulated are in subsection 3.3.1. The other two phases are the flight phase and the extended flight phase. During the flight phase, the two-segmented point mass is under the influence of gravity. While in the extended flight phase, the mass stays at the same height with zero velocity and zero acceleration. This phase is necessary for this experimental protocol and is explained in subsection 3.3.1.

3.3.1 Hopping Model

As previously mentioned in earlier chapters, a hopping model will be investigated in this thesis. The model consists of two phases, namely stance (contact) phase and flight (swing) phase. Stance phase is when the lower leg segment touches the ground. Flight phase refers to the period from take-off to right before the moment when leg segments touches the ground. Refer to figure 2-10.
Stance Phase

During the stance phase, the extensor muscle produces force in response to an imposed length change and electrical stimulus computed from the model. The response from the muscle is then fed back and used to compute appropriate length change and stimulus. The forward path is a transfer function from muscle stimulus to force output. This solely depends on muscle properties. The feedback path takes force as an input to the controller and returns a stimulation signal that ranges from 0 to 1, which then gets added to a constant offset, called stimulation bias or stimulation offset.

Figure 3-2: Positive Force Feedback Block Diagram. Constant stimulation level is represented by a constant stimulation bias (stimulation offset). Stimulation bias along with the computed command make up the stimulation signal. This stimulation signal is restricted between 0 and 1. Then nerve electrode uses this stimulation signal to compute appropriate pulses. Details on how stimulation signal is interpreted can be found in section 3.3.2. According to the nerve impulse and the imposed length change, the muscle responds according to its own dynamics, producing muscle force which is then used for computing the next iteration of stimulation signal.

The reflex control block is shown in figure 3-3. The force signal is delayed by \( t_d \) before it gets multiplied by the controller gain.
Figure 3-3: Inside Reflex Control Block. The golgi tendon organ is modeled as simple proportional feedback with time delay. Delayed force signal is multiplied by the feedback gain, producing stimulation signal, which is then added to a constant stimulation offset to produce overall stimulation of muscle.

The interpretation of stimulation signal is discussed in section 3.3.2.

**Flight Phase**

During the flight phase, the model is solely under the influence of gravity. The leg configuration remains the same throughout the flight phase. The muscle is not active during this period.

**Extended Flight Phase**

The extended flight phase happens after the flight phase and ends just before the stance phase. During this phase, all parameters are hold at values prior to entering the extended flight phase. The extended phase is added to the simulated environment in order to allow the muscle a longer time to rest before every stance period. For the case where motion command during stance period is abrupt, position controlled loop will not be able to quickly follow such command due to its own natural frequency. Overshoot and delay are expected before the response can catch up to match the desired output. However, since the simulated environment is highly dynamic, it will not be able to allow such overshoot and delay to take place without responding. Thus, by having the extended phase, one can allow for a more meaningful response and use the behavior of muscle to better understand the positive force feedback control.
3.3.2 Physiology and Muscle Nerve Stimulation Parameters

There are three stimulation parameters one must specify in order to stimulate the nerve, namely pulse amplitude, pulse width and pulse train frequency. The question remains how to adjust these parameters in order to replicate what is done in nature. The assumption put forth is based on physiology. In the nervous system, the signals sent down from the CNS to motor neuron, from sensory neuron to the CNS, or even in between the paths, are trains of action potentials (AP). These action potentials have almost identical shape and characteristics.

![Action Potential](http://en.wikipedia.org/wiki/Action_Potential)

The resting membrane potential is typically around -70 mV. Depolarization (increase in the membrane potential) can produce action potential depending on how much membrane is depolarized. If membrane potential increases enough that it exceeds the threshold, action potential will occur. The usual membrane potential threshold is approximately -55 mV. Action potentials are all-or-none nerve impulses with amplitude around 100 mV, and width of 1-2 ms [10].

However, one might wonder how these action potentials carry various information. Despite action potential’s invariant characteristics, variation in pulse frequency and amount of muscle nerves recruited lead to different force output.

Preliminary experiments were conducted to verify that such modulation in frequency can shape the force response curve. As mentioned earlier, not only the com-

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mand for different frequency must be sent out, but also the appropriate stimulation parameter such as pulse width, pulse amplitude and pulse period.

