Hill muscle model errors during movement are greatest within the physiologically relevant range of motor unit firing rates

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Abstract

This study evaluated the accuracy of Hill-type muscle models during movement. Hill-type models are ubiquitous in biomechanical simulations. They are attractive because of their computational simplicity and close relation to commonly measured experimental variables, but there have been surprisingly few experimental validations of these models during functionally relevant conditions. Our hypothesis was that model errors during movement are largest at the low motor unit firing rates most relevant to normal movement conditions. This hypothesis was evaluated in the cat soleus muscle activated either by electrical stimulation at physiological rates or via the crossed-extension reflex (CXR) thereby obtaining normal patterns of motor unit recruitment and rate modulation. These activation paradigms were applied during continuous movements approximately matched to locomotor length changes. The resulting muscle force was modeled using a common Hill model incorporating independent activation, tetanic length–tension and tetanic force–velocity properties. Errors for this model were greatest for stimulation rates between approximately 10–20 Hz. Errors were especially large for muscles activated via the CXR, where most motor units appear to fire within this range. For large muscle excursions, such as those seen during normal locomotion, the errors for naturally activated muscle typically exceeded 50%, supporting our hypothesis and indicating that the Hill model is not appropriate for these conditions. Subsequent analysis suggested that model errors were due to the common Hill model's inability to account for the coupling between muscle activation and force–velocity properties that is most prevalent at the low motor unit firing rates relevant to normal activation.

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1. Introduction

Physiologically relevant models of muscle force generation are essential for creating realistic large-scale simulations to examine the role of muscle properties in controlling movement and posture. Hill-like models incorporating length–tension and force–velocity properties (Zajac, 1989; Winters, 1990) have become ubiquitous in such studies (Gerritsen et al., 1996; van der Helm, 2000). These models are attractive because of their computational simplicity and close relation to commonly measured experimental variables, but there have been surprisingly few experimental validations of Hill models during functionally relevant conditions. This study evaluated Hill-model performance using physiologically relevant neural inputs and muscle movements to provide bounds on the accuracy provided by such models and clues as to how these models should be improved to best simulate muscle behavior.

The most common Hill model incorporates the assumption that muscle force–velocity, length–tension and activation properties are mutually independent, an assumption known to be incorrect (Jewell and Wilkie, 1960; Rack and Westbury, 1969; Zahalak, 1986; Brown et al., 1999). As a consequence, such models do not capture many nonlinear muscle properties such as history-dependent effects (Edman et al., 1993; Morgan, 1994), length and movement-dependent activation (Caputo et al., 1994; Bainave and Allen, 1996), and yielding (Nichols and Hous, 1976). Therefore, numerous groups have modified the Hill model by incorporating these and other properties (Brown et al., 1996;
Meijer et al., 1998; Shue and Crago, 1998; Brown and Loeb, 2000). Many of these properties, though, have been demonstrated only under laboratory conditions using length and activation inputs that typically are not seen during functional behavior. Therefore, it is not yet clear whether such complex properties are required to predict muscle force responses during normal function.

A previous study in our laboratory (Sandercock and Heckman, 1997) evaluated the common Hill model during simulated locomotor activity in electrically stimulated cat soleus muscle and found model errors to be moderate during muscle contraction (<10%). That study though, was restricted to electrically activated muscle and examined limited movements and stimulation patterns. The goal of the current work was to evaluate Hill model performance during more general conditions, including natural activation. Our specific hypothesis was that Hill model performance during movement is worst at the low motor unit firing rates most relevant to normal movement conditions.

2. Methods

Data were collected from the left hindlimb of 4 cats (2–3 kg). All procedures were approved by the Animal Care Committee at Northwestern University.

