NEUROMUSCULAR CONTROL ADAPTATION DURING RECOVERY FROM INJURY

BY

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DISSERTATION

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Abstract

When faced with acute musculoskeletal injuries, the human neuromuscular control system adapts to the imposed restrictions on motion in order to achieve desired kinematic outcomes. In some cases, these adaptations result in alternate compensation strategies that linger after the injury heals. By combining dynamical-systems modeling and clinical experimentation, this work attempts to explore and explain discontinuous changes in neuromuscular control, as well as the possibility of coexisting longitudinal neuromuscular control paths, during recovery from injury. The specific aims are to explore the origins of such nonlinear phenomena in the context of broken symmetry in simple dynamical-systems models, as well as to examine the clinical progression of adaptive changes in neuromuscular control throughout incremental recovery from a simulated ankle injury.

First, two studies are presented which deal with simple dynamical-systems models in which the purposeful introduction of a broken symmetry is interpreted as an injury. In the first study, the dynamics and adaptive compensations of a two-degree-of-freedom nonlinear oscillator under harmonic excitation are investigated. The adaptive strategy involves a frequency-dependent adjustment to the excitation amplitudes and phases on each degree of freedom in an attempt to maintain symmetric oscillations. The analysis shows the coexistence of distinct branches of control strategies, including a jump from one branch to the other while healing from a symmetry fault. In the second study, a more general periodic excitation is applied to the same oscillator. The frequency-dependent adjustments made to the harmonic excitation break the shape
of the more general periodic excitation, making it impossible to retain symmetry with a global adjustment to amplitude and phase. Nevertheless, the analysis shows that simple adjustments to timing and amplitude commands may suffice when attempting to walk in the presence of injury. Both studies discuss clinical implications to injury rehabilitation and the control of movement during injury.

The final two studies employ a clinical experiment to produce and examine neuromuscular control adaptations with a controlled recovery from ankle motion resistance. The first of these studies examines the compensations developed in response to a reduction in ankle range of motion via increased stiffness of an ankle orthosis. The second study analyzes the progression of compensation strategy changes during a systematic reduction in this stiffness until returning to normal. These studies demonstrate that subjects successfully maintain whole-limb motion during ankle perturbation through a combination of adaptations to kinematic and kinetic strategies. Most of these adaptations return to normal during recovery, but not at the same rate, suggesting a change of neuromuscular control strategies during recovery.

The modeling aspects of this dissertation provide in-depth analyses of broken symmetries in mechanical oscillators, bringing to light the effects of small nonlinearities and multi-harmonic excitations on compensation strategies during recovery from the symmetry fault. Further, the experimental studies extend these abstract findings into clinical relevance by detailing compensations during a simulated recovery from injury. In each case, results suggest that clinicians should consider the possibility that multiple compensation strategies can achieve the same kinematic motor goals, and further that these compensations can follow different paths during recovery.
To Linnea, Louis, and Isla
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6.1 Conclusions

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Chapter 1

Introduction

1.1 Injury and Gait

1.1.1 Introduction to gait

Walking is a fundamental task most people rely on throughout their daily routine. Patients suffering from many different injuries or pathologies strive to maintain their walking ability if possible, developing compensatory strategies to maintain a successful gait. Further, limitations to mobility can have not only physical effects, but also psychological effects such as fear of activity, loss of bone density and strength, and even decreased sense of wellness or depression [1–3]. Measures can be taken in a clinical setting to reduce the effects that some of these injuries and pathologies have on tasks such as gait, but care must be taken to ensure that the corrections are focused on the primary deficit, and not the resulting compensations [4].

Human gait is a quasi-periodic event, where analysis is generally simplified by looking at average behaviors within one gait cycle. A complete gait cycle is defined as heel contact of one limb, ipsilateral heel strike (IHS1), through the subsequent heel contact of the same limb (IHS2), illustrated in Fig. 1.1. This limb is in contact with the ground, termed the stance phase, between IHS1 until the toes lift off the ground, ipsilateral toe-off (ITO). During stance phase, the limb is responsible for supporting the weight of the body, and propelling it forward. At ITO, the limb enters the swing phase until the end of the gait cycle (IHS2), when the next cycle begins. During
swing phase, the limb advances forward in preparation for the next cycle. Perry [4] has divided stance and swing into smaller sub-phases important for different aspects of gait functionality. The stance phase consists of the following sub-phases:

**Initial Contact** The instant that the heel strikes the ground (IHS1).

**Loading Response** Body weight is accepted by the limb and the center of mass vertical deceleration is stopped, but forward deceleration occurs. This phase begins at IHS1 and ends at contralateral limb toe-off (CTO), when the opposite limb’s toes lift off the ground. This phase is also referred to as the initial double-support phase, as the opposite limb is still in contact with the ground.

**Mid-stance** Beginning of single-support phase, where the body weight is accelerated upward and the body begins to rotate over the foot/ankle. This phase begins at CTO and ends at weight alignment over the forefoot (WA), where the center of mass is over the ipsilateral toes.

**Terminal Stance** End of the single-support phase, where the body weight is pro-
pelled upward and forward to resist gravity and maintain gait speed. This phase begins at \( WA \) and ends at contralateral heel strike (CHS), when the opposite limb begins stance.

**Pre-swing**  Last sub-phase of stance, and also the second double-support phase. This phase begins at CHS and ends at ipsilateral toe-off (ITO), when the stance limb completes propulsion through the hallux and lifts off the ground.

The swing phase consists of these sub-phases:

**Initial Swing**  Initial sub-phase of swing where the foot is lifted enough for ground clearance and the limb begins swinging forward. This phase begins at ITO and ends at toe-ankle alignment (TAA), where the ipsilateral toes align with the contralateral ankle.

**Mid-swing**  Limb continues forward advancement and acceleration. This phase begins at TAA and ends at knee-ankle alignment (KAA), where the ankle is aligned with the knee (the tibia is vertical).

**Terminal Swing**  Final sub-phase of swing, where the limb decelerates and prepares for initial contact. This phase begins at KAA and ends at IHS2.

These sub-phases provide a method to discretize similar functional activities within the gait cycle. The events defining these phases may then be aligned temporally to provide more functionally consistent gait cycles when compiling average data, or comparing across subjects [5].

Human gait is a grossly symmetric activity, where clinicians focus rehabilitative efforts on regaining symmetry between limbs after injury [4]. Therefore, many researchers focus on deviations from symmetry as a means to qualitatively and quantitatively assess the effects of injuries and pathologies on gait [6–9]. Others focus on understanding the motor-control adaptation deployed by a person suffering an acute
injury to maintain a gait close to a symmetric walking pattern [10], or on the natural symmetry of the underlying central nervous system (CNS) [11, 12]. While there is debate as to how symmetric unimpaired gait truly is, many of the studies lack statistical power, or a consistent definition and quantification of gait symmetry [13, 14]. Forczek and Staszkiewicz [15] assessed the gait symmetry of 54 able-bodied subjects, finding that while there were asymmetries in the kinematics of the ankle, the temporal and phasic variables of each limb were similar. This suggests that the underlying control of unimpaired human gait is to move the limbs symmetrically, while small disturbances during gait create small deviations from the intended action.

1.1.2 Injury and recovery

Musculoskeletal injuries to the lower limb are studied in biomechanics because of their potential effects to such a basic and necessary task as locomotion in otherwise-healthy individuals [4]. For example, up to 300,000 anterior cruciate ligament (ACL) reconstructions are performed in the United States each year [16]. The CNS must balance intricate muscle commands with several key goals for successful bipedal locomotion: propulsion, weight support, and dynamic balance; while minimizing gross stride-to-stride variability to make the gait a stable and quasi-periodic event. Clinical gait analysis has been validated as a successful means to discover functional limitations of particular injuries, focus rehabilitative efforts on specific functional deficiencies of each patient, and track recovery [e.g., 17].

Many studies have focused on how the kinematics (e.g., body segment and joint angle motion), kinetics (e.g., ground reaction forces and joint torques), and/or muscle firing patterns (electromyography (EMG) recordings of muscle activation signals) of gait change to compensate for the effects of an injury. One of the most frequently studied injuries is anterior cruciate ligament (ACL) rupture due to its inability to heal without surgical intervention. Patients choosing to cope with the loss of the ACL have
been shown to develop altered movement and muscle firing strategies to stabilize the knee [18, 19], or even feed-forward upper-body motions during certain high-demand functional tasks [20]. In many cases, these altered strategies remain even when the kinematics and kinetics have returned [21]. The most commonly found compensation strategy during gait is increased hamstrings activity, which provides antagonistic force to the quadriceps and stiffens the knee [e.g., 22]. Interestingly, some researchers find that ACL deficient subjects adopt different compensation strategies. For example, Torry et al. [23] found that one subgroup of ACL deficient subjects compensated by increasing hip extensors, where a second group stiffened the knee and increased knee extension. Both groups had similar lower-limb kinematics and kinetics.

While ACL deficiency receives much attention, more focus is placed on the healing process of injuries, in order to refine rehabilitation methods and track recovery. Patients choosing to repair torn ACLs show similar deficits as those mentioned above, but as the reconstructed ACL heals, these patients generally regain most of the lost functionality and return to nearly-normal kinematic and kinetic patterns. Wojtys and Huston [24] found that after 18 months, patients returned to normal gait patterns even though significant muscle strength deficiencies persisted. Interestingly, the best-performing subjects activated their hamstrings early (similar to ACL deficient strategies), while the worst-performing subjects activated quadriceps first. These sub-groupings suggest that perhaps physical restrictions, or impairments to sensory feedback, remain for a subset of the subjects. Due to varying compensation strategies and varying demand on the reconstructed ACL, patients generally recover functional abilities like walking before more difficult tasks like hopping [25]. Hopper et al. [26] discovered that subjects recovering from ACL reconstruction returned to pre-injury performance of hopping in the forward direction before the lateral direction, perhaps suggesting more effective feed-forward compensation to the forward motion.

Achilles tendon repair also receives a lot of clinical focus. Some researchers find
persisting plantar-pressure asymmetries after return of normal gait motion patterns [e.g., 27]. In a detailed analysis, Don et al. [28] found that before the tendon could heal, subjects developed ankle co-contraction strategies that then delayed recovery of normal gait function even after full plantarflexor strength recovery. The initial strategy also created persisting muscle activation timing differences after return of normal gait kinematics and kinetics. Finally, to show some results outside of soft-tissue tears, consider a couple examples of recovery effects from orthopedic surgeries. Patients who have recovered from hip replacement have shown slightly more symmetric and faster gait, but limb-loading asymmetry was induced after the replacement, seen via maximal ground pressure under the foot [29]. Similarly, patients healing from ankle fracture surgery were able to maintain overall gait symmetry in terms of ground-reaction forces, while asymmetric plantar pressure profiles remained for several locations under the foot [30].

Due to the unexpected nature of most acute injuries, it is extremely difficult to obtain gait data of subjects before injury. Therefore, the aforementioned comparisons of changes during rehabilitation can usually only be made to an individual’s healed state or relative to a normative control group, which may not be reflective of the individual’s pre-injury state. Thus, there is a need to more fully understand the mechanisms of control change during rehabilitation from an injury that can be directly and confidently compared to the healthy state. Further, examining to what extent these aberrant compensation strategies exist may help separate physiological differences in healing from differences in motor adaptation strategies.
1.2 Considerations of the Control of Gait and Movement

1.2.1 Central pattern generators

It is generally believed that the underlying control of repetitive movements is through spinal-level central pattern generators (CPGs), where Brown [31] first proposed the half-center oscillator concept for mammalian locomotion. His work suggested that movement resulted from two pools of pre-motor neurons that would excite synergistic motor neurons while inhibiting antagonistic motor neurons, creating the oscillatory flexion and extension necessary for movements like gait. He also made assumptions that this “central mechanism” was causative to gait progression, while proprioceptive mechanisms were reactive. These conclusions were drawn from experiments with a deafferented cat where the ankle flexor-extensor muscles were excited to induce muscle activations similar to walking. Many researchers repeated similar experiments with deafferented cats, and many other species, providing further evidence that these central pattern generators can successfully produce rhythmic output in many species, including mammals [e.g., 32–34]. These cases also suggested that the CPGs could not self-initiate the rhythmic activity, indicating the existence a higher-level command [32]. In all of the research on decerebrated cats, perhaps the most clinically interesting results are the findings that cats undergoing complete spinal-cord transection can replicate almost normal gait patterns on a treadmill [35].

The evidence for CPGs in human locomotion is more sparse, partly due to the nature and ability to perform similar experiments as those performed on cats. Even so, there is a certain amount of anecdotal evidence supporting the hypothesis that the human CNS employs similar CPG mechanisms to cats, reviewed in detail by Duysens and Van de Crommert [32]. For example, electrical stimulation of the “flexor reflex
afferent” in patients with complete spinal cord injury (SCI) elicited similar reflexes to those found in cats, suggesting the possible existence of a CPG [36]. Also, while evidence of rhythmic activity after complete SCI is very rare, there is evidence of flexion-extension activity throughout all muscles of the lower limbs when lying in certain positions [e.g., 37]. Similar walking-like motions have been observed for both cats and humans during electrical stimulation of the lower spinal cord, providing further support that there may be similar CPG neural networking between cats and humans [32, 38]. Finally, while not always interpreted as such, infant walking development provides further evidence of human CPGs. For example, a study comparing the kinematic trajectories of various newborn and infant stepping patterns found that all movements (regardless of skill) shared a strong attraction to rhythmic, alternating leg movement [39]. Also, infants just beginning to step on their own produced immediate alternating stepping on a treadmill, closer to adult-like steps than newborn steps [40]. In both cases, an underlying CPG-like mechanism is evident even before infants develop enough motor skill to walk normally.

More recently, focus has been placed on creating models of CPG-based neural control, both to provide further evidence of their plausibility and to better understand the possible functionalities of CPGs. Pribe et al. [41] modeled quadruped and biped CPGs, finding that adjusting amplitude and frequency of the neural oscillators produced the different gaits seen in cats and humans. This work supports the possibility that once given the proper command from the CNS, believed to be proportional to desired gait velocity by a different modeling study [42], the CPG can effectively perform the different gaits required (e.g., walking and running). These simulations validate experimental work showing gait speed is related to mid-brain stimulation levels in cats [43]. Others focus modeling efforts on exploiting the symmetry properties of CPG models, providing further support of their plausibility in human gait [11, 12]. Rybak et al. [44] modeled CPGs to support the hypothesis that there were separate
rhythm generator and pattern formation layers for locomotion. The rhythm generator indicates the timing and phasing of muscle firing, while the pattern formation layer provides the activation levels, allowing adaptable motions from the same CPG neural networks. Modeling by Prochazka and Yakovenko [45] and Yakovenko et al. [46] support Brown’s postulation that CPGs require a combination of descending and sensory inputs to control the phasing of gait. In these works, CPG phasing was initially determined by a descending command. This phasing would closely match the actual kinematics if the CNS had predicted the movement well, while residual errors were corrected by sensory mechanisms [45]. Even though sensory feedback mechanisms play a prominent role in locomotion, it may be possible that, when faced with some physical asymmetry or injury, the CPG could shift in phase and amplitude to recover a symmetric gait after suitable motor adaptation or learning [10, 47].