For in vitro nerve stimulation, the typical pulse width is 0.1-0.5 ms, and tetanic frequency is 120-200 pulses per second [pps]. The magnitude of pulse amplitude varies from setup to setup, i.e. how good the contact between nerve and electrode is, etc. Preliminary experiments were carried out in order to identify the maximum activation state in which the force level stays constant regardless of any further increase in stimulation parameters.

From many experimental trials, maximally activated muscle is usually obtained by sending electrical pulse trains with 100-120 pps with pulse width of 0.1-0.2 ms.

**Range of Stimulus Frequency**

The explanted muscle is fixed at constant length during the experiment. Pulse train of certain frequency is delivered to the nerve while observing force output level. Maximum stimulation state is when muscle reaches the tetanic contraction state. At the tetanic fusion frequency, one should expect the force level to stay tetanized for the whole period of stimulation. See figure 3-5.
Figure 3-5: Tetanic Fusion Frequency

The first frequency selected for finding tetanic fusion frequency experiment is 30 pps since it is reasonably close to 50 pps which is the tetanic fusion frequency in mammalian muscles at body temperature [22]. Then stimulation frequencies are increased in the following order: 30, 50, 70, 100 and 110 pps.
Pulse Amplitude

We varied pulse amplitude until further increases in amplitude no longer increased force output. The typical range was around 5-10V depending on contact between electrode and nerve as well as nerve damage due to dissection procedure. Figure 3-6 shows a plot of force response due to a given stimulation with pulse frequency of 80 pps and pulse width of 0.2 ms.

Figure 3-6: Force Response to Stimulation Pattern. The stimulation pattern is shown in the dash line, whereas force response is represented by the solid line. The delay in force response is typically 30 - 40 ms. This is due to response from muscle itself since time delay due to computation and data acquisition is negligible.
3.4 Experimental Protocol

A pair of Plantaris Longus muscles was harvested from male Rana Pipiens. The sciatic nerve was left attached to the muscle for the purpose of this experiment. Each end of the muscle was securely sutured to a small chip, which was then attached to the apparatus. The Ringer's solution submerged the whole muscle. The experimental rest length of muscle was measured when the lower and upper limb were at a 90-degree angle before surgery. The frog's weight was also recorded.
At the beginning of the experiment, the coarse positioning stage was manually positioned such that muscle length corresponds to the rest length measured before dissection. The force reading at this location is recorded as a relative guideline to future change in muscle force. Few stimulation commands are executed to assure good conductance between electrode and nerve.

The first set of experiments were devoted to finding stimulation parameters, that defined maximum activation levels. Adjustment to system parameters such as the model mass, reference angle, or leg length were sometimes necessary to achieve variations in system dynamics. For example, by increasing the model mass, the contact time was effectively increased; however, in this case the muscle might not be strong enough to push the mass off from the ground, causing the leg to collapse. By increasing leg length, the contact time will increase. One factor taken into account during parameter tuning was the limited range of primary stage motion. In other words, displacement command to the VCM from the target computer must be between -3 mm and +3 mm. By decreasing the mass or increasing reference angle, a smaller range of movement was attained. For most experiments, parameters from table C were employed.

Three sets of experiments were then conducted. Due to fatigue, only a few trials were performed on each muscle. Occasionally, Ringer’s solution in the apparatus chamber was replaced with new solution since a highly ionized solution can causes over stimulation of muscle and potentiation. In order to minimize fatigue and potentiation, each experimental trial started approximately 5 minutes after the previous experiment.

The first experimental set has all the same parameters except stimulation offset (one of the control parameters) to show the effect of stimulation offset. The second set kept all parameters constant except for the feedback gain in order to show variation of system behavior due to change in the feedback gain. All parameters for the last experimental set remain constant except for the initial height. System behavior was then observed under different initial conditions. All plots are in section 4.3.
Chapter 4

Results

This chapter contains simulation and experimental plots as well as system performance comparison plots.