2.1. Surgical preparation

Initial surgical preparations were done under deep gaseous anesthesia (1.5–3.0% isoflurane), according to standard procedures in our lab (Sandercock and Heckman, 1997; Sandercock and Heckman, 2001). The soleus muscle was exposed by removing both heads of the gastrocnemius and the plantaris. The fine fascia surrounding the soleus was resected to fully expose the posterior surface of the muscle belly and its tendon. The soleus nerve was isolated and left in continuity. All other distal hindlimb nerves were cut, as were the nerves to the semitendinosus, semimembranosus, and biceps femoris. The distal soleus tendon was attached to a computer-controlled muscle puller via a calcaneal bone chip. Surgically exposed areas of the hindlimb were covered with a pool of mineral oil formed within the pulled-up skin flaps. A spinal laminectomy was performed from L4 to S1. Ipsilateral dorsal roots from L4 to S2 were transected to eliminate sensory feedback from the soleus. Contralateral dorsal roots were left intact, as were all ventral roots. A precollicular decerebration was performed by transecting the midbrain with an ophthalmic spatula and aspirating the entire forebrain. The calvarium was packed with saline-soaked cotton wool. The gaseous anesthesia was then discontinued and the animal was allowed to breathe room air. Radiant heat was used to maintain hindlimb and core temperatures within physiological limits. At the end of the experiment, animals were sacrificed with pentobarbital (100 mg/kg i.v.).

2.2. Protocols

Experiments were designed to evaluate the Hill model’s ability to predict muscle force during movement. Random muscle length changes were used to obtain a broad measure of the model’s capabilities. The nominal perturbation used in all experiments was matched to soleus length changes during unrestrained locomotion (Goslow et al., 1973). It had a length excursion of ±8 mm and bandwidth ranging from 0 to 5 Hz, and was generated by lowpass filtering a normally distributed random waveform. Additional length excursions (±1 and ±4 mm) and bandwidths (2.5 and 10 Hz) were tested in selected animals to differentiate between the effects of muscle length and muscle velocity. All length perturbations were centered about an operating point 8 mm less than physiological maximum. Maximum physiological length (0 mm) corresponded to approximately 4 mm beyond the peak of the tetanic length–tension curve.

Muscle length was controlled by a computer-controlled muscle puller [AV-50; ADI, Alexandria, VA] operating as a position servo. This device had a stiffness of greater than 60 kN/m and a small signal position bandwidth of approximately 50 Hz. Muscle length was measured by an LVDT [500 DC-B; Shaeveitz, Hampton, VA] attached to the puller shaft, and force was measured by a strain gage based transducer [Model 31(stiffness > 2 MN/m); Sensotec, Columbus, OH] in series with the shaft. Force and position data were sampled at 1 kHz [MIO-16; National Instruments, Austin, TX] and saved to a hard disk.

Muscle force was controlled using either electrical stimulation or natural activation. Three animals were used for the electrical stimulation protocol and two for the natural activation protocol, with one of these used in both protocols. Electrical stimulation was applied either using fine stainless steel wires in the proximal and distal portions of the muscle belly or via hook electrodes on the ventral roots. Similar results were obtained with both methodologies. A stimulus intensity 50–100% above that required to elicit full recruitment produced repeatable and consistent forces during all trials. Stimulus trains had constant interpulse intervals (IPIs) and uniformly distributed random IPIs spanning the range from 0.01 s to twice the desired mean IPI (10, 20 and 30 Hz). Data were also collected at a 100 Hz constant stimulation rate in one animal. Stimulation and movement onsets coincided during electrical stimulation (see Figs. 2 and 4). Natural motor unit recruitment and rate modulation patterns were generated using the crossed-extension reflex (CXR) (Powers and Rymer,
1988). This was elicited using manual skin compression at the contralateral ankle and knee joints. Skin compression was adjusted to obtain a range of steady muscle forces, as observed by visual feedback.

The passive response to each perturbation was also measured. Passive responses were subtracted from those measured during muscle contractions to determine the active muscle response. Passive and active trials were separated by a 30 s rest period. To minimize fatigue, at least one minute separated all active trials.

2.3. Selection of crossed-extension trials

A potential problem with the CXR is that activation may vary during the course of measurement. A two-stage process was used to screen out trials with non-constant activation. All trials were initially screened by visual inspection and those with abrupt force changes were removed from further processing. An automated procedure was then used to detect more subtle activation changes. This procedure compared the force response during natural activation to those obtained at three levels of constant frequency electrical stimulation (10, 20, and 30 Hz), approximately spanning the range of normal firing rates for cat soleus (Cordo and Rymer, 1982). A sequential quadratic programming algorithm for constrained optimization [Matlab; The Mathworks, Natick, MA] was used to determine the weighting factors of the three constant stimulation trials to best match each CXR response (Fig. 5A—Results), an approach feasible for the soleus muscle, which contains only slow motor units (Burke et al., 1974). Optimization was performed over the first half of the movement, and validated over the second half. Only trials with RMS errors less than 20% during both the first and second half of the movements were selected for further processing. Using these limits, approximately 30% of the collected CXR trials were retained. Changing the acceptable errors limits to 10% and 30% did not affect the qualitative results, but did influence the number of trials available for analysis.