1.2.2 Movement variance and task objectives

The human body has many more degrees of freedom than are necessary for a task such as gait, leading to the issue of motor redundancy [48]. It is widely believed that the CNS attempts to successfully mitigate the motor redundancy problem by minimizing variance related to goal performance and ignoring variance that does not affect the overall outcome. This CNS strategy has been referred to separately as the uncontrolled manifold hypothesis [49, 50] or the goal-equivalent manifold [51, 52]. Both hypotheses similarly assess the variance of certain motor tasks in terms of goal variance and body variance. Body variance that does not affect the goal lies in the uncontrolled manifold, and can be exploited for better task performance [53]. This ability also allows for more flexibility when overcoming an injury or limitation during the task. Todorov and Jordan [54] provide further evidence that human movement employs goal-level optimization, rather than individual control of all kinematics, through modeling of certain motor tasks with stochastic optimal control. They found
good agreement with experimental results, suggesting that noise not affecting the
goal is ignored (causing increased variance) because the controller gains nothing from
changing it.

A wealth of experimental work supports the uncontrolled manifold hypothesis
regarding human motor control [e.g., 49, 50, 53, 55–68]. For example, variability of
arm joint configurations affecting the aim of a pistol were greatly reduced compared to
those not affecting the targeting error [55]. Also, when subjects are tasked with force
generation with multiple fingers, multiple solutions are displayed which minimize the
variance of total force production across all fingers, as well as the moment about
the forearm midline [56]. Shifting focus to lower-body movements, subjects were
found to maximize stability (or repeatability) of center-of-mass (COM) position [49],
and the corresponding linear and angular angular momenta [57], during sit-to-stand
tasks. Similarly, Toney and Chang [58] showed that subjects utilized strategies that
minimized variance of COM motion during gait.

An equivalent goal might be to consider whole-limb motion (e.g., motion of the
vector from toe to hip), which is highly correlated with COM movement, and has been
shown to be of high importance to the CNS [69]. Assuming the difference between
COM and hip joint center movement are small, considering whole-limb motion rather
than COM motion is simply a Cartesian to polar coordinate transformation. This
transformation may be beneficial as the whole-limb angle then provides a direct com-
parison to individual joint angles when considering goal variability. Biomechanical
and neurophysiological studies support the idea that an important goal for locomo-
tion is limb-level function [69–75]. Simplified models of gait such as the inverted
pendulum or spring-loaded inverted pendulum provide useful templates for model-
ing walking and running across many species with low complexity [70–72]. There
is also neurophysiological and experimental evidence that cats maintain consistent
whole-limb movement, even in the presence of impairment to a joint’s function [69].
Humans have been shown to maintain consistent whole-limb movement while experiencing perturbations during vertical hopping [73, 74]. Also, human lower-limb gait kinematics can be reduced to a two-dimensional space correlated with whole-limb length and orientation [75]. If one of the main kinematic goals of human gait is in fact whole-limb motion, this behavior should also drive CNS adaptations when compensating for injuries or pathologies. A goal of whole-limb motion during gait also correlates well with the CPG theory above, in that the CPG oscillations would tend to directly control limb oscillations rather than other goals like COM movement.

1.3 Adaptation to Perturbations

In order to better understand the adaptation process of the human neuromuscular control system, researchers have analyzed both steady state and transient changes in experimental data of healthy subjects experiencing various gait perturbations. Several researchers have studied the metabolic and functional effects of limb weighting during gait, showing adaptation via timing asymmetry, increased metabolic cost and mechanical power [76–78]. Noble and Prentice [7] found similar adaptations while also analyzing the real-time transient adaptations during gait for unilateral limb weighting. They claim the successful adaptations indicate an adjustment to the internal model of the CNS to compensate for the added inertia. Further, they found secondary adaptations before subjects reached their steady-state compensations, where the foot was lifted higher during swing phase. They suggest this secondary compensation is a parallel adaptation allowing safe foot trajectories while the CNS recalibrates the internal model.

Other adaptation studies have examined the effects of external forces or joint torques applied during certain periods of gait. Blanchette and Bouyer [79] applied forward and upward force to the lower-limb during swing phase, finding a feed-forward
mechanism of increased hamstrings activation before toe-off occurs. The same group also found that subjects returning for a second day of exposure to the external forcing adapted much quicker [80]. Savin et al. [81] performed similar work, but pointed out that adaptations are seen in both limbs, not just the limb with external forcing. Blanchette et al. [82] found that subjects quickly adapted to external plantarflexion torque during swing, increasing tibialis anterior (TA) recruitment to counteract the initial deviations in ankle angle. As the perturbation was removed, the TA returned quickly to normal levels. However, patients still experienced significantly increased ankle dorsiflexion through swing. Gordon and Ferris [83] applied similar plantarflexor torque, but it was controlled by the subjects’ own soleus muscle activations. Adding the perturbation affected ankle angle briefly, but subjects adapted by reducing their soleus recruitment. These researchers also found retained adaptation strategies after three days.

In all of the adaptation experiments described above, it should be noted that negative adaptation was present upon removal of the perturbation, requiring less than about 50 gait cycles to return to normal. All of the researchers conjecture that this negative adaptation provides evidence of changes to the central command of gait. The studies finding retained adaptation patterns across different days further supports this evidence. In general, healthy subjects required about 5 minutes to adapt, or de-adapt, and settle onto their new control strategy for the given perturbation or its removal [7]. None of these previous studies have examined adaptation to injury by systematically moving from the largest perturbation, sequentially through several intermediate perturbations, and ending at zero perturbation in order to simulate various stages of recovery.

Because the ankle is instrumental in gait propulsion [84], ankle stiffness perturbation experiments have been performed previously with orthotic and prosthetic devices. A recent modeling study highlights the importance of proper AFO or prosthetic stiff-
ness [85]. They found that maximal energy storage of the device during gait was not the best solution. Rather, limitations of the kinematics must be taken into account to reduce metabolic cost for the user. This has been found clinically, where decreasing prosthetic foot stiffness increases energy storage, but hinders propulsion [86]. Isakov et al. [87] found that prosthetic ankle stiffness restricts limb advancement, which is counteracted by increased thigh muscle activity. Similarly, a review by Soares et al. [88] concluded that trans-tibial amputees walked with increased work at the hip joint to compensate for the ankle stiffness perturbation. Studies of AFO effects are most commonly performed on populations of stroke survivors. Here, the focus is not necessarily on how patients adapt to the AFO stiffness, but how the AFO stiffness can lead to more normal gait in the presence of their neurological deficiencies [e.g., 89–91].

Since AFOs are worn around the ankle-foot complex, they can also be used to explore how ankle stiffness affects healthy gait. Balmaseda Jr. et al. [92] found that wearing a semi-rigid plastic AFO created some changes to gait timings in a healthy population. Opara et al. [93] found similar results, as well as significant changes to ankle angle and step width. These works have been critical in advancing prosthetic and orthotic design, as well as understanding how ankle stiffness can affect pathological, amputee, and healthy gait. Guillebastre et al. [94] examined the effects of multiple AFO stiffness values on a healthy population. They mainly found significant effects for a rigid AFO, and not for flexible AFOs of varying stiffness, but their analysis was limited to spatiotemporal data. None of these studies on healthy populations have assessed ankle stiffness effects with a combination of kinematics and kinetics, nor have they systematically assessed ankle stiffness effects in a decreasing manner to simulate recovery from injury.
1.4 Specific Aims and Organization

When faced with acute musculoskeletal injuries, the human neuromuscular control system adapts to the imposed restrictions on motion. In some cases, this adaptation results in alternate compensation strategies that linger after the injury heals. This dissertation is focused on studying such control adaptations to injury during gait, as these may have additional impact on the development of rehabilitation interventions. In general, there is a lack of understanding how the altered kinematic and kinetic patterns due to injury are correlated with underlying neuromuscular control compensations, and how such correlations change during recovery. This work combines dynamical-systems modeling and clinical experimentation to explore and explain discontinuous changes in neuromuscular control, as well as the possibility of coexisting longitudinal neuromuscular control paths during recovery from injury.

The following specific aims were addressed:

1. Explore the origins of potential discontinuous control changes and coexisting neuromuscular adaptations in the context of broken symmetry in simple dynamical-system models.

2. Examine the clinical progression of changes in neuromuscular control throughout incremental recovery from a simulated ankle injury.

These specific aims have been addressed in the following chapters, as described below.

Aim 1 Even very simple systems encounter multiple solutions when introduced with a small nonlinearity. Studying such a system allows a better understanding of the dynamics involved, and how coexisting control solutions might emerge during a continual parameter change (i.e., healing). Chapter 2 addressed this aim by exploring the dynamics and symmetry compensations of a two-degree-of-freedom nonlinear oscillator. Coexisting control solution branches emerged, including a jump from one
to the other while healing from a symmetry fault. In Chapter 3, a more general periodic forcing was applied to the same oscillator. Here it was shown that even with a linear system, multi-harmonic excitation prevented the system from attaining perfect symmetry. This result has implications when considering the types of commands transmitted to lower-limb muscles via CPGs.

**Aim 2** A clinical experiment to produce and examine neuromuscular control adaptations with a controlled recovery from ankle motion resistance was performed. Focus was placed on the hypothesized goal of maintaining consistent whole-limb motion during gait, and how the individual joint angles compensate to maintain this goal. Consistent motion of the limb as a whole would indicate a higher-level goal of gross body motion (cf. Sec. 1.2.2, last paragraph). The corresponding neuromuscular contributions were also considered and analyzed via net muscle moments about the joints, and peak EMG activations. Chapter 4 detailed the compensations developed in response to the full ankle stiffness perturbation. The progression of changes to these compensations during systematic reduction in stiffness, i.e., simulating a recovery, were then analyzed and discussed in Chapter 5. These studies demonstrated that subjects successfully maintained whole-limb motion during ankle perturbation through a combination of adaptations to kinematic and kinetic strategies. Most of these adaptations returned to normal during recovery, but some small adaptations remained after full removal of the perturbation.
Chapter 2

Accounting for Nonlinearities in Open-Loop Protocols for Symmetry Fault Compensation

Abstract

In this chapter\(^1\), we consider model examples of dynamical systems with only a few degrees of freedom, and with desirable symmetry properties, and explore compensating control strategies for retaining robust symmetric system response even under symmetry-breaking defects. The analysis demonstrates the distinct differences between linear versions of these models, in which fault-compensating strategies are always found, and weakly nonlinear counterparts with varying degrees of asymmetry, for which a multitude of locally optimal solutions may co-exist. We further formulate a candidate optimization protocol for fault compensation applied to self-healing systems, which respond to symmetry-breaking defects by a continuous process of fault correction. The analysis shows that such a protocol may exhibit discontinuous changes in the control strategy as the self-healing system successively regains its original symmetry properties. In addition, it is argued that upon return to a symmetric configuration, such a protocol may result in a different control strategy from that applied prior to the occurrence of a fault.

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2.1 Introduction

This chapter concerns the notion of fault compensation strategies in dynamical systems and possible pit-falls that may arise due to nonlinearities. Here we consider a fault to be a discrete system failure, and fault compensation to be the control strategy employed that attempts to correct the fault or recover some form of functional system operation or output. Specifically, we are interested in systems that exhibit natural structural symmetry, and that might experience faults that break this symmetry. When such faults occur, a control strategy may be desired that not only maintains a symmetric system response, but also attempts to maintain the pre-fault response amplitude while minimizing controller work. While it will be proven possible to achieve successful fault-compensation strategies for certain linear systems with harmonic excitation, a globally-optimal solution may not be attainable with the addition of even a small nonlinearity.

The breaking of symmetry is an important topic of investigation in terms of fault correction. Analytical and numerical nonlinear dynamics analysis techniques have been used to study such systems, for example in bladed disc assembly vibrations [97–99]. Slight mistuning or deformation of rotor blades can create unexpectedly large amplitude vibrations during normal operation, severely decreasing the rotor’s fatigue life from the expected value. Since manufacturing inaccuracies are inevitable, these faults are corrected by careful design of operating conditions [97]. An example of real-time fault correction, while not necessarily part of a symmetry-breaking fault, can be found in electromagnetic motor design. Here, control strategies are sought to maintain smooth and high torque values in the presence of current faults [100].

The current study is motivated by the biomechanics of injury recovery, and the resulting control adaptations necessary to maintain successful walking gait. Focus is placed on the natural symmetry of human gait [11, 12], on studying the deviations
from symmetry caused by various pathologies and injuries [6, 7, 14], and on understanding the motor-control adaptation deployed by a person suffering an acute injury to maintain a gait close to a symmetric walking pattern [10]. As an example, rehabilitation literature suggests that patients recovering from anterior cruciate ligament (ACL) damage may exhibit discontinuous and coexisting longitudinal neuromuscular control changes during functional tasks, including the possibility of an alternate “healed” state distinct from the pre-injury state [21, 25, 26]. The purpose of this work is to explore the origin of such changes in the motor-control adaptation as well as the apparent coexistence of locally-optimal control solutions in the context of simple dynamical-system models. In the extension, the results of this work could then have implications to our understanding of the human neuromuscular control system’s response to injury recovery, and help clinicians better focus rehabilitative efforts for more effective recovery from these injuries.

In the discussion below, we begin by collecting results from linear analysis that demonstrate that a globally optimal control strategy may always be found to retain a symmetric response, even as symmetry-breaking changes are imposed on the system. The subsequent treatment shows that with the introduction of even small nonlinearities, a compensatory strategy may result in non-trivial bifurcations and the emergence of multiple solutions for maintaining the pre-fault response amplitude. We document these observations in terms of the relationship between forcing frequency and response amplitude, using a combination of numerical continuation and analytical perturbation analyses. Finally, we propose a locally-optimal strategy that attempts to maintain a desired response amplitude with minimal excitation amplitude, and discuss how the nonlinearity affects the system response as the fault continuously heals.
2.2 Inherent Dynamics of Underlying System

We demonstrate below that, in a linear system of a particular form, it is always possible to choose the excitation so as to render the system response invariant under a suitable symmetry operation. As shown by the analysis, in the case that the dynamical system is equivariant under this symmetry operation, the same holds for the excitation.

2.2.1 General theory for linear systems

Let \( x \in \mathbb{R}^n \) be the state of a linear, harmonically excited dynamical system of the form

\[
\dot{x} = A \cdot x + B \cdot u
\]  

(2.1)

where \( A \in \mathbb{R}^{n \times n} \) is a constant matrix,

\[
B := \begin{pmatrix} b & b^* \end{pmatrix}
\]

(2.2)

where \( b \in \mathbb{C}^n \) and superscript * denotes complex conjugation, and

\[
u := \begin{pmatrix} e^{i\omega t} \\ e^{-i\omega t} \end{pmatrix}
\]

(2.3)

Let \( I_n \) denote the \( n \times n \) identity matrix and suppose that the matrix \( i\omega I_n - A \) is invertible. The method of undetermined coefficients then yields a \textit{steady-state solution} of Eq. (2.1) of the form

\[
x = \begin{pmatrix} c & c^* \end{pmatrix} \cdot u
\]

(2.4)

where \( c \in \mathbb{C}^n \) satisfies the linear equation

\[
i\omega c - A \cdot c - b = 0
\]

(2.5)

Let \( T \in \mathbb{R}^{n \times n} \) be an invertible matrix with a nonempty fixed space, i.e., such that at least one of \( T \)'s eigenvalues equals 1. We refer to a solution \( x \) of the linear dynamical
system in Eq. (2.1) as symmetric if $T \cdot x = x$, i.e., if $x$ lies in the corresponding eigenspace $E_1$ for all time. It follows that the steady-state solution in Eq. (2.4) is symmetric if and only if $c$ lies in $E_1$. From Eq. (2.5), the steady-state solution is symmetric provided that

$$b \in (i\omega I_n - A) \cdot E_1$$  \hspace{1cm} (2.6)

We say that the autonomous system obtained by letting $B = 0$ in Eq. (2.1) is symmetric (or equivariant under the symmetry transformation $T$, see [101]) for some choice of $A$, say $A = A_0$, if it holds that $T^{-1} \cdot A_0 \cdot T = A_0$. In this special case, Eq. (2.5) yields that the steady-state solution is symmetric provided that $b \in E_1$.