4.1 Simulation Results

The plots are divided into three sections. The first section compares work loop plots for the case of constant activation and positive force feedback controller. The second section shows the convergence of steady state hopping heights given various initial heights. In the last section, two contour plots show variation in steady state hopping height and hopping frequency due to change in the feedback gain and the stimulation offset.
4.1.1 Elastic Leg Behavior

Positive Force Feedback

Figure 4-1: Simulated Work Loop Plot for Positive Force Feedback with Normalized Gain of 1 and stimulation offset of 0.01. The leg force is almost linear in leg compression and the area enclosed by the graph is small. This represents spring-like leg behavior which is found to be desirable in locomotion. The parameters used for this simulation can be found in table C.
Figure 4-2: Simulated Work Loop Plot for Constant Activation. Both positive and negative areas enclosed by work loop plot are large, signifying non-elastic leg behavior. This plot is generated using the same parameter set as in the previous figure, figure 4-1 except for stimulation offset (stimulation bias), which now equals to 1.
4.2 Emergent Control Property: Steady State Hopping Height Convergence for Various Initial Heights

Figure 4-3: Steady State Hopping Height at Different Initial Conditions. The hopping height of model represents the maximum steady state displacement ($y(t)$). The model is tested with the feedback gain of 1 and stimulation bias of 0.01. At each data point, the hopping model runs based on different initial heights ($y_0$) which are shown on the x axis. Despite the various initial heights, steady state hopping height is constantly maintained at approximately 617 mm.
4.2.1 Effect of Change in Control Parameters on System Performance

Feedback Gain and Stimulation Offset Effect on Hopping Height

Figure 4-4: Steady State Hopping Height Variation with Control Parameters. As stimulation offset (bias) decreases and/or normalized feedback gain increases, steady state height increases. However, the change in hopping height is not prominent when varying normalized gain that is already high, and keeping stimulation offset constant.
Feedback Gain and Stimulation Offset Effect on Hopping Frequency

Figure 4-5: Hopping Frequency Variation with Control Parameters. As stimulation bias decreases and/or feedback gain increases, the hopping frequency decreases.
4.3 Experimental Results

The experimental plots are divided into four sections as previously described in section 4.1.

4.4 Elastic Leg Behavior

Positive Force Feedback

Figure 4-6: Experimental Work Loop Plot for Positive Force Feedback with Normalized Gain of 0.5 and stimulation offset of 0.1. The system parameters are displayed in table C. Note that the area enclosed by this work loop plot is not large, implying an almost spring-like leg behavior.
Constant Activation

Figure 4-7: Experimental Work Loop Plot for Constant Activation. Notice that the large area enclosed by the work loop plot implies non elastic leg behavior.
Figure 4-8: Steady State Hopping Height at Different Initial Conditions. Each experiment depicted by each data point has different initial heights (as shown on the x axis). However, the steady state hopping height (y axis) does not vary much.
Figure 4-9: Convergence of Hopping Height from Different Initial Conditions. This figure has three separate experiments. Each run starts with different initial height. Eventually the steady state height is achieved. As seen from the three plots, the hopping height \( Y_{CM} \) in each experiment converges to similar steady state value.
4.4.2 Effect of Change in Control Parameters on System Performance

Feedback Gain Effect on Hopping Height and Hopping Frequency

Figure 4-10: Hopping Height and Frequency Variation with Feedback Gain. Both plots exhibit two experiment trials. The upper plot monitors the steady state hopping height while changing controller's feedback gain. The lower plot monitors the hopping frequency in two experiments of different feedback gains. As feedback gain increases, the steady state hopping height increases, whereas hopping frequency decreases. Due to the limited number of experimental trials, neither qualitative nor quantitative agreement can be concluded.
Stimulation Bias Effect on Hopping Height and Hopping Frequency

Figure 4-11: Hopping Height and Frequency Variation with Stimulation Bias. Similarly, no qualitative or quantitative agreement is concluded due to the limited experimental data.
Chapter 5

Discussion and Future Work

5.1 Discussion

Previous studies suggest the existence of positive force feedback in legged locomotion. This thesis investigates closed loop system performance indices, namely elastic leg behavior, emergent control property of positive force feedback and how feedback control parameters can affect steady state height, and hopping frequency. Pilot experimental data has shown qualitative agreement with predicted simulation results in terms of elastic leg behavior and the convergence of hopping height with distinct initial conditions. However, the difference between theoretical prediction and experimental results are substantial between gain parameters and system hopping parameters such as frequency and amplitude. In this section, speculations on plausible causes of such quantitative disagreement are discussed in detail.