2.4. Hill-model estimation

The simplest possible Hill-type model, consisting of a contractile element in series with an elastic element, was simulated. The contractile element produces force according to Eq. (1), where \( A(t) \) is the muscle activation function, \( F_{LT}(L) \) is the normalized tetanic length–tension relationship, and \( F_{TV}(V) \) is the normalized tetanic force–velocity relationship, measured at the muscle length corresponding to the peak of \( F_{LT}(L)F_{TV}(V) \) are normalized with respect to the maximal tetanic force (100 Hz) at the peak of the \( L–T \) curve, \( F_{max} \). Based upon previous results (Sandercock and Heckman, 1997), the series elastic element was modeled as a piecewise exponential spring.

\[
F_{CE} = F_{max}A(t)F_{LT}(L)F_{TV}(V).
\]  

The Hill model parameters describing \( F_{LT}(L) \), \( F_{TV}(V) \) and the series elasticity were measured directly for each muscle, as described in detail previously (Sandercock and Heckman, 1997). In contrast, activation, \( A(t) \), is difficult to define in Hill-type models. To avoid an arbitrary model, activation was defined using the experimental data. This was accomplished by applying each stimulation pattern during isometric conditions, measuring muscle force, and computing the activation that caused the Hill model to exactly recreate this force using the previously established \( F_{LT}(L) \) and \( F_{TV}(V) \) curves. Because the equations describing the Hill model are one-to-one functions, they can be inverted to solve for \( A(t) \) (Fig. 1). Timing between isometric and movement trials was carefully controlled to avoid fatigue and potentiation.

The equations describing the Hill-type model were numerically integrated using a fourth order Runge–Kutta method (Press et al., 1986). The model inputs were muscle–tendon length, muscle–tendon velocity and \( A(t) \). Errors between experimentally measured muscle force and that predicted by the Hill model were quantified using percent root mean square (RMS) values (Eq. (2)).

\[
\text{Error} = \sqrt{\frac{\sum_{N}(F_{\text{exp}} - F_{\text{Hill}})^2}{\sum_{N}F_{\text{exp}}^2}} \times 100\%.
\]
3. Results

During constant frequency electrical stimulation, Hill-model errors increased with decreasing stimulation rate and increasing perturbation amplitude (Fig. 2). These errors were manifested as a movement-related decrease in muscle force that was not accounted for by the Hill model. Results were consistent across the three cats used in this protocol, and did not vary with changes in the randomization of the length perturbation.

Decreased Hill-model performance with increased perturbation amplitude was associated primarily with increased muscle velocity, not increased muscle length (Fig. 3). To determine which of these factors was most important, a more complete set of perturbation bandwidths was tested in one animal. Bandwidth was varied by changing the displacement sequence output rate. Therefore, muscle velocity was proportional to the product of the bandwidth and amplitude. Trials with identical velocities had nearly identical Hill-model errors (Fig. 3A), even though displacement amplitudes differed. Note for example the errors associated with the 5 Hz, 8 mm trial and the 10 Hz, 4 mm trial. At each stimulation frequency, there was a nearly linear relationship ($r^2 > 0.98$) between the RMS muscle velocity and Hill-model error (Fig. 3B).

Hill-model errors for randomly stimulated muscle were similar to those obtained with constant stimulation for average stimulation rates of 20 Hz and 30 Hz, but errors at 10 Hz were substantially lower with random stimulation than with constant stimulation (Fig. 4). As with constant stimulation, errors were smaller for the
lower perturbation amplitudes. Again, these results were invariant with changes in the length and stimulation randomization. These results suggest that for stimulation rates above 20 Hz, Hill model performance is not critically dependent upon stimulation dynamics, but rather only mean stimulation rate.