Finally, suppose that $b$ satisfies Eq. (2.6) for some $A = A_0$. By the smoothness of the matrix inverse, small perturbations to $A$ away from $A_0$ then result in small perturbations to the value of $c$ away from symmetry.

### 2.2.2 Two-degree-of-freedom example

![Figure 2.1: A two-degree-of-freedom coupled mechanical oscillator used to illustrate the fault-correcting strategies and the influence of nonlinearity on the robustness of such a compensatory scheme.](image)

As a special case of the general treatment, consider the two degree-of-freedom, coupled mechanical system shown in Fig. 2.1, where $\zeta$ represents a hardening effect on the middle spring. When $\zeta = 0$, the system dynamics are governed by the following linear equations of motion
\[\ddot{x}_1 + \delta \dot{x}_1 - \eta \dot{x}_2 + \alpha x_1 - \beta x_2 = a_1 \cos(\omega t) \quad (2.7a)\]
\[\ddot{x}_2 - \eta \dot{x}_1 + \delta \dot{x}_2 - \beta x_1 + \gamma x_2 = a_2 \cos(\omega t - \phi) \quad (2.7b)\]

where the parameters \(\alpha, \beta, \gamma, \delta, \eta, a_1, a_2,\) and \(\omega\) are all positive, and \(\phi \in [0, 2\pi)\).

Equation (2.7) takes the form of Eq. (2.1) provided that \(x = (x_1, \dot{x}_1, x_2, \dot{x}_2)^T\).

Consider the transformation \(T\) generated by the reflection \(x_1 \leftrightarrow -x_2\) such that

\[E_1 = \text{span} \left\{ \begin{pmatrix} 0 \\ -1 \\ 0 \\ 1 \end{pmatrix}, \begin{pmatrix} -1 \\ 0 \\ 1 \\ 0 \end{pmatrix} \right\} \quad (2.8)\]

The equations of motion are symmetric provided that \(\alpha = \gamma\). Moreover, a symmetric steady-state response is obtained if and only if

\[a_2 e^{-i\phi} = -a_1 \frac{\beta + \gamma + i(\delta + \eta + i\omega)\omega}{\alpha + \beta + i(\delta + \eta + i\omega)\omega} \quad (2.9)\]

In particular, \(a_2 = a_1\) and \(\phi = \pi\) for the symmetric system. It is straightforward to show that \(\phi < \pi\) when \(\alpha > \gamma\) and \(\phi > \pi\) when \(\alpha < \gamma\), independently of any dependence of \(\omega\) on \(\alpha\).

From Eqs. (2.4) and (2.5), the symmetric steady-state solution is now given by

\[x_1(t) = -x_2(t) = a_1 \Re \left[ \frac{e^{i\omega t}}{\alpha + \beta + i(\delta + \eta + i\omega)\omega} \right] \quad (2.10)\]

i.e., a harmonic response with amplitude

\[\|x_1\|_\infty = \frac{a_1}{\sqrt{(\alpha + \beta - \omega^2)^2 + (\delta + \eta)^2 \omega^2}} \quad (2.11)\]

and phase shift

\[\arctan \left( \frac{(\delta + \eta)\omega}{\alpha + \beta - \omega^2} \right) (+\pi \text{ when } \alpha + \beta < \omega^2) \quad (2.12)\]

The maximum response amplitude is obtained for

\[\omega_{\max} = \sqrt{\alpha + \beta - \frac{1}{2}(\delta + \eta)^2} \quad (2.13)\]

provided that the expression within the radical is positive, as will be assumed hence-
forth. Equivalently, the smallest excitation amplitude $a_1$ required to achieve a desired response amplitude $\|x_1\|_{\infty, \text{des}}$ is obtained by substituting from Eq. (2.13) into Eq. (2.11) to yield

$$a_{1,\text{min}} := \|x_1\|_{\infty, \text{des}} (\delta + \eta) \sqrt{\alpha + \beta - \frac{1}{4} (\delta + \eta)^2} \quad (2.14)$$

For $a_1 = a_{1,\text{min}}$ and $\omega = \omega_{\text{max}}$ it follows that $a_2 = a_{2,\text{sol}}$, where

$$a_{2,\text{sol}} = 2\|x_1\|_{\infty, \text{des}} \sqrt{(\alpha - \gamma)^2 + (\delta + \eta)^2 \left(\beta + \gamma - \frac{1}{4} (\delta + \eta)^2\right)} \quad (2.15)$$

It is straightforward to show that, as a function of $\alpha$, Eq. (2.15) is symmetric about $\alpha = \gamma$ and that the value $a_{2,\text{sol}}$ obtained when $\alpha = \gamma$ is a global minimum.

For $a_1 = a_{1,\text{min}}$ and $\omega = \omega_{\text{max}}$ it further follows that the phase shift of the steady-state response relative to the excitation applied to the first degree of freedom becomes

$$\arctan \left( \frac{2}{\delta + \eta} \sqrt{\alpha + \beta - \frac{1}{2} (\delta + \eta)^2} \right) \quad (2.16)$$

which increases monotonically with $\alpha$. For $\alpha \gg \gamma, \beta, \delta, \eta$, it follows that $\omega_{\text{max}} \sim \sqrt{\alpha}$, $a_{1,\text{min}} \sim \|x_1\|_{\infty, \text{des}} (\delta + \eta) \sqrt{\alpha}$, and $a_{2,\text{sol}} \sim 2\|x_1\|_{\infty, \text{des}} \alpha$. Then, Eq. (2.9) implies that

$$e^{-i\phi} \sim -\frac{i}{2} \quad (2.17)$$

i.e., that $\phi \to \pi/2$ as $\alpha$ grows without bound. In this limit, the phase shift of the steady-state response relative to the excitation applied to the second degree of freedom goes to zero.

In the presence of $\mathcal{O}(\epsilon)$ uncertainty in the model parameters $\alpha, \beta, \gamma$, and $\delta$, the system response may deviate from the symmetric behavior obtained by applying Eq. (2.9) in the absence of uncertainty. In this case, let the function

$$C_{\text{sym}} := \frac{\int_0^{2\pi/\omega} \left( x_1(t) + x_2(t) \right)^2 \, dt}{\sqrt{\int_0^{2\pi/\omega} \left( x_1^2(t) + x_2^2(t) \right) \, dt}} \quad (2.18)$$

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characterize the magnitude of deviation. By the final observation in the previous section, it follows that \( C_{\text{sym}} = \mathcal{O}(\epsilon) \). The open-loop control strategy proposed above thus maintains a symmetric response to the order of uncertainty in model parameters.

### 2.3 Interactions with External Environment

We characterize the loss of symmetry in the linear system models considered in the previous section as a *system fault* that requires active correction in order to retain a symmetric system response. As demonstrated above, such correction is always available, given the invertibility of the matrix \( i\omega I_n - A \). In the special case of the two-degree-of-freedom mechanical system, a symmetric response was attainable for any deviation \( \alpha - \gamma \) from model symmetry by *shaping* the open-loop excitation as per Eq. (2.9). In the previous section, we also considered the additional design objective of achieving a desired response amplitude of \( x_1 \) with the least excitation amplitude \( a_1 \), by suitable selection of the excitation frequency \( \omega \).

We proceed to consider the possible influence of nonlinearities on the system response, given the continued use of the linear shaping of the open-loop excitation as obtained in the absence of such perturbations. Although we imagine that the source of nonlinearities may lie in interactions of the system with its environment, we restrict attention to nonlinearities dependent only on the system state. It remains to be determined whether retaining the linear excitation framework will allow for fault correction, and further allow for an optimal choice of excitation frequency and remaining excitation amplitude, even in the presence of nonlinearities.

To this end, we return to the two-degree-of-freedom mechanical system, but now with \( \zeta \neq 0 \) corresponding to a cubic dependence of the elastic force between the two masses on the relative displacement \( x_2 - x_1 \). Provided that \( \alpha = \gamma \), we again expect to find a symmetric steady-state response (although this may no longer be unique as
a function of \( \omega \)). In the presence of a symmetry-breaking fault \( \alpha \neq \gamma \), however, it may not be possible to achieve a symmetric response, much less find a formula for \( a_2 \) and \( \phi \) in terms of the model parameters, \( a_1 \) and \( \omega \). As per the discussion above, we choose to continue using the linear prescription for \( a_2 \) and \( \phi \), given by Eq. (2.9), and treat this as a control decision arrived at by studying the linear system.

The system dynamics are now governed by the nonlinear equations of motion

\[
\begin{align*}
\ddot{x}_1 + \delta\dot{x}_1 - \eta\dot{x}_2 + (\alpha_0 + \gamma) x_1 - \beta x_2 &= \zeta (x_2 - x_1)^3 + a_1 \cos(\omega t) \quad (2.19a) \\
\ddot{x}_2 - \eta\dot{x}_1 + \delta\dot{x}_2 - \beta x_1 + \gamma x_2 &= \zeta (x_1 - x_2)^3 + a_2 \cos(\omega t - \phi) \quad (2.19b)
\end{align*}
\]

where \( \alpha \) in Eq. (2.7b) has been replaced by \( \alpha_0 + \gamma \) so that \( \alpha_0 = 0 \) denotes a symmetric system. By substituting \( x_1 \leftrightarrow -x_2 \) into Eq. (2.19a) and adding the result to Eq. (2.19b), we achieve a necessary condition for a symmetric system response:

\[
x_1 = -x_2 = \frac{a_1 \cos(\omega t) + a_2 \cos(\omega t - \phi)}{\alpha_0} \quad (2.20)
\]

for \( \alpha_0 \neq 0 \) and

\[
a_1 \cos(\omega t) + a_2 \cos(\omega t - \phi) = 0 \quad (2.21)
\]

for \( \alpha_0 = 0 \). A solution to Eq. (2.21) is given by \( a_2 = a_1 \) and \( \phi = \pi \), which agrees with the condition in Eq. (2.9). In the asymmetric case, we see that the solution, by necessity, must be harmonic with frequency \( \omega \). However, the presence of the cubic term in Eq. (2.19) implies that any periodic solution is expected to contain higher harmonics, for example, with a frequency of \( 3\omega \). It is, consequently, not possible to satisfy Eq. (2.20) exactly, although approximate satisfaction may be possible if the amplitudes of the higher harmonics are very small.

### 2.3.1 Numerical continuation

We proceed to explore the system response in the presence of nonlinearities and possible symmetry faults and with \( a_2 \) and \( \phi \) given by Eq. (2.9), with particular emphasis
on periodic steady-state solutions. In this section, we rely on numerical parameter continuation techniques to track families of such periodic orbits in the vicinity of the linear resonance frequency corresponding to the symmetric, normal mode in the symmetric system. In particular, the results reported below are obtained using a recently developed MATLAB-based Computational Continuation Core (referred to below as coco) [102], and an orthogonal collocation toolbox for discretization of periodic orbits, included with this package. We restrict attention to tracing solution manifolds under variations in $a_1$ and/or $\omega$. Unless otherwise stated, $\beta = 1$, $\gamma = 2$, $\delta = 0.1$, and $\eta = \zeta = 0.05$.

Figures 2.2 through 2.5 show the response amplitude’s dependence on excitation frequency and amplitude. In all cases, the average values of $x_1$ and $x_2$ equal 0. The symmetric system ($a_0 = 0$), seen in Fig. 2.2, behaves like the linear system at small forcing amplitude ($a_1$), where the response amplitude has a resonance near the second modal frequency of the undamped system. As $a_1$ grows, this resonance exhibits the hardening behavior inherent in Duffing-type systems, and sub-harmonic resonances also appear. Figure 2.3a displays an interesting behavior that emerges once we move slightly away from symmetry (e.g., $\alpha = 0.1$). The general shape of the main resonance remains. However, for increasing $a_1$, a plateau emerges for $\omega$ to the left of the main resonance peak. As the system moves further from symmetry (e.g., $a_0 = 2$), as seen in Fig. 2.4a, the plateau widens and develops a significant peak near the main resonance. The prominence of this second peak near the second modal frequency is a potential source of difficulty in seeking to design $\omega$ to maximize $\|x_1\|_\infty$, as will be expanded upon in a later section.

Aside from the multistability associated with the fold bifurcations seen with the hardening resonance, a pair of isola and branch-point bifurcations may be observed for critical parameter choices that may help explain the formation of a plateau along the main resonance. In particular, as observed in Fig. 2.5, an isolated branch of periodic
trajectories is born at an isola bifurcation and subsequently grows and merges with the main resonance curve at a branch-point bifurcation. This merging of solution branches leads to a widening of the resonance peak, ultimately forming the additional peak for certain values of $\alpha_0$ and $a_1$.

As discussed previously, it is not generally possible to sustain a symmetric response for the nonlinear system in the presence of a symmetry fault. We assess the extent to which the compensation strategy given by Eq. (2.9) is able to yield at least an approximately symmetric solution by evaluating the cost function $C_{\text{sym}}$ given in Eq. (2.18). As expected, the symmetric system always exhibits the desired symmetric response (i.e., $C_{\text{sym}} = 0$). Values of $C_{\text{sym}}$ are plotted for the asymmetric cases in Figs. 2.3b and 2.4b. Away from resonances, the system exhibits responses where $C_{\text{sym}} \approx 0$, even in the presence of symmetry-breaking faults. Significant deviations from symmetry are observed for larger oscillations, however, where the effects of the nonlinearity are more pronounced. The highest values of $C_{\text{sym}}$ are found at the primary modal resonance (where the natural dynamics would create in-phase symmetry) and the additional peak on the secondary modal resonance, formed from the isola merging at the branch-point bifurcation. Values of $C_{\text{sym}}$ at the hardening peak are relatively small compared to these other peaks. The additional resonance near the second mode, induced by the nonlinearity and use of the linear excitation conditions in Eq. (2.9), thus creates a region of undesirable symmetry properties close to our optimal excitation frequency. We will discuss below how this might affect our chosen optimization strategy, beyond the possible loss of response symmetry.
Figure 2.2: The response amplitude $\|x_1\|_\infty$ versus $\omega$ for the symmetric system ($\alpha_0 = 0$) for $a_1 = \{0.1, 0.25, 0.75, 1.5, 3\}$.

Figure 2.3: (a) The response amplitude $\|x_1\|_\infty$ versus $\omega$ for a system near symmetry ($\alpha_0 = 0.1$) for $a_1 = \{0.1, 0.25, 0.75, 1.5, 3\}$, and (b) corresponding $C_{\text{sym}}$ values from Eq. (2.18).

Figure 2.4: (a) The response amplitude $\|x_1\|_\infty$ versus $\omega$ for an asymmetric system ($\alpha_0 = 2$) for $a_1 = \{0.1, 0.25, 0.75, 1.5, 3\}$, and (b) corresponding $C_{\text{sym}}$ values from Eq. (2.18). Rectangle denotes plot range of Fig. 2.5.
Figure 2.5: The response amplitude $\|x_1\|_\infty$ versus $\omega$ for $\alpha_0 = 2$ near a branch-point bifurcation, inside the rectangle shown in Fig. 2.4a. Arrows indicate increasing $a_1$ ($a_1 \in [0.1, 0.98]$).