5.1.1 Passive Stiffness in Muscles

During experiments, the virtual environment sees muscle tissue as contractile element without series elastic element. The series elastic element component, however, is modeled separately as a nonlinear spring attached to one end of the muscle tissue. By adding this extra virtual spring to an existing passive stiffness of tendon, which is left intact with muscle tissue can produce quantitative discrepancy between the-
oretical prediction and experimental results. Note that virtual spring stiffness used for generating simulation results for figure 4-10 and 4-11 was tuned to achieve better quantitative agreement with experiments. The tuned passive stiffness is higher than that in software for experimental trials. Note that the stiffness adds when they are in series.

5.1.2 Maximum Shortening Velocity

The maximum shortening velocity of Plantaris Longus is known to be around 2-3 \( Lo/s \) where \( Lo \) is the muscle fiber length. However, a better quantitative agreement between simulations and experiments can be obtained by varying this parameter. In most cases, the maximum shortening velocity used in software for experiments is around 5 \( Lo/s \) for smaller-sized frogs and 6 \( Lo/s \) for larger-sized frogs.

5.1.3 The Steady State Force Assumption

The critical assumption in order to attain mathematical tractability of this model is the independency of force due to activation, force due to length, and force due to velocity. As it turns out, in most cases this assumption is not necessary realistic. As demonstrated by the data in Joyce et al. [14], force-length and force-velocity curves do not scale with activation level. Particularly, at low activation level, muscle starts to yield instead of lengthening. Even though a Hill type model cannot capture such yielding behavior of muscle, Zahalak’s version of the sliding filament theory can predict such characteristics [35]. The limitation of Zahalak’s distribution moment model is both mathematical intractability and the difficulty in defining model parameters. Furthermore, Heckman showed that stimulation pulse interval alters the properties of force-length and force-velocity in a single motor unit [6].

In addition to how activation level modifies the force-length and the force-velocity curves, the force length relationship is not completely invariant to the dynamics of muscle length input. For example, during quick stretches, muscle exhibits a substantially different stiffness property.
5.1.4 Potentiation and Fatigue

Potentiation and fatigue are not included in the simulation model. Post-tetanic potentiation (PTP) results in longer relaxation and contraction time as well as an increase in the force output [18]. These time-varying properties of muscle are possible sources for significant differences of experimental results from the theoretical predictions.

5.2 Future Work

The emergent control property of positive force feedback is the key to stabilization of the system. In chapter 4, a system with positive force feedback is able to converge to the same energy state in spite of different initial energy states or similarly initial heights. In order to investigate this property further, disturbances can be added to the system while monitoring the response. Thus, a more convincing evident would be the ability to converge to a particular energy state given time varying disturbances.

Numerical integration error is also of a significant concern regarding the accuracy of these experimental results. However, the limitation of apparatus and real-time workshop only allow fixed step integrators. And even the most complex fixed step solver (ode5) cannot capture the same behavior as variable step solvers, i.e. ode45. Adjustments to current software structure might be able to minimize the differences. For example, having the extend phase leads to some numerical errors due to the discretization of states, which causes the muscle to turn on at slightly different time stamp than the system without the extended phase.

5.3 Thesis Summary

This thesis suggests an alternative approach of employing positive force feedback for neural control in legged locomotion. Three hypotheses concerning positive force feedback systems are investigated. The first hypothesis concerns spring-like leg behavior and how a system with the positive force feedback control tends to introduce such leg operation. According to the simulation and experimental results we have, a hop-
ping model with the positive force feedback control exhibits spring-like leg operation, which is highly desirable in locomotion. We also hypothesize that a system with positive force feedback will converge to a fixed steady state energy level independent of the initial mechanical energy state of the system. Our data supports this second hypothesis. However, we cannot conclude any agreement or disagreement to the third hypothesis, which states that hopping height, hopping frequency and leg stiffness can be modulated by adjusting the feedback gain and/or the stimulation offset due to the limited number of experimental trials. We hope that this work will motivate further studies on the interaction between leg dynamics, skeletal muscle mechanics and neural control of movement.
Appendix A