The decreased Hill-model errors for the 10 Hz random stimulation relative to those for the 10 Hz continuous stimulation were due to improved model performance for IPIs less than 100 ms. An analysis of the IPIs contained in the 10 Hz random stimulation pattern showed that approximately 60% corresponded to instantaneous firing rates between 4 and 10 Hz and approximately 30% to rates above 20 Hz. To determine if this distribution could explain the relatively low model errors with 10 Hz random stimulation, we collected data in one animal using 5 Hz constant stimulation and a perturbation amplitude of ±8 mm. Under these conditions, model errors were 43%, down from an average of 118% for the constant 10 Hz stimulation in this same animal. Hence, it appears that Hill-model errors peak in the vicinity of 10 Hz and decrease for both lower and higher stimulation rates. These results, coupled with the

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Fig. 4. Model errors with random stimulation IPIs were similar to those with continuous IPIs for average stimulation rates above 20 Hz. Figure format is identical to Fig. 2. (A) Typical data for one animal. (B) Summary data for all three animals in which this protocol was performed.

Fig. 5. Muscle force responses during natural activation most closely matched those obtained with 10–20 Hz constant frequency stimulation. (A) Typical force response to an applied perturbation (thick line) and the corresponding Hill-model prediction with a constant activation chosen to match the CXR force level before perturbation onset. The medium weight line shows the optimal combination of stimulation responses matched to this trial; the close match implies that CXR activation was nearly constant during this trial, indicating that variations in muscle activation did not contribute significantly to Hill-model errors. As with electrical stimulation, actual muscle forces were consistently less than those predicted by the Hill model. (B) Hill-model errors for one cat as a function of the pre-perturbation force level. (C) Force responses for the CXR trials (gray lines) normalized by the pre-perturbation force level and the corresponding normalized forces in response to constant electrical stimulation (black lines). (D) Estimated contributions of different motor unit firing rates to whole muscle force production. The force contributed by motor units firing at approximately 10, 20, and 30 Hz is plotted as a function of total muscle force. Computed from the same data presented in (B) and (C).
distribution of interspike intervals in the random stimulation patterns, explain the smaller errors for the 10 Hz random stimulation relative to the 10 Hz constant stimulation.

Hill-model errors for naturally activated muscle were most similar to the largest errors obtained during electrical stimulation. Again, actual muscle forces were consistently less than those predicted by the Hill model (Fig. 5A). Similar results were obtained from both cats in which the CXR was used to activate the soleus muscle. Percent RMS errors during movement decreased with increasing force level (Fig. 5B). At low forces, error magnitudes were comparable or higher than those obtained with 10 Hz constant stimulation. Errors decreased with increasing force level, but most remained above those measured at constant stimulation rates of 20 Hz and greater. Even though trials with large CXR activation changes were not analyzed, small fluctuations within the tolerance of our screening process may have added to the variability in the error force relationship.

One explanation for the large errors with CXR activation is that naturally recruited motor units fire predominately in the 10–20 Hz range, where the errors to constant electrical stimulation were found to be greatest. This is supported by the normalized force responses obtained with CXR activation (Fig. 5C). Most cross-extension responses lie between the normalized force responses for the 10 and 20 Hz constant stimulation rates. The actual firing rate distributions can be approximated using the optimization results used to select trials with steady activation by examining the weights chosen for each stimulation frequency (Fig. 5D). Over the forces tested, which were up to 80% of the maximum tetanic force in this animal, more than 85% of the total muscle force was contributed by motor units firing between approximately 10–20 Hz.

4. Discussion

This study evaluated Hill-muscle model accuracy during experimentally simulated functional conditions that included electrical activation at physiological rates and natural activation using the CXR. These activation paradigms were applied during continuous movements approximately matched to the length changes that occur during locomotion. Our results support the hypothesis that Hill-model errors during movement are greatest for the stimulation rates most relevant to normal movement conditions. For large muscle excursions, the errors for naturally activated muscle typically exceeded 50%, indicating that the Hill model is not appropriate for these conditions. Previous studies have reported substantially lower Hill-model errors (~10%) during movement, but have used only tetanic or near-tetanic stimulation (van Ingen Schenau et al., 1988; Sanderson and Heckman, 1997). Therefore, our current results underscore the importance of investigating more functionally relevant stimulation rates. These results were robust with respect to different length randomizations and stimulation patterns with variable IPIs, suggesting that our conclusions are general in nature.