2.3.2 Perturbation analysis

Small asymmetry

We make assumptions about the relative scaling of the model parameters in order to carry out a perturbation analysis to verify our numerical results. Specifically, we assume that the excitation amplitudes, damping coefficients, nonlinearity coefficient, and asymmetry coefficient are all $O(\epsilon)$:

\begin{align}
\ddot{x}_1 + \epsilon \delta \dot{x}_1 - \epsilon \eta \dot{x}_2 + (\epsilon \alpha_0 + \gamma) x_1 - \beta x_2 &= \epsilon \zeta (x_2 - x_1)^3 + \epsilon a_1 \cos(\omega t) \\
\ddot{x}_2 - \epsilon \eta \dot{x}_1 + \epsilon \delta \dot{x}_2 - \beta x_1 + \gamma x_2 &= \epsilon \zeta (x_1 - x_2)^3 + \epsilon a_2 \cos(\omega t - \phi)
\end{align}  \hspace{1cm} (2.22a)

and use the multiple-scales perturbation technique [103] to approximate the solution to Eq. (2.19). Scaling the forcing and damping to be on the same order as the nonlinearity is standard practice for this type of problem [104]. Here we have also scaled the asymmetry ($\alpha_0$) to explore the steady-state behavior for near-symmetric systems.

When $\epsilon = 0$, we obtain the linear, homogeneous, symmetric, conservative system:

\begin{align}
\ddot{x}_1 + \gamma x_1 - \beta x_2 &= 0 \\
\ddot{x}_2 - \beta x_1 + \gamma x_2 &= 0
\end{align}  \hspace{1cm} (2.23)
with eigenfrequencies
\[ \omega_1 = \sqrt{-\beta + \gamma}; \quad \omega_2 = \sqrt{\beta + \gamma} \] (2.24)
and corresponding mode shapes
\[ \Psi_1 = \begin{pmatrix} 1 \\ 1 \end{pmatrix}; \quad \Psi_2 = \begin{pmatrix} 1 \\ -1 \end{pmatrix} \] (2.25)
Our interest lies in excitation frequencies near the second eigenfrequency and mode shape, \( \omega_2 \) and \( \Psi_2 \) respectively, where the linear system exhibits solutions symmetric under the transformation \( T \) given above.

In the case that \( 0 < \epsilon \ll 1 \), let \( T_0 = t, T_1 = \epsilon t \), and \( \omega = \omega_2 + \epsilon \sigma \), and consider the ansatz
\[ x_i(t; \epsilon) = x_{i0}(T_0, T_1) + \epsilon x_{i1}(T_0, T_1) \] (2.26)
Substituting these expansions into Eq. (2.22) and equating the coefficients of successive powers of \( \epsilon \) on both sides of the differential equation gives, for \( \epsilon^0 \):
\[ \begin{align*}
\partial_0^2 x_{10} + \gamma x_{10} - \beta x_{20} &= 0 \\
\partial_0^2 x_{20} - \beta x_{10} + \gamma x_{20} &= 0
\end{align*} \] (2.27)
and for \( \epsilon^1 \):
\[ \begin{align*}
\partial_0^2 x_{11} + \gamma x_{11} - \beta x_{21} &= -2 \partial_0 \partial_1 x_{10} - \delta \partial_0 x_{10} + \eta \partial_0 x_{20} - \alpha_0 x_{10} - \zeta (x_{10} - x_{20})^3 \\
&\quad + \frac{1}{2} a_1 \left( e^{i(\omega_2 T_0 + \sigma T_1)} + e^{-i(\omega_2 T_0 + \sigma T_1)} \right) \\
\partial_0^2 x_{21} - \beta x_{11} + \gamma x_{21} &= -2 \partial_0 \partial_1 x_{20} + \eta \partial_0 x_{10} - \delta \partial_0 x_{20} + \zeta (x_{10} - x_{20})^3 \\
&\quad + \frac{1}{2} a_2 \left( e^{i(\omega_2 T_0 + \sigma T_1 - \phi)} + e^{-i(\omega_2 T_0 + \sigma T_1 - \phi)} \right)
\end{align*} \] (2.28)
The order-\( \epsilon^0 \) equations give the unperturbed system of Eq. (2.23) after time rescaling. Focusing on the second mode, we consider solutions of the form
\[ x_0(T_0, T_1) = \begin{pmatrix} | \\ c_0(T_1) \\ | \\ c_0^*(T_1) \end{pmatrix} \cdot u \] (2.29)
where \( x_0 = (x_{10}, x_{20})^T \), \( c_0 = (c_{10}, c_{20})^T \), and \( u \) is given by Eq. (2.3) with \( \omega \to \omega_2 \).
and $t \to T_0$. Note that due to the symmetry on this order of the perturbation expansion, $c_{20} = -c_{10}$ from Eq. (2.25). Had we not scaled $\alpha_0$ by $\epsilon$, then the resulting magnitudes of $x_{10}$ and $x_{20}$ would not be equal, and our approximate solutions would be asymmetric. Here $c_{10}$ will be determined by the need to eliminate secular terms from Eq. (2.28) after substitution of the zeroth-order solution into the right-hand side.

Similar to Eq. (2.29), the order-$\epsilon^1$ equations will have solutions of the form

$$x_1(T_0, T_1) = \left( \begin{array}{c}
c_1(T_1) \\
c_1^*(T_1)
\end{array} \right) \cdot u$$

(2.30)

Substituting this into the left-hand side of Eq. (2.28) and substituting Eq. (2.29) into the right-hand side, the resulting equations to balance coefficients of $e^{i\omega_2 T_0}$ are given by

$$A_c c_1 = b_c,$$

where

$$A_c = \begin{pmatrix}
\gamma - \omega_2^2 & -\beta \\
-\beta & \gamma - \omega_2^2
\end{pmatrix}$$

(2.31)

$$b_c = \begin{pmatrix}
-2i\omega_2 c_{10}' - (\alpha_0 + i(\delta + \eta)\omega_2) c_{10} - 24\zeta c_{10}' c_{10}^* + \frac{1}{2} a_1 e^{i\sigma T_1} \\
2i\omega_2 c_{10}' + i(\delta + \eta)\omega_2 c_{10} + 24\zeta c_{10}' c_{10}^* + \frac{1}{2} a_2 e^{i(\sigma T_1 - \phi)}
\end{pmatrix}$$

(2.32)

and primes denote differentiation with respect to $T_1$. $A_c$ is singular since $\omega_2 = \sqrt{\beta + \gamma}$.

By the Fredholm alternative, a solution for $c_1$ may be obtained if $b_c$ is in the image of $A_c$ (i.e., $b_c \perp N(A_c^T) = (1, 1)^T$). Therefore, we arrive at our solvability condition for secular terms as $b_c \cdot N(A_c^T) = 0$. After substituting the polar decomposition

$$c_{10}(T_1) = \frac{1}{2} r(T_1) e^{i(\sigma T_1 - \theta(T_1))}$$

(2.33)

where $r \in \mathbb{R}$ and $\theta \in \mathbb{R}$, into the resulting equation and splitting into real and imaginary parts, we can solve for steady-state motions by setting $r' = \theta' = 0$, resulting in the following expression relating the amplitude $r$ and the frequency detuning $\sigma$:

$$\left( \sigma - \frac{\alpha_0}{4\omega_2} - \frac{3\zeta}{\omega_2} r^2 \right)^2 = \frac{a_1^2 + a_2^2}{16\omega_2^2 r^2} - \frac{(\delta + \eta)^2}{4} - \frac{a_1 a_2}{8\omega_2^2 r^2} \cos(\phi)$$

(2.34)

The approximation of our steady-state solution can be found by first substituting...
Eq. (2.33) into Eq. (2.29) and the result of this operation into Eq. (2.26), giving
\[ x_{10} = -x_{20} = r \cos(\omega T_0 + \sigma T_1 - \theta). \]
Now, substituting the frequency detuning and time scaling into this result gives
\[ x_1 = -x_2 = r \cos(\omega t - \theta) + O(\epsilon) \] (2.35)
Therefore, to lowest order, the approximation of the steady-state response near the second resonance is tuned to the excitation frequency, with amplitude given by \( r \), and a phase shift of \( \theta \).

We can rearrange Eq. (2.34), multiply by \( \epsilon^2 \), and make the following substitutions
\[ r^2 \rightarrow x, \ \epsilon \sigma \rightarrow \Delta \omega, \ \epsilon a_0 \rightarrow \Delta \alpha, \ \epsilon \zeta \rightarrow \zeta \epsilon a_1 \rightarrow a_1, \ \epsilon a_2 \rightarrow a_2, \ \epsilon \delta \rightarrow \delta, \ \epsilon \eta \rightarrow \eta \] (2.36)
to achieve an equation in terms of the original parameters before scaling:
\[ f(x, \Delta \omega, a_1) = 0 \] (2.37)
where
\[ f(x, \Delta \omega, a_1) = 144\zeta^2 x^3 + (24\Delta_\alpha \zeta - 96\zeta \omega_2 \Delta \omega) x^2 \\
+ (\Delta_\alpha^2 - 8\Delta_\alpha \omega_2 \Delta \omega + 4(4\Delta^2 + (\delta + \eta)^2) \omega_2^2) x \\
- a_1^2 - a_2^2 + 2a_1 a_2 \cos(\phi) \] (2.38)
Noting that, here, \( \alpha = \gamma + \Delta \alpha \) and \( \omega = \omega_2 + \Delta \omega \), we may now substitute our linear excitation conditions on \( a_2 \) and \( \cos(\phi) \) given by Eq. (2.9) into this result. The solutions to Eq. (2.37) are then the branches of steady-state solution amplitudes \( (\sqrt{x} = r) \) in terms of forcing frequency \( (\omega_2 + \Delta \omega) \). We locate fold bifurcations by solving Eq. (2.37) and
\[ \frac{\partial f(x, \Delta \omega, a_1)}{\partial x} = 0 \] (2.39)
simultaneously for \( x \) and \( \Delta \omega \). Similarly, isola and branch-point bifurcations may be located by solving Eqs. (2.37) and (2.39) together with
(a) $\Delta_\alpha = 0$, $a_1 = \{0.2, 0.3, 0.4\}$.

(b) $\Delta_\alpha = 2$, $a_1 = \{0.1, 0.175, 0.25\}$.

Figure 2.6: The graph of $f (x_b (\Delta_\omega, a_1), \Delta_\omega, a_1)$ versus $\Delta_\omega$ of the symmetric (a) and asymmetric (b) system for increasing values of $a_1$.

\[
\frac{\partial f (x, \Delta_\omega, a_1)}{\partial \Delta_\omega} = 0 \tag{2.40}
\]

for $x$, $\Delta_\omega$, and $a_1$.

Let $x_b (\Delta_\omega, a_1)$ denote the solution to Eq. (2.39). The emergence of the saddle-node bifurcations can then be visualized by plotting $f (x_b (\Delta_\omega, a_1), \Delta_\omega, a_1)$ versus $\Delta_\omega$ for increasing $a_1$ until the resulting curve crosses zero, as seen in Fig. 2.6. For the symmetric system (Fig. 2.6a), as the excitation increases, the graph crosses the axis at two points corresponding to the fold bifurcations that bound a range of values of $\Delta_\omega$ for which there exists three roots of $f$. For each value of $\Delta_\omega$ outside this range, there exists only one root of $f$. Once asymmetry is introduced (e.g., $\Delta_\alpha = 2$ in Fig. 2.6b), the fold bifurcations emerge for slightly lower values of $a_1$. As seen in the figure, there now exists a window of $a_1$ values in which four fold bifurcations may be found. This can be compared to the results of numerical continuation in the previous section, where excitation amplitudes $a_1$ on either side of the branch-point bifurcation resulted in two regions of multistability under variations in the excitation frequency $\omega$ (cf. Fig. 2.5).

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(a) $\Delta_\alpha = 0$, $a_1 = \{0.1, 0.25, 0.75, 1.5, 3\}$.  
(b) $\Delta_\alpha = 2$, $a_1 = \{0.1, 0.25, 0.75, 1.5, 3\}$.

Figure 2.7: The response amplitude, $\sqrt{x}$ (similar to $\|x_1\|_\infty$ in previous section), versus $\Delta_\omega$ of the symmetric (a) and asymmetric (b) system for increasing values of $a_1$. Rectangle denotes plot range of Fig. 2.8.

Figure 2.8: The response amplitude, $\sqrt{x}$ (similar to $\|x_1\|_\infty$ in previous section), versus $\Delta_\omega$ for an asymmetric system ($\Delta_\alpha = 2$) near a branch-point bifurcation, inside the rectangle shown in Fig. 2.7b. Arrows indicate increasing $a_1$ ($a_1 \in [0.1, 1]$).

Explicit solution of Eq. (2.37) for $x$ results in the frequency-response curves shown in Fig. 2.7 for $\Delta_\alpha = 0$ and $\Delta_\alpha = 2$. As excitation grows in the symmetric system (Fig. 2.7a), the hardening behavior of the response amplitude is more curved when compared to the numerical results (see Fig. 2.2) near this resonance. As seen in Fig. 2.7b, the perturbation analysis also reproduces the additional peak observed in the numerical results near resonance. While not as dramatic, the addition of the second peak on the main resonance is verified. Figure 2.8 shows the prediction of the isola and branch-point bifurcations, similar to the continuation results (Fig. 2.5).
Due to the assumptions we have made in performing this perturbation analysis, the bifurcation is predicted for a lower forcing amplitude, \( a_1 \), than in the numerical analysis.

Numerical analysis of Eqs. (2.37-2.40) shows the existence of an isola bifurcation at \( \{ \Delta_\omega \approx 0.510442, a_1 \approx 0.127686 \} \) and a branch-point bifurcation at \( \{ \Delta_\omega \approx 0.491303, a_1 \approx 0.161899 \} \) when \( \Delta_\alpha = 2 \). These bifurcations disappear as the system becomes more symmetric. It should be noted that in order to exhibit these features, \( \Delta_\alpha = \epsilon \alpha_0 = 2 \) violates our assumption that the asymmetry is a very small perturbation from symmetry. We also violate the small excitation assumption in order to produce curves with excitation on the order of those shown with continuation. This oversight is common when predicting solutions using multiple scales, as an \( \mathcal{O}(1) \) excitation amplitude would make the zeroth-order solution unbounded.

**Large asymmetry**

We investigate next the dependence of the multiple-scales prediction on the assumption of small asymmetry. To this end, we repeat the perturbation analysis of Eq. (2.22), except that \( \alpha_0 \) will not be scaled by \( \epsilon \):

\[
\begin{align*}
\ddot{x}_1 + \epsilon \delta \dot{x}_1 - \epsilon \eta \dot{x}_2 + (\alpha_0 + \gamma) x_1 - \beta x_2 &= \epsilon \zeta (x_2 - x_1)^3 + \epsilon a_1 \cos(\omega t) \quad (2.41a) \\
\ddot{x}_2 - \epsilon \eta \dot{x}_1 + \epsilon \delta \dot{x}_2 - \beta x_1 + \gamma x_2 &= \epsilon \zeta (x_1 - x_2)^3 + \epsilon a_2 \cos(\omega t - \phi) \quad (2.41b)
\end{align*}
\]

When \( \epsilon = 0 \), we obtain the linear, homogeneous, conservative system:

\[
\begin{align*}
\ddot{x}_1 + (\alpha_0 + \gamma) x_1 - \beta x_2 &= 0 \\
\ddot{x}_2 - \beta x_1 + \gamma x_2 &= 0
\end{align*}
\]

(2.42)

with eigenfrequencies

\[
\omega_1 = \sqrt{\frac{1}{2} \left( \alpha_0 + 2\gamma - \sqrt{\alpha_0^2 + 4\beta^2} \right)}; \quad \omega_2 = \sqrt{\frac{1}{2} \left( \alpha_0 + 2\gamma + \sqrt{\alpha_0^2 + 4\beta^2} \right)}
\]

(2.43)

and corresponding mode shapes

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\[ \Psi_1 = \left( 1, \frac{1}{2\beta} \left( \alpha_0 + \sqrt{\alpha_0^2 + 4\beta^2} \right) \right)^T; \quad \Psi_2 = \left( 1, \frac{1}{2\beta} \left( \alpha_0 - \sqrt{\alpha_0^2 + 4\beta^2} \right) \right)^T \] (2.44)

We again consider excitation frequencies near the second eigenfrequency \( \omega_2 \).