Equation of Motion Derivation

Figure A-1: Point Mass Hopping Model

\[ y = 2L_s \sin(\theta/2) \]

\[ \frac{dy}{dt} = L_s \cos(\theta/2) \frac{d\theta}{dt} \]

\[ \frac{d^2y}{dt^2} = L_s \cos(\theta/2) \frac{d^2\theta}{dt^2} - \frac{1}{2} L_s \sin(\theta/2) \frac{d\theta}{dt}^2 \]
Compute the Lagrangian:

\[ T = \frac{1}{2} m \dot{y}^2 = \frac{1}{2} m L^2 \dot{\theta}^2 \cos^2(\theta/2) \]
\[ V = mg(2L_s \sin(\theta/2)) \]
\[ L = T - V = \frac{1}{2} m L^2 \dot{\theta}^2 \cos^2(\theta/2) - mg(2L_s \sin(\theta/2)) \]

\[ \delta \theta : \]

\[ \frac{d}{dt} \left( \frac{\partial L}{\partial \dot{\theta}} \right) - \frac{\partial L}{\partial \theta} = \tau_m \]
\[ \frac{\partial L}{\partial \theta} = mL^2 \dot{\theta} \cos^2(\theta/2) \]
\[ \frac{d}{dt} \left( \frac{\partial L}{\partial \dot{\theta}} \right) = mL^2 \ddot{\theta} \cos^2(\theta/2) - mL \dot{\theta}^2 \cos(\theta/2) \sin(\theta/2) \]
\[ \frac{\partial L}{\partial \dot{\theta}} = -\frac{1}{2} mL^2 \dot{\theta}^2 \cos(\theta/2) \sin(\theta/2) - mgL \cos(\theta/2) \]

Therefore,

\[ mL \cos(\theta/2) \left[ L \cos(\theta/2) \ddot{\theta} - L \sin(\theta/2) \dot{\theta}^2 + \frac{1}{2} L \dot{\theta}^2 \sin(\theta/2) + g \right] = \tau_m \]
\[ mL \cos(\theta/2) \left[ L \cos(\theta/2) \ddot{\theta} - \frac{1}{2} L \dot{\theta}^2 \sin(\theta/2) + g \right] = \tau_m \]

Note that

\[ \ddot{y} = L \cos(\theta/2) \frac{d^2 \theta}{dt^2} - \frac{1}{2} L \sin(\theta/2) \frac{d \theta^2}{dt^2} \]

Hence,

\[ mL \cos(\theta/2) \left[ \ddot{y} + g \right] = \tau_m \]
\[ \ddot{y} = \frac{\tau_m}{mL \cos(\theta/2)} - g \]

System of Equations:

\[ \ddot{y} = v \]
\[ \dot{v} = -g + \frac{\tau_m}{mL \cos(\theta/2)} \]

Or,

\[ \dot{\theta} = \omega \]
\[ \dot{\omega} = \frac{1}{L \cos(\theta/2)} \left[ -g + \frac{\tau_m}{mL \cos(\theta/2)} + \frac{1}{2} L \sin(\theta/2) \omega^2 \right] \]
Appendix B

Modeling of Excitation Contraction Coupling

Muscle activation signal ($ACT(t)$) is sent from central nervous system which, in locomotion, is mostly the spinal cord. To activate the muscle, calcium ions need to be released to enable the splitting of ATP, which releases energy. Calcium ion channels are opened due to synapses. The stimulus signal ($stim(t)$) is the signal to stimulate the muscle, where the activation signal is signal sent to activate the calcium dynamic. This process can be modeled as a first order system with time constant of $\tau$, namely the excitation-contraction coupling time constant.

$$stim(t) = ACT(t) + \frac{1}{\tau} \int ACT(t) dt$$

In addition, stimulus signal represents a feedback control signal limited to range between 0 and 1. This control signal is a positive feedback signal, taking into account the force measurement from muscle.