The common Hill model simulated in this study assumes that muscle activation, force–velocity and length–tension properties are independent. This assumption is known to be incorrect. Activation depends on muscle length (Close, 1972; Stephenson and Wendt, 1984; Balnave and Allen, 1996), and also strongly influences length–tension and force–velocity properties (Joyce et al., 1969; Rack and Westbury, 1969; Roszek et al., 1994; Brown et al., 1999; Sanderson and Heckman, 2001). It is likely that these coupling effects led to the observed errors.

Length-related changes in calcium sensitivity result in length-dependent muscle activation (Close, 1972; Stephenson and Wendt, 1984; Balnave and Allen, 1996). This effect is greatest at low stimulation rates, when the response to intracellular calcium concentrations has not saturated. At lower rates, calcium sensitivity increases with muscle length, at least over the ascending limb of the tetanic length–tension curve. This length-dependent sensitivity may explain the shift in peak muscle force to longer lengths at lower stimulation rates (Rack and Westbury, 1969; Brown et al., 1999), and for naturally activated muscle (Sanderson and Heckman, 2001). Huijing has shown that stimulation dependent shifts in the length tension properties can lead to Hill-type model error predictions of up to 50% during isometric conditions (Huijing, 1998). These errors are similar to those reported in this study, and a similar mechanism may account for our results. However, during the dynamic conditions investigated in our experiments, Hill-model errors were related primarily to changes in muscle velocity, not length. Hence, strictly length-dependent changes in activation are not likely to account for our observations, although they may become more important in muscles with shorter fiber lengths and correspondingly steeper length–tension relations than the soleus.

The observed velocity-dependent errors were at least in part due to a movement-related decrease in muscle force that was not predicted by the Hill model. Similar velocity-dependent errors have been noted previously (Shue et al., 1995), as have decreases in mean force during continuous movement (Joyce et al., 1969; Kirsch et al., 1994). This movement-related force decrement is most prevalent at low stimulation rates, implying a coupling between activation and force-velocity properties. This phenomenon can be demonstrated in cross-bridge models incorporating a dependence between
physiologically based activation and cross-bridge attachment (Zahalak and Ma, 1990). It may also be facilitated by movement-enhanced calcium release from troponin, which can accelerate muscle relaxation (Caputo et al., 1994), or by a movement-related decrease in cooperativity between neighboring actin–myosin force generating sites (Gordon et al., 2000).

Two recent studies have attempted to modify the Hill model to account for the errors associated with assuming an invariant normalized force–velocity relationship. Shue et al. (1995, 1998) implemented a direct coupling between activation, length and velocity. Brown et al. (1999, 2000) coupled activation to a delayed version of muscle length, a method that indirectly allowed their model to replicate some of the known interactions between muscle velocity and activation. Both methodologies improved model performance over a range of experimental conditions, indicating that better representation of these interactions is key to improving Hill-model performance. Our results emphasize the importance of such modifications if Hill-type models are to be used for naturally activated muscle.

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References


Adaptive Control of Cyclic Movements as Muscles Fatigue Using Functional Neuromuscular Stimulation

JoAnne Riess and James J. Abbas

Abstract—For individuals with spinal cord injuries, functional neuromuscular stimulation (FNS) systems can be used to activate paralyzed muscles in order to restore function, provide exercise, or assist in movement therapy. In previous work, the pattern generator/pattern shaper (PG/PS) adaptive controller was evaluated on subjects with spinal cord injuries and was able to automatically adjust stimulation parameters to account for individual subject differences and system response nonlinearities. In this study, the PG/PS control system was utilized in extended trials. Results indicated that the controller adapted stimulation patterns in an online manner to account for changes in system properties due to fatigue.

Index Terms—Adaptive control, fatigue, feedforward control, functional neuromuscular stimulation (FNS), neural network, paraplegia.

I. INTRODUCTION

Functional neuromuscular stimulation (FNS) is a rehabilitative technology in which electrical current pulses excite the motor neurons of paralyzed muscles and cause the muscles to contract to perform a task [1]–[3]. This technology is being used to restore hand grasp function in the upper extremity and to assist in standing, stepping, maneuvering, and exercise in the lower extremities of people with spinal cord injuries [3]–[9].