The multiple-scales ansatz then yields the following equations for coefficients of \( \epsilon^0 \):

\[
\partial_0^2 x_{10} + (\alpha_0 + \gamma) x_{10} - \beta x_{20} = 0
\]
\[
\partial_0^2 x_{20} - \beta x_{10} + \gamma x_{20} = 0
\] (2.45)

and for \( \epsilon^1 \):

\[
\partial_0^2 x_{11} + (\alpha_0 + \gamma) x_{11} - \beta x_{21} = -2\partial_0 \partial_1 x_{10} - \delta \partial_0 x_{10} + \eta \partial_0 x_{20} - \zeta (x_{10} - x_{20})^3 + \frac{1}{2} a_1 (e^{i(\omega_2 T_0 + \sigma T_1)} + e^{-i(\omega_2 T_0 + \sigma T_1)})
\]
\[
\partial_0^2 x_{21} - \beta x_{11} + \gamma x_{21} = -2\partial_0 \partial_1 x_{20} + \eta \partial_0 x_{10} - \delta \partial_0 x_{20} + \zeta (x_{10} - x_{20})^3 + \frac{1}{2} a_2 (e^{i(\omega_2 T_0 + \sigma T_1 + \phi)} + e^{-i(\omega_2 T_0 + \sigma T_1 + \phi)})
\] (2.46)

We again consider solutions to Eq. (2.45) of the form in Eq. (2.29), where the frequency and mode shape are given in Eqs. (2.43) and (2.44). Let \( e_2 \) be defined such that \( \Phi_2 = (1, e_2)^T \), i.e., \( c_{20} = e_2 c_{10} \). The resulting magnitudes of \( x_{10} \) and \( x_{20} \) are no longer equal unless \( \alpha_0 = 0 \). Again, \( c_{10} \) will be determined by the need to eliminate secular terms from Eq. (2.46) after substitution of the zeroth-order solution into the right-hand side.

Eqs. 2.46 have solutions of the form given in Eq. (2.30). Following the remainder of the procedure for small \( \alpha_0 \) (cf. Eqs. (2.31) through (2.34)), we arrive at a similar expression relating the frequency detuning parameter \( \sigma \) to the response amplitude \( r \):

\[
r^2 \left( \frac{r^2 \mu_2}{\mu_3} + \sigma \right)^2 = \frac{16}{\mu_3^2} \left( a_1^2 \mu_1^2 + \mu_4^2 - r^2 \mu_5^2 + 2a_1 \mu_1 \mu_4 \cos(\phi) \right)
\] (2.47)

where
\[
\mu_1 = \alpha_0 + \sqrt{\alpha_0^2 + 4\beta^2}; \quad \mu_2 = 3\zeta(e_2 - 1)^3(\mu_1 + 2\beta); \quad \mu_3 = 8\omega_2(\mu_1 - 2\beta e_2)
\]

\[
\mu_4 = -2\beta a_2; \quad \mu_5 = \omega_2(2\beta(e_2 - \eta) + \mu_1(e_2 - \delta))
\]

(2.48)

Equation (2.47) reduces to Eq. (2.34) for the symmetric case \((\alpha_0 = 0)\). Multiplication by \(\epsilon^2\) and the substitutions

\[
r^2 \rightarrow x, \epsilon \sigma \rightarrow \Delta_\omega, \epsilon \zeta \rightarrow \zeta, \epsilon a_1 \rightarrow a_1, \epsilon a_2 \rightarrow a_2, \epsilon \delta \rightarrow \delta, \epsilon \eta \rightarrow \eta
\]

(2.49)

again result in an equation of the form in Eq. (2.37), where here

\[
f (x, \Delta_\omega, a_1) = \mu_2 x^3 + 2\mu_2 \mu_3 \Delta_\omega x^2 + (\mu_2^2 \Delta_\omega^2 + 16\mu_2^3) x
\]

\[-16\mu_2^2 a_1^2 - 64\beta^2 a_2^2 + 64\beta \mu_1 a_1 a_2 \cos (\phi)
\]

(2.50)

Recalling that, here, \(\alpha_0 = \gamma + \Delta_\alpha\) and \(\omega = \omega_2 + \Delta_\omega\), we may substitute the linear excitation conditions on \(a_2\) and \(\cos (\phi)\) given by Eq. (2.9) into this result. For the symmetric system \((\Delta_\alpha = 0)\), \(f (x, \Delta_\omega, a_1)\) reduces to the expression obtained in the small \(\alpha_0\) analysis above.

Fold bifurcations are again found where \(f (x_b(\Delta_\omega, a_1), \Delta_\omega, a_1) = 0\), provided that \(x_b(\Delta_\omega, a_1)\) corresponds to the vanishing of the partial derivative \(\partial f / \partial x\). The results in the presence of asymmetry differ slightly from those obtained in the case of \(\alpha_0 = \mathcal{O}(\epsilon)\), as seen in Fig. 2.9. For the parameters chosen, the coexistence of four fold bifurcations, which indicates existence of the isola bifurcation, lies in a much smaller window of forcing amplitudes, and disappears for larger values of \(\alpha_0\) relative to the previous analysis. As shown in Fig. 2.10a, the general frequency-response characteristics persist, however. Additionally, as seen in Fig. 2.10b, the isola bifurcation is predicted for values of \(a_1\) closer to the numerical continuation results than in the small-\(\alpha_0\) case. While this analysis does not produce a symmetric response, it does predict the general system behavior found in the numerical results, without making any assumptions on the relative scale of the symmetry fault.
Figure 2.9: The graph of $f(x_b(\Delta_\omega, a_1), \Delta_\omega, a_1)$ versus $\Delta_\omega$ of an asymmetric system ($\Delta_\alpha = 2$) for $a_1 = \{0.2, 0.245, 0.275\}$.

Figure 2.10: The response amplitude, $\sqrt{x}$ (similar to $\|x_1\|_\infty$ in previous section), versus $\Delta_\omega$ of an asymmetric system ($\Delta_\alpha = 2$) for increasing values of $a_1$. Rectangle in (a) denotes plot range of (b).

2.4 Optimization Method

2.4.1 Proposed control strategy

In the linear analysis, we used Eq. (2.9) to satisfy the first design objective, retaining a symmetric system response even in the presence of a symmetry fault. The second design objective was then to choose an optimal $\omega$ that achieves the desired response amplitude of $x_1$ with minimal excitation input, $a_1$. In the linear case, an explicit expression for the unique critical values for $\omega$ and $a_1$ were available in Eqs. (2.13-2.14). As seen in the previous section, in the case of broken symmetry, it is not...
possible to obtain conditions on the excitation that will ensure a perfectly symmetric response once nonlinearity is introduced. Instead, we chose to apply Eq. (2.9) as an approximate solution to the first design objective and to subsequently explore the symmetry properties and frequency-response relationship under variations in $\omega$ and $a_1$. The question now arises, whether a systematic and robust strategy may be deployed to find optimal values for $\omega$ and $a_1$ under these conditions, in order to still satisfy the second control objective.

In practice, one can easily monitor the response amplitude of a system, but the relationship between the response amplitude, excitation, and nonlinearity are usually unknown. In this case, a controller would most likely search for an optimal solution for a range of $\omega$ and $a_1$ values. Therefore, we propose that the system be designed to search for a locally-optimal excitation frequency that achieves the desired response amplitude with minimal excitation amplitude. The possibility of multiple, coexisting steady-state solutions for certain parameter ranges in the nonlinear system introduces the possibility of multiple solutions to the optimization problem as well.

Consider, e.g., the frequency-response characteristics shown in Fig. 2.2, for some initial excitation amplitude $a_1$. For each $a_1$, an optimal value of $\omega$ that achieves a maximal response amplitude then clearly corresponds to the peak of the frequency-response curve. Conversely, for a given response amplitude, the optimal choice of $\omega$ that would minimize $a_1$ corresponds to the peak of the frequency-response curve for that $a_1$. Suppose that such an optimal choice has been found for some response amplitude $\|x_1\|_{\infty, \text{des}}$ in the case of a symmetric system. Should a symmetry fault now occur, the frequency-response characteristics become those in Fig. 2.4a, where there may now exist multiple choices for locally-optimal values of $\omega$ and $a_1$. Suppose the controller we have designed picks the left peak of the prominent resonance. As this symmetry-breaking fault heals, we see that the peak we have chosen will disappear upon return to symmetry. Should we choose $\|x_1\|_{\infty, \text{des}}$ small enough, Fig. 2.3a sug-
gests this chosen peak will disappear at some point prior to the return to symmetry. Once this peak disappears, the controller would move to the original peak chosen for the symmetric system, causing a discontinuous change in excitation frequency and amplitude during a continuous fault correction.

### 2.4.2 Numerical continuation along optimal solution

We implement the proposed design strategy—i.e., searching for a locally-optimal excitation frequency that achieves the desired response amplitude with minimal excitation amplitude—in COCO. More specifically, we establish certain constraints on the continuation problem associated with periodic responses, in order for the continuation routine to remain on the locally optimal solution under variations in deviation from symmetry. In particular, we perform simultaneous continuation of two one-dimensional families of periodic orbits, with the additional condition that the excitation frequencies differ by $10^{-3}$. During continuation, we monitor $\|x_1\|_\infty$ and locate the parameter values where the difference in response amplitude of the two periodic orbits vanishes, corresponding to the approximate location of a peak in the frequency-response curve. We proceed to enforce the condition that the response amplitudes be equal and perform continuation under variations in the excitation amplitude $a_1$, while allowing $\omega$ to change, as necessary, until we locate a value of $a_1$ for which $\|x_1\|_\infty = \|x_1\|_{\infty,\text{des}}$. Finally, we constrain the solution by enforcing this equality on the response amplitude, and perform continuation under variations in the asymmetry $\alpha_0$ while allowing $\omega$ and $a_1$ to change, as necessary.

Let $\beta = 1$, $\gamma = 2$, $\delta = 0.2$, $\eta = 0.1$, and $\zeta = 0.05$. The value $\|x_1\|_{\infty,\text{des}} = 3.798$ then corresponds to the peak response amplitude obtained with $\alpha_0 = 0$, $a_1 = 1.5$, and $\omega = 2.685$. To achieve this response amplitude in the presence of a symmetry fault, consider the frequency response curve shown in Fig. 2.11 for the case when $\alpha_0 = 2$ and $a_1 = 1.5$. The left peak corresponds to the response amplitude $\|x_1\|_\infty = 4.649$.\[39\]
obtained at $\omega = 2.253$. We proceed to use this as the initial solution for a continuation in which we remain at a peak of the frequency-response curve while reducing the value of $a_1$ until $\|x_1\|_\infty = \|x_1\|_{\infty,\text{des}}$, as occurs for $a_1 = 0.9546$ and $\omega = 2.262$. We now constrain $a_1$ to maintain the peak at this amplitude, and continue in $\alpha_0$ to follow the solution branch back to the symmetric system ($\alpha_0 = 0$).

The results of this analysis are plotted in Fig. 2.12 as parameterized by the asymmetry $\alpha_0$, the excitation amplitude $a_1$, and excitation frequency $\omega$. We note here the existence of two geometric folds, corresponding to the occurrence of a saddle-node bifurcation in the frequency-response curve. In particular, as $\alpha_0 \to 0$, the chosen peak disappears in a collision with the local minimum along the frequency-response curve. The solution manifold then curves back toward increasing $\alpha_0$ following the local minimum (which also satisfies the nonlinear continuation problem). At the second fold point, the second peak collides with the local minimum. From this point, the solution manifold follows the right-most peak along the frequency-response curve until $\alpha_0 = 0$. We note, in particular, the existence of an interval in $\alpha_0$ over which the two peaks co-exist, as well as the absence of one or the other peak outside of this range.
The fold points along the solution manifold correspond to points of discontinuity in the excitation frequency and amplitude obtained from the locally-optimizing design strategy. Here, the disappearance of the left peak at the lower fold results in a discontinuous jump to a point along the portion of the solution manifold corresponding to the right peak, even during a process of continuous self-healing of the symmetry fault.

2.5 Conclusion

As stated in the introduction, the concern of this chapter is on the characteristics of corrective open-loop harmonic excitation schemes in response to symmetry faults and in the presence of small nonlinearities, during self-healing of such faults. The analysis demonstrates the existence of unique excitation conditions for a class of linear systems that ensure a response with particular symmetry properties and desirable response and excitation amplitudes, even in the case of a symmetry-breaking fault. In
the special case of the two-degree-of-freedom coupled oscillator, the analysis further
demonstrates the deterioration of performance that results with the introduction of
nonlinearity and the sources of this phenomenon.

It is clear that the techniques deployed here, viz., numerical continuation and
multiple-scales perturbation analysis, are restricted in their applicability to physically
more realistic systems. The analysis, nevertheless, points to some essential features of
an open-loop corrective strategy and their success, or failure, at retaining the desired
system response in the presence of unanticipated interactions with the environment,
here modeled by weak nonlinearities. For example, the discontinuous changes in
system excitation observed in the analysis resemble those suggested in some of the
rehabilitation literature cited in the introduction. For more complicated models, the
presence of nonlinearities could introduce coexisting solutions even in the symmetric
model, opening up for the possibility of the autonomous fault correction returning
to a different control strategy once a fault heals. On a related note, it might be
interesting to explore both the observed phenomenology and analysis used here for
other periodic excitations or nonlinearities. Similar analysis could be completed for
systems with periodic bang-bang forcing, for example (see Chapter 3), as well as
other forms of nonlinear coupling.

It is perhaps natural to ask of the importance of the issues raised here to a system
that includes closed-loop feedback control. In response, one notes the advantage,
and sometimes necessity, of open-loop corrective schemes due to cost and hardware
requirements. Faults may also occur in the feedback control, at which point open-loop
corrective schemes may be the last bastion against failure.
Chapter 3

Difficulties in Symmetry Compensation of Periodically Excited Oscillators and Their Implications for Human Gait

Abstract

In this study, the theory from the previous chapter is applied to a more general class of periodic excitations in order to determine the extensibility of the symmetry-compensation strategy used in that work. Bang-bang excitation is applied to the linear system of Chapter 2, approximated by Fourier series expansion in order to directly extend the previous theory. The analysis demonstrates that the frequency-dependent adjustments made to the original harmonic excitation break the shape of the more general bang-bang periodic excitation, making it impossible to retain symmetry with a global adjustment to amplitude and phase. Nevertheless, the analysis shows that simple adjustments to timing and amplitude commands may suffice when attempting to walk in the presence of injury.