$$stim(t) = \begin{cases} 
stim_{\text{actual}} = stim_0 + K_{\text{gain}}F_m(t - \Delta T) & \text{if } 0 < stim_{\text{actual}} < 1, \\
0 & \text{if } stim_{\text{actual}} \leq 0, \\
1 & \text{if } stim_{\text{actual}} \geq 1.
\end{cases}$$

where,

$stim_0$ is stimulation bias (control parameter)
$K_{\text{gain}}$ is the gain (control parameter)
$\Delta T$ is the force signal transport delay
$F_m$ is the muscle force
Appendix C

Parameter Set

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<thead>
<tr>
<th>Parameter Name</th>
<th>Parameter Value</th>
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<tr>
<td>Maximum Isometric Force</td>
<td>1.5 N</td>
</tr>
<tr>
<td>Contractile Element Rest Length</td>
<td>28 mm</td>
</tr>
<tr>
<td>Series Elastic Element Rest Length</td>
<td>7 mm</td>
</tr>
<tr>
<td>Leg Length</td>
<td>0.5 m</td>
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<tr>
<td>Landing Height</td>
<td>0.99 m</td>
</tr>
<tr>
<td>Reference Angle</td>
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</tr>
<tr>
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<tr>
<td>moment arm</td>
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<td>$c$</td>
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<td>$K$</td>
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<td>$e_{ref}$</td>
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<tr>
<td>Signal Transport Delay</td>
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</table>

Table C.1: Simulation Parameters used in software
<table>
<thead>
<tr>
<th>Parameter Name</th>
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<td>moment arm</td>
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<tr>
<td>Signal Transport Delay</td>
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</tr>
</tbody>
</table>

Table C.2: Experimental Parameters used in software
Appendix D

Additional Plots
D.1 Effect of Change in Time Delay and System Performance

Time Delay Effect on Hopping Height

Figure D-1: Time Delay and Steady State Hopping Height. This plot shows simulated results for hopping height (y axis) at different time delay in the force feedback loop (x axis). This depicts how steady state hopping height changes with respect to time delay. Moreover, it also suggests that the model can attain a cyclic motion for a large range of time delays.
Time Delay Effect on Hopping Frequency

Figure D-2: Time Delay and Hopping Frequency. This plot shows simulated results for hopping frequency (y axis) at different time delay in the force feedback loop (x axis). This depicts how hopping frequency changes with respect to time delay. Moreover, it also suggests that the model can attain a cyclic motion for a large range of time delays.
Time Delay Effect on Leg Stiffness

Figure D-3: Time Delay and Average Leg Stiffness. This plot shows simulated results for leg stiffness (y axis) at different time delays of the force feedback loop (x axis).
D.2 Feedback Gain and Stimulation Offset Effect on Leg Stiffness

Figure D-4: Leg Stiffness Variation with Control Parameters. The simulated data shows variation in leg stiffness as control parameters (feedback gain, stimulation bias) change. Leg stiffness increases as stimulation offset increases and is quite invariant with different feedback gains.
Figure D-5: Experiment 024. The experimental data are plotted in three graphs. The top most plot shows the dynamics of muscle displacement commands and actual muscle lengths. The response follows displacement command very well as the dash line almost overlaps with the dotted line. The middle plot has the center of mass trajectory as a function of time. The model starts off slightly above 1000 mm and eventually converges to a steady state height of around 1200 mm. The shaded area signifies the period when muscle is active. The last plot shows muscle nerve stimulation signal in a dotted line and signal from force sensor in a dash line.
Figure D-6: Experiment 043. The experimental data are plotted in three graphs. The top most plot shows the dynamics of muscle displacement commands and actual muscle lengths. The response follows displacement command very well as the dash line almost overlaps with the dotted line. The middle plot has the center of mass trajectory as a function of time. The model starts off slightly above 86.89 mm and eventually converges to a steady state height of around 111.6 mm. The shaded area signifies the period when muscle is active. The last plot shows muscle nerve stimulation signal in a dotted line and signal from force sensor in a dash line.
D.4 Force Modulation

Figure D-7: Force Modulation and Stimulation Pattern. This experimental data shows stimulation pattern in a dotted line and force response in a dash line.
Bibliography