In many FNS systems, the ability to accurately and repeatably control movements is hindered by the response properties of electrically stimulated muscle. These nonlinearities include force–velocity properties, 

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length–tension properties, and nonlinear recruitment properties, as well as muscle fatigue, which alters the muscle response characteristics over time [3]–[5], [10], [11]. Fatigue can be induced rapidly with FNS because the larger, fatiguable muscle fibers are recruited at low levels of stimulation before the more fatigue-resistant fibers are activated [1], [3], [12].

A few control algorithms have been used in extended FNS trials to investigate automatic fatigue compensation. For example, Chen et al. [13] used fuzzy logic to adjust the stimulation intensity on fixed stimulation patterns. Veltink [14] investigated a proportional–integral–derivative (PID) controller that controlled the duration of the stimulation burst delivered to the muscle from one cycle to the next. The controller parameters were based on the passive swing properties of each muscle. Both groups were able to maintain sufficient angle excursion for a longer amount of time with their controller than without it.

In previous work, the pattern generator/pattern shaper (PG/PS) adaptive neural network controller was able to automatically develop a stimulation pattern for the quadriceps muscles of different subjects in order to follow both cyclic isometric force trajectories and movement trajectories [15], [16]. These studies demonstrated the ability of the PG/PS controller to account for nonlinear and dynamic system properties, but did not explicitly evaluate the ability of the control algorithm to account for muscle fatigue. The purpose of the experiments reported here was to characterize the performance of the PG/PS controller in extended trials that resulted in muscle fatigue and to evaluate the need for continual adaptation during cyclic movements. In order to assess the performance of the PG/PS controller, adaptation was intermittently disabled and enabled at fixed points in the trials.

II. METHODS

A. Controller Design

The PG/PS controller is an adaptive neural network controller that has been described previously in [15]–[18]. During this study, the PS component, an adaptive filter, was implemented online as a single-layer neural network. The network was composed of a set of neurons. The output of each neuron was a raised cosine function that was shifted in time with respect to the other neurons. Each neuron generated a nonzero output only over part of the cycle. The PG/PS controller determines the stimulation at each time step in the cycle based on the weighted sum of the neuron outputs. The equation [17], [18] for the controller output is

\[ z(t) = \sum_{j=1}^{m} w_{j}(t) y_{j}(t) \]  

where

- \( z(t) \) controller output;
- \( m \) number of neurons in the PS;
- \( w_{j}(t) \) output weight for the \( j \)th neuron at time \( t \);
- \( y_{j}(t) \) output of the \( j \)th neuron at time \( t \).

To change the stimulation pattern during operation [17], [18], the weights were adjusted (adaptation enabled) based on a scaled sum of previous neuron outputs

\[ \Delta w_{j}(t) = \eta e(t) \sum_{k=1}^{n} \frac{1}{n} y_{j}(t - kT) \]  

where

- \( \Delta w_{j}(t) \) change in output weight \( j \) of the PS;
- \( \eta \) constant coefficient of adaptation affecting the size of the weight change (learning rate);
- \( e(t) \) error measured between the desired and actual output of the muscle contraction;
- \( y_{j} \) output of the \( j \)th neuron at time \( t \).

Fig. 1. Block diagram of the setup. In these experiments, the musculoskeletal system consisted of the quadriceps muscle group and the lower leg. The weights of the pattern shaper were adapted online \( \Delta w(t) \) based on the error between the desired angle \( \theta_{d}(t) \) and the measured angle \( \theta_{m}(t) \). In this study, the pattern generator produced a periodic signal and was implemented as a look-up table.

- \( n \) number of past neuron outputs used by the algorithm (filter order);
- \( T \) sampling period.

In this study, the sampling period was set equal to the stimulation period of 50 ms. By adjusting the weights, the algorithm changes the contribution of each of the PS neuron outputs and, therefore, shapes the controller output (stimulation) pattern.

In this study, adaptation was disabled for periods of 50 cycles. During these intervals, the weights were fixed and, therefore, the same pattern of stimulation was used for each cycle. The controller parameters (learning rate = 0.0002, filter order = 15, and number of neurons = 50) used in this study were determined in previous work [16], [18] and the initial weights on the PS neurons were set to zero. With the initial weights set to zero, no stimulation was delivered until an error occurred and the weights on the neurons were adjusted.