3.1 Introduction

This chapter extends the theory presented in Chapter 2 to a more general class of periodic excitations. In the previous work, an open-loop control strategy was used to maintain symmetric output of simple mechanical system with natural structural symmetry in the face of asymmetry-inducing faults. The symmetry-compensation
strategy tuned the phase and amplitude of the single-frequency harmonic excitations of the system in order to regain symmetric oscillation in the presence of the faults. During the peer review process of that work, a question was posed as to whether the analysis could be extended to other excitations, such as periodic forcing. At the time, we had assumed that a similar analysis could be performed for any periodic forcing function, as there would be more free tuning parameters than in the single-harmonic case. The current study considers bang-bang forcing, as it is perhaps the most simplistic periodic excitation outside of the harmonic excitation considered in the previous chapter. More specifically, a zero-mean square-wave excitation is considered in order to maintain similarity with the phasing and amplitude relationships of the previous chapter’s forcing terms. The purpose of this work is to determine how the more general periodic excitation affects the symmetry-compensation strategy developed in the previous chapter, and to discuss the implications of its performance on human gait and motor control.

Similar to the previous chapter, this study is motivated by the biomechanics of compensation to injury. Human gait is a grossly symmetric activity, where clinicians focus rehabilitative efforts on regaining symmetry between limbs after injury [4]. Therefore, many researchers focus on deviations from symmetry as a means to qualitatively and quantitatively assess the effects of injuries and pathologies on gait [6–9]. While there is debate as to how symmetric unimpaired gait truly is, many of the studies lack statistical power, or a consistent definition and quantification of gait symmetry [13, 14]. Forczek and Staszkiewicz [15] assessed the gait symmetry of 54 able-bodied subjects, finding that while there were asymmetries in the kinematics of the ankle, the temporal and phasic variables of each limb were similar. This finding suggests that the underlying control of unimpaired human gait is to move the limbs symmetrically, while small disturbances during gait create small deviations from the intended action.
It is generally believed that the underlying control of repetitive movements is through spinal-level central pattern generators (CPGs), where Brown [31] first proposed the half-center oscillator concept for mammalian locomotion. His work suggested that movement resulted from two pools of pre-motor neurons that would excite synergistic motor neurons while inhibiting antagonistic motor neurons, creating the oscillatory flexion and extension necessary for movements like gait. Many researchers repeated similar experiments across multiple species, providing further evidence that these central pattern generators can successfully produce rhythmic output in mammals, and plausibly in humans as well [e.g., 32–34]. This concept has been supported by modern experiments and simulations [12, 37, 41–43, 45, 46], including focus on symmetry in CPG models [11] and motor pattern adaptability [47]. There is a consensus that the core periodic motions of the lower limbs are controlled by a set of CPGs through particular rhythm generating and pattern formation layers [44]. The rhythm generator indicates the timing and phasing of muscle firing, while the pattern formation layer provides the activation levels throughout the cycle. In addition to the CPG, there is an intricate network of feedback sensory systems and higher-level commands to adapt the motor control commands in the face of disturbances and to provide more stable gait [42]. Even though sensory feedback mechanisms play a prominent role in locomotion, it may be possible that, when faced with some physical asymmetry or injury, the CPG could shift in phase and amplitude to recover a symmetric gait after suitable motor adaptation or learning [10, 47]. This idea will be discussed later, in the context of the mathematical results of the following sections.

Below, we will review the pertinent results from Chapter 2 on symmetry compensation in a coupled mechanical system with single-frequency harmonic forcing. Next, the linear analysis will be extended to accommodate the more general periodic square-wave forcing. This extension shows that the system must satisfy certain properties (e.g., physical symmetry), or else break the square-wave form of excitation, in
order to maintain symmetric output. Then several methods to minimize asymmetry while maintaining square-wave excitation will be analyzed. While these methods do not fully eliminate asymmetry, it is found that simply using a lowest-order symmetry condition is nearly as effective as a numerical minimization routine. Finally, the results will be discussed in the context of the neural control of symmetry in gait via CPGs, including clinical evidence and implications of these behaviors.

3.2 A Review of the General Linear Theory

In the previous chapter, we demonstrated that the excitation of certain forms of linear systems could be designed to provide an invariant response under suitable symmetry operations. We now review the general linear framework and results pertinent to this chapter.

3.2.1 General linear theory

Consider a linear dynamical system of the form

\[ \dot{x} = A \cdot x + B \cdot u \]  \hspace{1cm} (3.1)

where \( x \in \mathbb{R}^n \), \( A \in \mathbb{R}^{n \times n} \),

\[ B := \begin{pmatrix} \begin{array}{c|c} b & b^* \end{array} \end{pmatrix} \] \hspace{1cm} (3.2)

where \( b \in \mathbb{C}^n \) and superscript * denotes complex conjugation, and

\[ u := \begin{pmatrix} e^{i \omega t} \\ e^{-i \omega t} \end{pmatrix} \] \hspace{1cm} (3.3)

A steady-state solution of Eq. (3.1) is given by

\[ x = \begin{pmatrix} \begin{array}{c|c} c & c^* \end{array} \end{pmatrix} \cdot u \] \hspace{1cm} (3.4)

where \( c \in \mathbb{C}^n \) satisfies the linear equation
\[ i\omega c - A \cdot c - b = 0 \]  
(3.5)

assuming the matrix \( i\omega I_n - A \) is invertible, where \( I_n \) is the \( n \times n \) identity matrix.

Let \( T \in \mathbb{R}^{n \times n} \) be an invertible matrix with a nonempty fixed space. Denote the eigenspace formed by \( T \)'s fixed space (eigenvalues equal to 1) as \( \mathcal{E}_1 \). We refer to a solution \( x \) of the linear system in Eq. (3.1) as symmetric if \( T \cdot x = x \), i.e., if \( x \in \mathcal{E}_1 \). Therefore, the steady-state solution in Eq. (3.4) is symmetric if and only if \( c \in \mathcal{E}_1 \).

From Eq. (3.5), this symmetry condition is satisfied by

\[
b \in (i\omega I_n - A) \cdot \mathcal{E}_1
\]  
(3.6)

The autonomous system \( (B = 0 \text{ in Eq. (3.1)}) \) is symmetric for \( A = A_0 \), if \( T^{-1} \cdot A_0 \cdot T = A_0 \) (i.e., equivariant under the symmetry transformation \( T \), see [101]). For this special case, Eq. (3.5) provides that the steady-state solution is symmetric if \( b \in \mathcal{E}_1 \).

### 3.2.2 Two-degree-of-freedom example

![Two-degree-of-freedom coupled mechanical oscillator](image)

Figure 3.1: A two-degree-of-freedom coupled mechanical oscillator used to illustrate symmetry-maintenance strategies for periodic excitation.

In Chapter 2, a special case of the above general system was considered. The two degree-of-freedom coupled oscillator shown in Fig. 3.1 is governed by the following equations of motion

\[
\begin{align*}
\ddot{x}_1 + \delta \dot{x}_1 - \eta \dot{x}_2 + \alpha x_1 - \beta x_2 &= F_1 \\
\ddot{x}_2 - \eta \dot{x}_1 + \delta \dot{x}_2 - \beta x_1 + \gamma x_2 &= F_2
\end{align*}
\]  
(3.7a) (3.7b)
where $\{\alpha, \beta, \gamma, \delta, \eta\} > 0$. Equation (3.7) takes the form of Eq. (3.1) if $x = (x_1, \dot{x}_1, x_2, \dot{x}_2)^T$, and if the excitation is harmonic. In the previous work, $F_1 = a_1 \cos(\omega t)$ and $F_2 = a_2 \cos(\omega t - \phi)$, with $\phi \in [0, 2\pi)$ and $\{a_1, a_2, \omega\} > 0$.

A transformation $T$ was then considered, given by

$$T = \begin{pmatrix}
0 & 0 & -1 & 0 \\
0 & 0 & 0 & -1 \\
-1 & 0 & 0 & 0 \\
0 & -1 & 0 & 0
\end{pmatrix} \quad (3.8)$$

which generated the reflection $x_1 \leftrightarrow -x_2$ such that

$$\mathcal{E}_1 = \text{span} \left\{ \begin{pmatrix} 0 \\ -1 \\ 0 \\ 1 \end{pmatrix}, \begin{pmatrix} -1 \\ 0 \\ 1 \\ 0 \end{pmatrix} \right\} \quad (3.9)$$

The system (3.7) is then symmetric with respect to $T$ provided that $\alpha = \gamma$, noting that $\alpha$ and $\gamma$ are rescalings of the stiffness terms $k_1$ and $k_2$ in Fig. 3.1, respectively. A symmetric steady-state response is achieved for the general system if and only if

$$a_2 e^{-i\phi} = -a_1 \frac{\beta + \gamma + i(\delta + \eta + i\omega)\omega}{\alpha + \beta + i(\delta + \eta + i\omega)\omega} \quad (3.10)$$

In particular, $a_2 = a_1$ and $\phi = \pi$ for the symmetric system.

It was explicitly shown in the previous chapter that a symmetric steady-state response may be achieved for any $\alpha \neq \gamma$, assuming the matrix $i\omega I_n - A$ is invertible, by choosing $a_2$ and $\phi$ to satisfy Eq. (3.10). Further, the remaining free excitation parameters ($a_1$ and $\omega$) were used to achieve a desired response amplitude of $x_1$ while minimizing excitation amplitude $a_1$, by suitable selection of the excitation frequency $\omega$. In other words, a physical symmetry fault to the linear system is always correctable by shaping the excitation forcing to satisfy Eq. (3.10).
3.3 Extension to Other Periodic Excitations

The above results from Chapter 2 considered simple single-frequency harmonic excitations on each mass. In the current chapter, we wish to explore how well those results extend to more general periodic forcing on the same class of systems. As a new example, square-wave excitations are considered. This class of periodic signals is desirable because the lowest-order Fourier approximation is exactly the single-frequency harmonic excitation reviewed above.

3.3.1 Defining the excitation signals

The question of how our coupled oscillator behaves when the single-frequency harmonic excitations are replaced with square-wave excitations must start with modeling the actual signals. We will consider the class of periodic, zero-mean, square-wave signals. First, we define the unit square wave of period $T_P$ as

$$y(t) := \begin{cases} 
1, & 0 \leq t < \frac{1}{2}T_P \\
-1, & \frac{1}{2}T_P \leq t < T_P
\end{cases}$$  \hspace{1cm} (3.11)

where $y(t \pm T_P) = y(t)$. Next, the forcing function on the first mass will simply be an amplitude scaling of the unit square wave:

$$F_1(t) = a_1 \cdot y(t)$$  \hspace{1cm} (3.12)

Similar to the previous study, the forcing on the second mass may change by amplitude and time shift\(^1\) from the first:

$$F_2(t) = a_2 \cdot y(t - \tau)$$  \hspace{1cm} (3.13)

where $0 \leq \tau < T_P$.

The Fourier series expansion of the unit square wave, in trigonometric and exponential form, is

\(^1\)Note the change from $\phi$ in the previous work to $\tau$ to indicate a time shift rather than phase shift.
Figure 3.2: Approximations to the unit square wave, \( y^{(m)} \), for \( m = 1 \) (dotted), 2 (dashed), and 6 (solid).

\[
\begin{align*}
y^{(m)}(t) &= \frac{4}{\pi} \sum_{k=1}^{m} \frac{\sin((2k-1)\omega t)}{(2k-1)} \\
&= \frac{2i}{\pi} \sum_{k=1}^{m} \frac{e^{-(2k-1)i\omega t} - e^{(2k-1)i\omega t}}{(2k-1)} 
\end{align*}
\]

where \( \omega = 2\pi/T_p \) and \( \lim_{m \to \infty} y^{(m)}(t) = y(t) \), except that \( y^{(m)}(nT_p/2) = 0 \) for \( n = 0, 1, 2, \ldots \). Similar to Eqs. (3.12) and (3.13), it follows that the Fourier series expansions of our forcing functions are

\[
\begin{align*}
F^{(m)}_1(t) &= a_1 \cdot y^{(m)}(t) \\
F^{(m)}_2(t) &= a_2 \cdot y^{(m)}(t - \tau)
\end{align*}
\]

In the sections below, it will be necessary to approximate \( F_i \) with these summations. Figure 3.2 shows several degrees of approximation of the unit square wave \( y(t) \) for \( T_p = 1 \). At the lowest mode, \( y^{(1)} \) is a single-frequency harmonic function. Using this approximation for \( F_i \), the system would behave in exactly the same manner as the example reviewed in Sec. 3.2.2.
3.3.2 Extending the general linear theory

The general linear theory of Sec. 3.2.1 can easily be extended to consider the Fourier approximations of square-wave forcing by replacing the term $B \cdot u$ in Eq. (3.1) with the appropriate summation:

\[
\dot{x}^{(m)} = A \cdot x^{(m)} + \sum_{k=1}^{m} B_k \cdot u_k \tag{3.16}
\]

where

\[
B_k := \begin{pmatrix} | & | \\ b_k & b_k^* \end{pmatrix} \tag{3.17}
\]

and

\[
u_k := \begin{pmatrix} e^{i\omega_k t} \\ e^{-i\omega_k t} \end{pmatrix} \tag{3.18}
\]

where $\omega_k = (2k - 1)\omega$. When $m = 1$, we are left with Eq. (3.1). The steady-state solution of Eq. (3.16) is then

\[
x^{(m)} = \sum_{k=1}^{m} C_k \cdot u_k \tag{3.19}
\]

where

\[
C_k := \begin{pmatrix} | & | \\ c_k & c_k^* \end{pmatrix} \tag{3.20}
\]

and $c_k \in \mathbb{C}^n$ satisfies the linear equation

\[
i\omega_k c_k - A \cdot c_k - b_k = 0 \tag{3.21}
\]

assuming the matrix $i\omega_k I_n - A$ is invertible, where $I_n$ is the $n \times n$ identity matrix.

We again consider a general transformation $T$ described in Sec. 3.2.1, with a fixed space $\mathcal{E}_1$. For the new excitation, our steady-state symmetry requirement that $x^{(m)} \in \mathcal{E}_1$ is satisfied if and only if each $c_k \in \mathcal{E}_1$. From Eq. (3.21), these conditions are satisfied by

\[
b_k \in (i\omega_k I_n - A) \cdot \mathcal{E}_1 \tag{3.22}
\]
Following the previous case, equivariance of the autonomous system provides that the steady-state solution is symmetric if \( b_k \in \mathcal{E}_1 \).

Considering the implementation of square-wave excitation into Eq. (3.16), we note that the vectors \( b_k \) have a particular relationship to each other dependent on Eq. (3.14). However, the conditions placed on \( b_k \) by Eq. (3.22) are dependent on \( \omega_k \) in general. In order to satisfy Eq. (3.22) while maintaining a square-wave form, \( b_k \) must not depend on \( \omega_k \). This requirement is necessary for any general non-harmonic wave form. It is possible to have \( b_k \) independent of \( \omega_k \) if and only if \( A \cdot \mathcal{E}_1 \in \mathcal{E}_1 \), in which case \( b_k \in \mathcal{E}_1 \) satisfies the symmetry conditions in Eq. (3.22). As we saw above, this condition is satisfied when \( A \) is equivariant, i.e., \( T^{-1} \cdot A_0 \cdot T = A_0 \) for some \( A = A_0 \). However, the general condition \( A \cdot \mathcal{E}_1 \in \mathcal{E}_1 \) may also hold for certain \( A \) that do not commute with \( T \).