B. Experimental Protocols

The experimental setup for this study (Fig. 1) was similar to that of [16]. The PG/PS controller was utilized to determine the stimulation pattern delivered to the knee extensor muscle group in order to control cyclic movement of the swinging lower leg. A single channel of stimulation (pulse amplitude modulated by the controller) was used to stimulate each leg of three subjects with complete thoracic-level spinal cord injury (T4: male, T10/T11: female, T6: male). The Ocestim (Preller, Santa Cruz, CA) neuromuscular stimulator delivered biphasic pulses of a fixed width of 360 μs at a frequency of 20 Hz to PALS (Axelgaard Manufacturing Company Limited, Fallbrook, CA) self-adhering rectangular 5 cm × 9 cm surface electrodes. The Flock of Birds (Ascension Technology Corporation, Burlington, VT) motion sensors were placed on the anterior calf of each leg (vertical = 0°, extension = positive angle excursion) and the acquired angle measurements were used in controller calculations.

Each trial included 300 cycles of movement in which the desired output trajectory was a raised cosine with amplitude of 30°. The cycle period was 2.5 s with a duty cycle of 60%. During the trial, adaptation was intermittently enabled and disabled every 50 cycles (125 s). In one session, each leg performed the 300-cycle trial twice, with at least 25 min rest between consecutive trials on one muscle. The session was repeated on a separate day. This protocol resulted in four trials per muscle for each of six muscles.

At the beginning of each session and after every trial, static input–output (I/O) data were also acquired. These data were collected by stimulating the quadriceps muscle group for approximately 3 s at each of 30 different stimulation levels to obtain the mean angle excursion of the lower leg when stimulated at each different level. The
stimulation levels were randomized and a rest period of 17 s followed every 3 s of stimulation. The I/O data were used to select the minimum value for the stimulation range of each muscle and to document I/O properties at three stages in the experimental session.

The three evaluation indices calculated for each cycle of movement in these experiments were the peak angle output, the peak stimulation as a percent of the total range of stimulation for that subject, and the root-mean-squared error (rms) between the actual output and the desired trajectory as a percent of the maximum desired trajectory. These variables were denoted as $\theta_{peak}$, $Z_{peak}$, and $e_{rms}$, respectively. The rms error reflects error over the entire cycle including undershoot and overshoot during the rest period. The mean $e_{rms}$ over the 10 cycles before and after each transition in adaptation mode (enabled $\rightarrow$ disabled, disabled $\rightarrow$ enabled) were also calculated for each trial. An ANOVA was performed using SPSS software (SPSS Incorporated, Chicago, IL) on these data and a pair-wise comparison was made based on the estimated marginal means.

### III. Results

In Fig. 2, five cycles before and after each transition mode are shown for one trial from one leg of subject 2. These plots demonstrate that when adaptation was disabled at 125 s (after cycle 50), the stimulation pattern used to activate the muscle elicited the desired response. However, at the end of the interval with adaptation off (cycles 96–100), the stimulation pattern was no longer suitable for generating the desired contraction. When adaptation was reenabled at 250 s (after cycle 100), the network weights adapted rapidly to alter the stimulation pattern and improve movement tracking.

Fig. 3 shows the rms error, peak angle output, and peak stimulation per cycle for the same trial shown in Fig. 2. The accuracy that was obtained in the first 10 cycles of the trial was maintained throughout the remaining cycles of the first interval with adaptation enabled. This performance level was characterized by an $e_{rms}$ of less than 10% and a peak angle output near 30°. Although performance was relatively constant after the first half of the first interval, the peak stimulation per cycle steadily increased. When adaptation was disabled for the first time at the beginning of cycle 51, performance degraded (peak angle output decreased and rms error increased). When adaptation was again enabled, performance improved as the PG/PS controller adjusted the stimulation pattern. As the trial proceeded, this pattern of improved performance with adaptation enabled and degradation in performance with adaptation disabled was repeated throughout the trial. However, the change in performance at the transitions was less pronounced toward the end of the trial.

The ensemble average across all trials with all subjects displays a similar pattern [Fig. 4(a)] as described above. Results from the analysis of variance (ANOVA) [see Fig. 4(b)] indicated that there was a significant change in performance over 50 cycles when a fixed pattern of stimulation was sent to the muscles, and performance significantly improved after adaptation was enabled. Performance was also more consistent with adaptation on.