As an example, consider the transformation \( T \) given in Eq (3.8), with \( \mathcal{E}_1 \) given in Eq. (3.9). The fixed space \( \mathcal{E}_1 \) is composed of any vector of the form \( v = (a, b, -a, -b)^T \). If \( A \cdot v \) also maintains this form for any \( a \) and \( b \), then \( b_k \in \mathcal{E}_1 \) satisfies symmetry on the solution, and we can maintain the square-wave excitation while satisfying this symmetry. Any real matrix of the form

\[
\hat{A} = \begin{pmatrix}
    a_1 & a_2 & a_3 & a_4 \\
    b_1 & b_2 & b_3 & b_4 \\
    c_1 & c_2 & a_1 - a_3 + c_1 & a_2 - a_4 + c_2 \\
    d_1 & d_2 & b_1 - b_3 + d_1 & b_2 - b_4 + d_2
\end{pmatrix}
\]

satisfies \( \hat{A} \cdot v \in \mathcal{E}_1 \) for this \( T \). However, \( \hat{A} \) is not generally equivariant with respect to \( T \), i.e., \( T^{-1} \hat{A} T = \tilde{A} \) where \( \tilde{A} \neq \hat{A} \) for certain \( \hat{A} \) satisfying Eq. (3.23).

### 3.3.3 Extending the example system

The example system in Sec. 3.2.2 can be extended to square-wave excitation by replacing \( F_i \) from Eq. (3.7) with \( F_i^{(m)} \) from Eq. (3.15). We again consider the transformation \( T \) given in Eq. (3.8), corresponding to \( x_1 \Rightarrow -x_2 \), with \( \mathcal{E}_1 \) given in Eq. (3.9). For
completeness, our example system gives

\[
A = \begin{pmatrix}
0 & 1 & 0 & 0 \\
-\alpha & -\delta & \beta & \eta \\
0 & 0 & 0 & 1 \\
\beta & \eta & -\gamma & -\delta
\end{pmatrix}
\]  \quad (3.24)

Considering the discussion above, we can only produce a symmetric steady-state solution with square-wave forcing if \( A \cdot \mathcal{E}_1 \in \mathcal{E}_1 \). This condition is only satisfied in our example system if \( \alpha = \gamma \), which in turn produces our equivariant system \( A_0 \) from the previous section. Therefore, outside of the symmetric system it is impossible to guarantee symmetry of the solution while maintaining a square-wave excitation.

Since we cannot maintain symmetry of the solution with respect to \( T \) with square-wave excitations, we can relax the conditions between \( b_k \) to see how the form of \( F_2 \) must change to actually maintain symmetry if \( A \cdot \mathcal{E}_1 \notin \mathcal{E}_1 \). Here, we will maintain use of the square-wave form for \( F_1 \), given in Eq. (3.15a). However, we must redefine the forcing on the second mass in order to satisfy the symmetry conditions of Eq. (3.22) on each Fourier mode. In particular, the amplitude and time shift of each mode must be independent of each other, as their solutions to satisfy Eq. (3.22) depend on \( \omega_k \).

To this end, let the following expression replace \( \hat{F}_2^{(m)} \) in our example system:

\[
\hat{\hat{F}}_2^{(m)} = 4 \sum_{k=1}^m \frac{a_{2,k} \sin \left( \omega_k (t - \tau_k) \right)}{(2k - 1)}
\]

\[
= 2i \sum_{k=1}^m \frac{a_{2,k} \left( e^{-i\omega_k (t - \tau_k)} - e^{i\omega_k (t - \tau_k)} \right)}{(2k - 1)}
\]  \quad (3.25)

The resulting system is given by Eq. (3.7) where \( F_1 \) and \( F_2 \) are given by \( F_1^{(m)} \) and \( \hat{\hat{F}}_2^{(m)} \) in Eqs. (3.15a) and (3.25), respectively.

Due to linearity, we can consider each Fourier mode separately. Doing so, the steady-state solution is symmetric with respect to the transformation \( T \) if and only if \( b_k \) from each mode satisfies Eq. (3.22). Each individual mode is essentially our original single-harmonic case with \( \omega = \omega_k \), and following Eq. (3.10), each \( b_k \) must
Figure 3.3: Numerical examples of $F^{(6)}_1$ (dashed) and $\hat{F}^{(6)}_2$ (solid) for an asymmetric system satisfying the symmetry conditions of Eq. (3.26).

satisfy

$$a_{2,k}e^{-i\omega_k \tau_k} = -a_1 \frac{\beta + \gamma + i (\delta + \eta + i \omega_k) \omega_k}{\alpha + \beta + i (\delta + \eta + i \omega_k) \omega_k} \tag{3.26}$$

to maintain symmetry of the steady-state response in Eq. (3.19). Recall that our square-wave forcing signals were defined in Eq. (3.15) such that the amplitudes $a_i$ were outside the summation, and each Fourier mode of $F^{(m)}_2$ was time-shifted by the same amount $\tau$. If we now require that Eq. (3.26) holds, the amplitude and time shift of each Fourier mode in $\hat{F}^{(m)}_2$ is dependent on $\omega_k$. This result will generally break the square-wave form of $F_2$, unless $\alpha = \gamma$.

As an example of what the forcing might look like, consider the asymmetric system described immediately above. Let $m = 6$, $\alpha = 2$, $\beta = 2$, $\gamma = 1$, $\delta = 0.1$, $\eta = 0.05$, $a_1 = 1$ and $\omega_1 = \sqrt{3}$. The system will oscillate symmetrically as long as $\hat{F}^{(6)}_2$ satisfies the symmetry conditions in Eq. (3.26) for each Fourier mode. Satisfying these conditions, the resulting excitation signals $F^{(6)}_1$ and $\hat{F}^{(6)}_2$ are shown in Fig. 3.3. Clearly, $\hat{F}^{(6)}_2$ does not resemble a square-wave approximation, but is instead a rather complex signal.
3.4 Minimizing Asymmetry with Square-Wave Excitation

The preceding section has proven that when our system exhibits physical asymmetry \( \alpha \neq \gamma \), it is impossible to maintain perfect symmetry in the solution, \( x \), without \( F_2 \) deviating from a square wave. It remains to be determined how effective the square-wave forcing is at minimizing the solution’s asymmetry. In order to do this, we must develop a framework for quantifying the symmetry of our solution.

Focusing only on square-wave excitations, we must return to using Eq. (3.15b) for \( F_2 \), i.e., \( a_2 \) and \( \tau \) do not depend on \( k \) in the summation. With this in mind, the symmetric steady-state solution can be found using Eqs. (3.4) and (3.5) with excitation given in Eq. (3.15):

\[
\begin{align*}
x_1^{(m)} &= \sum_{k=1}^{m} \Re \left[ \frac{-4ie^{i\omega_k t}}{(2k-1)\pi d} \left( a_1 (\gamma + i\omega_k(\delta + i\omega_k)) + a_2 e^{-i\omega_k \tau} (\beta + i\eta\omega_k) \right) \right] \\
x_2^{(m)} &= \sum_{k=1}^{m} \Re \left[ \frac{-4ie^{i\omega_k t}}{(2k-1)\pi d} \left( a_1 (\beta + i\eta\omega_k) + a_2 e^{-i\omega_k \tau} (\alpha + i\omega_k(\delta + i\omega_k)) \right) \right] \\
\end{align*}
\]

(3.27)

where

\[
d = -\beta^2 + \alpha\gamma + i ((\alpha + \gamma)\delta - 2\beta\eta) \omega_k - (\alpha + \gamma + \delta^2 - \eta^2) \omega_k^2 - 2i\delta\omega_k^3 + \omega_k^4
\]

(3.28)

Since we desire \( x_1 = -x_2 \) and vice-versa, we quantify the magnitude of deviation from this symmetric behavior as

\[
C_{\text{sym}} := \sqrt{\frac{\int_0^{2\pi/\omega} (x_1(t) + x_2(t))^2 \, dt}{\int_0^{2\pi/\omega} (x_1^2(t) + x_2^2(t)) \, dt}}
\]

(3.29)

\(^2\)Recall, this expression corresponds to a stiffness asymmetry between \( k_1 \) and \( k_3 \) (cf. Fig. 3.1)
3.4.1 Applying symmetry conditions from lowest Fourier mode

The lowest Fourier mode of Eq. (3.15) permits the symmetry condition given in Eq. (3.26), with \( k = 1 \). Once we add more terms to the summation to make \( F_i^{(m)} \) more closely-resemble square waves, this relationship fails unless we want a more complex forcing. Further, we showed above that perfect symmetry was not possible if \( a_2 \) and \( \tau \) were the same in each term. Here, we will begin by considering the symmetry conditions on \( a_2 \) and \( \tau \) from the lowest Fourier mode, as they at least produce perfect symmetry at the lowest order of our solution. The amount of asymmetry remaining in the system response will then be quantified by Eq. (3.29).

The expression for \( C_{\text{sym}} \) becomes quite complex as more terms are added to \( F_i^{(m)} \), requiring that we evaluate it numerically. We can take the solution given in Eq. (3.27) and substitute the symmetry conditions of the lowest mode, then numerically evaluate \( C_{\text{sym}} \) for \( \beta = 2, \gamma = 1, \delta = 0.1, \eta = 0.05, a_1 = 1, \omega = \sqrt{3}, m = 11 \) and \( \alpha = \{1, 2, 3\} \).

The integrations in Eq. (3.29) were evaluated using the \texttt{NIntegrate} routine within Mathematica (Mathematica 9; Wolfram Research, Champaign, IL). The resulting values of \( C_{\text{sym}} \), as well as the values of \( a_2 \) and \( \tau \) that satisfy the symmetry conditions of the lowest Fourier mode, are given in the left-hand-side of Table 3.1. As expected, these results are not able to perfectly maintain symmetry for \( \alpha \neq \gamma = 1 \). However, when visualizing the actual responses in Fig. 3.4a, and the deviation from symmetry in Fig. 3.4b, we see that these conditions do quite well at minimizing asymmetry while maintaining square-wave forcing.
Table 3.1: Quantification of asymmetry from Eq. (3.29) and resulting values of $a_2$ and $\tau$, when using symmetry conditions from the lowest Fourier mode and when numerically minimizing $C_{sym}$, for the symmetric ($\alpha = \gamma = 1$) and asymmetric system.

<table>
<thead>
<tr>
<th></th>
<th>Lowest Fourier mode symmetry</th>
<th>Minimized $C_{sym}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\alpha$</td>
<td>$C_{sym}$</td>
<td>$a_2$</td>
</tr>
<tr>
<td>1</td>
<td>$5.86 \times 10^{-16}$</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>$10.82 \times 10^{-3}$</td>
<td>0.2515</td>
</tr>
<tr>
<td>3</td>
<td>$19.73 \times 10^{-3}$</td>
<td>0.1288</td>
</tr>
</tbody>
</table>

Figure 3.4: System response for $\alpha = 3$, using symmetry conditions of the lowest Fourier mode on the forcing.

### 3.4.2 Numerically minimizing asymmetry

Even though the symmetry conditions of the lowest Fourier mode reduced the asymmetry between $x_1$ and $x_2$ in a reasonable manner, we are still left with $C_{sym} > 0$. It remains to be determined whether there are other values of $a_2$ and $\tau$ that might further reduce $C_{sym}$. To this end, we again take the solution given in Eq. (3.27), but this time we will not substitute symmetry conditions. Using the same parameter values listed above, we will numerically minimize $C_{sym}$ with respect to $a_2$ and $\tau$, via Mathematica’s `FindMinimum` routine. The minimized values of $C_{sym}$, as well as the resulting values of $a_2$ and $\tau$, are given in the right-hand-side of Table 3.1.

Interestingly, we see that minimizing the asymmetry based on Eq. (3.29) provides very little improvement to using the lowest-mode symmetry conditions. Throughout
Figure 3.5: Contour plot of $C_{\text{sym}}$ vs. $a_2$ and $\tau$ for $\alpha = 3$, from Eq. (3.29). Darker shades indicate lower values.

In the range of $\alpha$ values that were evaluated, $C_{\text{sym}}$ improves by less than $3 \times 10^{-5}$, with slightly larger corresponding changes to $a_2$ and $\tau$. The results in Table 3.1 suggest that if we insist on maintaining square-wave excitation for our system, it may be acceptable to minimize asymmetry between the masses by using the lowest-mode symmetry conditions rather than an optimization routine, assuming the amount of remaining asymmetry is acceptable. In other words, we can consider the forcing to be a single-frequency harmonic signal, and exactly repeat the results from Sec. 3.2 (or Chapter 2). A contour map of $C_{\text{sym}}$ for this example with $\alpha = 3$ is shown in Fig. 3.5, where darker shades indicate lower values. We can see that our results in Table 3.1 fall within the darkest contour level, ensuring we have found the correct minimum for this example.
3.5 Discussion

The previous two sections showed the behavior of a simple linear coupled oscillator with square-wave forcing during a fault that created physical asymmetry in the system. Repeating the general analysis of Chapter 2 proved that unless our system had a single-frequency harmonic excitation, symmetry of the response could not be maintained unless $A \cdot E_1 \in E_1$. For the example system, this relationship was only true for the symmetric case ($\alpha = \gamma$). Attempting to satisfy the symmetry conditions for each Fourier mode resulted in a very complex signal for $F_2$ that deviated greatly from a square-wave approximation (Fig. 3.3). However, simply using symmetry conditions of the first Fourier mode appeared to maintain a decent amount of symmetry in the response with square-wave forcing, at least for the chosen parameter values. Further, a local minimization showed that the first Fourier mode solution was almost equal to finding the lowest value of $C_{\text{sym}}$ with respect to phase and amplitude of $F_2$. However, the square waveform has relatively low amplitude contribution above the first Fourier mode, so other periodic waveforms may not share this finding.

3.5.1 Implications to human gait

The findings in this chapter can be abstractly tied to gait. First, consider the presumed nature of the central pattern generator (CPG) for human gait, introduced in Sec. 1.2.1. The core periodic motions of the lower limbs are controlled by a set of CPGs through particular rhythm generating and pattern formation layers [44]. The rhythm generator indicates the timing and phasing of muscle activations, while the pattern formation layer provides the activation levels, which relate to force amplitude. Both are believed to be controlled via a higher-level gait velocity command [42]. These periodic control signals are not single-frequency harmonic excitations like those needed to ensure symmetric oscillation in Chapter 2. Therefore, modulating
the timing and amplitude of the limbs through the CPG may not return symmetric motion in the face of a physical asymmetry caused by injury. Patients must then learn to cope with the additional asymmetry until sufficient healing occurs, which can lead to added stress on areas of the body away from the original injury [4]. For this reason, clinicians tend to focus rehabilitative efforts on regaining or maintaining symmetry between the lower limbs [6, 7, 14].

Based on the current knowledge of CPG functions reviewed above, it is likely that patients learning to regain symmetry in the face of a physical limitation must develop active strategies that complement the CPG [10, 42, 47]. This might lead to added stress away from the original injury, including overuse of the muscles requiring additional activation. It is also possible that a new CPG oscillator might be designed in the face of injury, where rather than simply adjusting the excitation amplitudes within the pattern formation layer, an entirely new, more complex pattern is created [47]. In the case of the latter two possibilities, the new active strategies or CPG patterns may remain after the injury is healed, perhaps for ever, or at least until de-adaptation can take place [10]. If true, this could potentially explain clinical results where control adaptations to injury remain after full recovery [e.g., 21, 28].