Fig. 5 shows the muscle I/O properties of one leg from two different subjects. The slope of the curves and maximum angle excursions are different for the two muscles. The differences in the input/output data before and after the 300-cycle trial for each muscle reflect the effects of the stimulation exercise on the muscle.

### IV. Discussion

In this study, we used alternating intervals with adaptation enabled and then disabled to assess the role of adaptation to compensate for fatigue and assess the role of adaptation during repeated activation of one muscle. The muscle was not stimulated to the point of total fatigue to avoid tissue damage. However, the changes in the I/O properties before and after the 300-cycle trials, like those seen in Fig. 5, indicate that alterations in the I/O properties occurred over time, presumably due to fatigue. A change in performance also occurred during the intervals with a fixed stimulation pattern. Over the 50 cycles without adaptation, performance degraded between the first 10 cycles and last 10 cycles of the interval. However, after adaptation was enabled, the controller was able to alter the stimulation pattern to compensate for
fatigue, in most instances. In some trials, performance did not improve with adaptation enabled because the maximum stimulation values had already been reached, indicating that the muscles had fatigued to the point where they could not generate a contraction of sufficient strength.

On the average, the stimulation pattern developed by the controller elicited a more repeatable response during the last interval when adaptation was off than during the first interval. This result is due to the recruitment properties of electrically stimulated muscles, the way in which these muscle fibers fatigue, and the way the stimulation pattern is developed by the controller to minimally stimulate the muscle. At the beginning of the trial, the muscles were activated with values in the lower region of the stimulation range (see Fig. 3). The muscle fibers recruited initially with FNS are the fast-twitch, fatigable fibers. Therefore, when adaptation could be adjusted to account for the fibers that were fatiguing, performance was repeatable. When adaptation was disabled for the first time, the stimulation pattern was adequate for a few cycles at the beginning of the interval, but performance degraded quickly. However, by the second and third time adaptation was disabled, performance did not degrade as drastically. This improvement may be explained by the fact that the controller had increased the stimulation and more fatigue-resistant fibers had been recruited.

McNeal et al. [19] found that fatigue occurred quickly when trying to determine a stimulation pattern for one muscle by manually adjusting the stimulation envelope. In that study, the movement pattern degraded after about 1–2 min of cyclic movements. In the experiments reported here, the stimulation pattern was automatically developed by the controller within 1 min and the adaptive controller was able to adjust the stimulation pattern to account for the degradation in performance due to fatigue. McNeal et al. [19] recommended that a closed-loop controller be implemented in FNS systems to account for fatigue. However, past work has shown that the adaptive controller performed better than a proportional-derivative feedback controller during short cyclic trials [16]. In the longer trials reported here, results showed that the controller was able to adjust the stimulation pattern to account for decreased performance due to fatigue and obtain a repeatable performance until the limits of the stimulator were reached or adaptation was disabled.

The same set of controller parameters (learning rate = 0.0002, filter order = 15, and number of neurons = 50) were used in all trials reported here. This set of parameters worked well for five of the six legs tested (at least two of the trials from each leg had an \( \varepsilon_{	ext{rms}} < 15\% \) during the first two intervals with adaptation on). However, the four trials with one of the six legs had the worst performance (highest rms error and highest variability across the trials). The I/O properties for this muscle exhibited a very steep slope (left leg of subject 3 in Fig. 5). This subject participated in additional trials with a learning rate that was reduced by 75% from the value used in all previous tests and performance improved to the extent that results resembled those shown in Fig. 3. These findings indicate that a simple reduction in the learning rate enabled the controller to obtain good performance in a muscle with high recruitment gain. Further testing would be required to determine if this lower value of learning rate would provide suitable performance on other muscles.

The primary objective of these investigations is to find a stimulation paradigm that can account for intersubject and day-to-day variability in muscle strength and endurance. A controller such as the PC/PS, which can develop a stimulation pattern and account for changes in the musculoskeletal system due to fatigue, would enhance FNS systems in situations where cyclic contractions are desired, such as in exercising...
and stepping. Sensors would be needed when adaptation is enabled, because this controller requires information about position or force to alter the stimulation pattern. The simplest implementation of this controller in a practical system would be one that uses a single learning rate for all muscles. Future evaluations will involve integrating this controller into a multimuscle and multijoint system as well as further characterizing the tradeoffs between speed of adaptation and stability of learning with different learning rates.

REFERENCES