The effects of injury or perturbation on gait symmetry as they pertain to this chapter are seen widely in the clinical biomechanics literature [e.g., 15, 29, 30, 80, 88, 105]. To start, while there may be small asymmetries found even in the motions of healthy populations, the general timing and phase are very symmetric [15]. Healthy subjects experiencing an elastic force perturbation on their leg during swing exhibit increased thigh muscle activity in order to retain their normal motion patterns and symmetry [80]. Both results seem to exhibit the effectiveness and ability to tune the CPG timing and amplitude to maintain symmetry in a healthy system. When considering physical effects to the body, we see more evidence of the need for compensatory actions or motor adaptations to try and achieve symmetry. For example, patients even-
tually regained symmetric ground-reaction force patterns after recovery from ankle fracture, but asymmetry remained in their plantar pressure profiles [30]. In another study, patients recovering from total hip arthroplasty showed mildly improved step-length symmetry, but increased load-bearing in the affected limb [29]. Both of these results suggest an active compensation outside of the CPG in an attempt to maintain successful gait and minimize asymmetry. Finally, we consider the finding that gait is asymmetric for unilateral trans-tibial amputees, based on the physical restrictions imposed by the prosthetic device. However, the symmetry may be improved by careful design of the prosthetic ankle’s dynamics [105], indicating that the system’s physical asymmetry must be reduced to increase gait symmetry. A review of clinical gait results of unilateral trans-tibial amputees found that the primary compensatory mechanism during gait is increased work within the amputated limb’s hip joint [88]. This finding suggests evidence of a change in the CPG pattern intensity being used to approach symmetric gait, leaving the rest of the improvements to physical design.

3.5.2 Further thoughts

The focus of Chapter 2 was on the effects of a small nonlinearity on the linear symmetry conditions developed for the system. Briefly, explicit symmetry conditions were impossible to derive for the nonlinear system due to the presence of higher-order harmonics in the solution. The single-harmonic excitation produced these higher-order oscillations due to interactions with the cubic stiffness term introduced in the previous chapter. Unless the system was physically symmetric, the condition that $x_1 = -x_2$ could only be satisfied if $x_1$ was a single-harmonic oscillation, which was impossible given the cubic nonlinearity. These linear symmetry conditions used in Chapter 2 are what the current chapter uses as well. However, the current analysis stops with the linear system, as the results and their implications to gait are significant. It should be noted that perhaps the higher-order harmonics used above in approximating the
square-wave excitation, or those produced when satisfying the symmetry conditions for each order (Fig. 3.3), might actually help suppress the higher-order solutions to the nonlinear system. Alternatively, they may simply lead to even higher frequencies than those experienced in Chapter 2.

A numerical or theoretical analysis similar to those in the previous chapter may help answer the question of higher harmonics in the nonlinear case, but this is left for future work. Also left to future work is the effect of altering the dead zone, or the timing ratio between minimum and maximum, in the excitation signal. This would create a more general bang-bang type of forcing rather than a square wave, which would not allow such a simple reduction at lowest order to the linear work in Chapter 2. However, adding the additional degree of freedom to the symmetry compensation may prove beneficial. Addition of feedback terms to the control strategy would be interesting to explore as well. Feedback would most likely return symmetry to the asymmetric square-wave system, and might provide further insight into how the CPG interacts with sensory feedback mechanisms in the body. Finally, it may be interesting to investigate how the optimization results of Sec. 3.4.2 correlate with the nonlinear system of the previous chapter. Minimizing $C_{\text{sym}}$ with respect to the amplitude and phase of $F_2$ would most likely produce different results than simply using the linear symmetry conditions. This would complicate the existing analysis, as new minima must be found for the entire operating space of $F_1$.

In summary, this chapter was focused on extending the theory presented in Chapter 2 to a more general class of periodic excitations, determining how this excitation affected the symmetry-compensation strategy developed in the previous chapter, and discussing the implications of its performance on human gait and motor control. The extension to more general periodic forcing proved that unless the system satisfies certain properties with respect to the symmetry transformation, square-wave forcing cannot produce symmetric oscillations. However, for the numerical example chosen,
asymmetry can be minimized relatively well with square-wave forcing. Further, it was shown that using the lowest-order symmetry-compensation strategy minimized asymmetry almost identically to a local optimization routine, at least with square-wave excitation. Finally, clinical results as they pertain to CPG control of gait were discussed with the above modeling results in mind. It seems quite plausible that adjustments to CPG timing, phasing, and amplitude are the primary mechanisms for reducing asymmetry during prolonged injury or perturbation in gait.
Chapter 4

Compensations to Increased Ankle Stiffness During Gait

Abstract

This study is focused on producing and examining neuromuscular compensations to restrictions in ankle function during gait, via increased joint stiffness. This perturbation is created by an orthotic device to simulate the effects of injury in a controlled experimental environment with known conditions. It is expected that subjects will develop both kinematic and kinetic compensation strategies to maintain normal gait, and that overall limb-level behavior will be maintained at the expense of individual joint kinematics and kinetics. Ten subjects walked without, then with, the added ankle stiffness to assess their compensation strategies to the perturbation via lower-limb kinematics, kinetics, and muscle activation patterns. The results demonstrate that subjects successfully maintained whole-limb motion during the ankle perturbation, through a combination of adaptations of kinematic and kinetic strategies at the individual joints. These results support the hypothesis that a major goal of human gait is limb-level function, rather than the behaviors of individual joints, even during isolated injury to one joint.

4.1 Introduction

Chapters 2 and 3 focused on modeling and analysis of dynamical systems with key characteristics found in the control of gait. Specifically, compensations for loss of
symmetry were formulated and analyzed under the effects of nonlinearity and multi-harmonic periodic excitation. However, the simplicity of the chosen models left only an abstract connection to human gait, except for the similarity to the half-center oscillator model of central pattern generators [31] discussed in Chapter 3. The current chapter is concerned with discovering and analyzing compensations to injury during human gait, in an effort to extend some of the ideas postulated in the previous chapters to a clinical context. In particular, in order to directly compare the healthy and injured states, a systematic increase in unilateral ankle stiffness in a laboratory experiment was used to simulate the effects of an ankle injury. The resulting compensations were then analyzed and discussed in the context of neuromuscular controller goals.

Musculoskeletal injuries to the lower limb are studied in biomechanics because of their potential effects on such a basic and necessary task as locomotion in otherwise-healthy individuals [4]. The central nervous system (CNS) must balance intricate muscle commands with several key goals for successful bipedal locomotion: propulsion, weight support, and dynamic balance; while minimizing gross stride-to-stride variability to make gait a stable and quasi-periodic event. Emphasis is placed on the natural symmetry of human gait [6, 7, 9, 14], and on understanding the neuromuscular control adaptations employed by a person suffering an acute injury to maintain a gait close to a symmetric walking pattern [10]. Many studies have focused on how kinematics, kinetics, and/or muscle firing patterns change due to particular injuries [e.g., 17, 25, 27, 28, 106]. There is also evidence that normal gait kinematics can be retained even while the kinetic patterns (joint moments, muscle activations, etc.) are significantly different than normal [e.g., 21]. However, due to the nature of acute injuries, there is a lack of comparison to the true healthy state.

The human body has many more degrees of freedom than are necessary for a task such as gait, leading to the issue of motor redundancy [48]. It is widely believed
that the CNS attempts to successfully mitigate the motor redundancy problem by minimizing variance related to goal performance and ignoring variance that does not affect the overall outcome. This CNS strategy has been referred to separately as the uncontrolled manifold hypothesis [e.g., 49, 50] or the goal-equivalent manifold hypothesis [e.g., 51, 52]. Both hypotheses similarly assess the variance of certain motor tasks in terms of goal variance and body variance. Body variance that does not affect the goal lies in the uncontrolled manifold, and can be exploited for better task performance [53]. The exploitation of variance in the uncontrolled manifold also allows for more flexibility when overcoming an injury or limitation during the task.

Biomechanical and neurophysiological studies support the idea that an important goal for locomotion is limb-level function [69–75]. Simplified models of gait such as the inverted pendulum or spring-loaded inverted pendulum provide useful templates for modeling walking and running across many species with low complexity [70–72]. There is also neurophysiological and experimental evidence that cats maintain consistent whole-limb movement, even in the presence of impairment to a joint’s function [69]. Humans have been shown to maintain consistent whole-limb movement while experiencing perturbations during vertical hopping [73, 74]. Also, human lower-limb gait kinematics can be reduced to a two-dimensional space correlated with whole-limb length and orientation [75]. If one of the main kinematic goals of human gait is in fact whole-limb motion, this behavior should also drive CNS adaptations when compensating for injuries or pathologies.

CNS adaptations in the context of limb-level function during gait have been examined in terms of long-term and short-term variability for a cat peripheral nerve injury model [69]. The authors were motivated to examine individual joint behaviors in conjunction with the maintenance of whole-limb behavior. Long-term variability was quantified by the change in average kinematic behavior between conditions using linear regression. Short-term variability was then quantified by the amount of
cycle-to-cycle variance for each kinematic behavior, summed across the gait cycle. Changes to long-term variability would indicate that those angular kinematics were adapted to maintain successful gait during the perturbation. Changes to short-term variability would indicate that those angular kinematics were less consistent from cycle to cycle, and perhaps were less integral to the overall goals of gait. To support the idea that limb-level motion is an important goal for the CNS, long-term variability of the whole-limb behavior should be minimal, meaning the original motion would be conserved after perturbation or injury. Also, the whole-limb short-term variability should be lower than for the individual joints. Quantification of similar variability measures related to whole-limb motion could provide further evidence of limb-level control in human gait.

The underlying goal of this study was to explore how the CNS works to compensate for perturbations to ankle stiffness during gait. To address this goal, we focused on several research questions. First, how does a simulated injury affect gait-cycle timing and phasing? The treadmill should force a certain amount of symmetry and timing consistency, but we expect individual sub-phases of gait to change in timescale due to the perturbation. Second, what specific compensations are present in the kinematics, kinetics, and muscle activations? We expect that subjects will develop adaptations throughout both limbs in order to maintain normal gait kinematics. Finally, is limb-level behavior a primary focus of gait, and does it remain so during simulated injury? We expect subjects will maintain consistent whole-limb kinematics at the expense of individual joint angle kinematics, both in terms of long-term and short-term variability. These questions were evaluated by simulating an injury to the ankle using a custom ankle-foot orthosis (AFO) with adjustable bidirectional resistance to ankle motion. The ankle was chosen due to its importance in gait propulsion [84], and the AFO provided a controlled experimental environment for simulating abnormal gait with known conditions [cf. 8, 107].
4.2 Methods

4.2.1 Experimental design

Figure 4.1: The custom ankle-foot orthosis (AFO) outfitted with a linear pneumatic cylinder. Valves were closed after known pressure was added, to create an air spring. A bidirectional load cell measured the force produced by the air spring.

Ten able-bodied male subjects, age 25.5 ± 5.1 years, mass 77.4 ± 11.4 kg, and height 1.77 ± 0.05 m (mean ± SD), participated in the study after giving written informed consent. The protocol was approved by the University of Illinois at Urbana-Champaign Institutional Review Board.

A custom AFO was fabricated for the left lower limb (Fig. 4.1). The device was large enough to allow varying degrees of padding, providing a custom fit for each subject. The AFO consisted of carbon fiber shank and foot pieces, connected by bilateral low-friction joints. The sole of the foot piece was constructed of high-density foam and shaped to create a natural toe-rocker motion. Aluminum brackets were fabricated to allow attachment of a linear pneumatic cylinder (DSNU-5/8”-5”-P; Festo Inc., Hauppauge, NY) on the posterior side of the AFO. This cylinder was
used as a passive restraint to ankle motion by filling both ends with a known air pressure and closing the ports manually with flow-control valves (GR-QB-5/32-U; Festo Inc.). The force produced by the cylinder was measured with a bi-axial load cell (LC202-100; OMEGA Engineering Inc., Stamford, CT). The AFO with all added components weighed 1.44 kg. Some conditions described below required removal of the cylinder and load cell, reducing the weight to 1.21 kg.

Subjects were tested on two consecutive days. On the first day, after protocol review and informed consent, the subject was fitted with the AFO to ensure minimal ankle motion relative to the AFO joint. An eleventh subject (not included) experienced noticeably affected gait while wearing the AFO without resistance, and was excluded from participation. Next, surface electromyography (EMG) sensors were placed over the tibialis anterior (TA), soleus (SOL), vastus lateralis (VL), and hamstrings (HAM) of each leg (Bagnoli-16; Delsys Inc., Natick, MA). Locations were shaved, cleaned, and marked for the next day. The subject was then asked to walk on an instrumented split-belt treadmill (Instrumented Treadmill; Bertec Corp., Columbus, OH) while wearing the AFO without the cylinder. After identifying a comfortable walking speed, the subject was asked to continue walking for a total of 30 minutes to acclimate to the free-moving AFO. On both days, live video of the subject’s feet were projected on a screen in front of him to aid in minimizing treadmill belt crossover without looking down unnaturally.

On the second day, EMG sensors were reattached and signal quality was reconfirmed. Reflective markers were prepared for three-dimensional motion capture via a passive five-camera infrared system (460 Datastation; Vicon, Oxford, UK). The reflective markers were bilaterally attached over the anterior-superior iliac spine, greater trochanter of the femur, lateral mid-thigh, lateral epicondyle of the femur, tibial tuberosity, lateral malleolus, heel, and the first and fifth metatarsal heads; as well as the L5-S1 sacral junction. Permanent markers were also attached to the treadmill
and to the AFO at the medial and lateral ankle joint, and the lateral surfaces of the two aluminum brackets. While wearing the AFO, markers were removed from the subject’s lateral malleolus and heel. These two positions were approximated using the markers on the AFO, offset with static corrections relative to an embedded AFO coordinate system. Accuracy of these approximations was high since the AFO is essentially two rigid bodies.

The following assessments were then performed in order for each subject:

**base** To capture his baseline walking behavior, the subject walked on the treadmill in his running shoes for seven minutes, then rested for 10 minutes.

**NC** While wearing the AFO with zero resistance (no cylinder) on the left foot and running shoe on the right, the subject walked for seven minutes to re-acclimate to the AFO, then rested for 10 minutes.

**p100** The cylinder was attached and the AFO was adjusted to the “full” perturbation\(^1\), where the cylinder was charged with 100 psig of air. The subject walked for 15 minutes then rested for 15 minutes.

**p60** The AFO was adjusted to 60 psig, and the subject walked for seven minutes, then rested for 10 minutes. It was anticipated that the subject would require less time to reach steady state walking patterns since the resistance change is much lower than in **p100**, and moving toward normal.

**p20** Condition **p60** was repeated at 20 psig for seven minutes.

**NC2** Condition **NC** was repeated for 15 minutes.

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\(^1\)Note that *full perturbation* refers to the maximum cylinder pressure used, and not a full restriction of ankle motion.
4.2.2 Data collection and analysis

During each of the assessments described above, three-dimensional motion-capture marker positions, ground-reaction forces and moments on each treadmill belt, EMG signals, and axial force of the AFO cylinder (for p100 through p20) were recorded for 30-second trials at each minute. Marker positions were recorded by the Vicon workstation at 100 Hz, as well as the analog signals from the treadmill at 1000 Hz. The EMG signals were recorded at 1000 Hz using a custom MATLAB function (R2012b; The MathWorks Inc., Natick, MA) integrated with a digital-acquisition board (NI-USB-6251; National Instruments, Austin, TX). A separate digital-acquisition board (NI-USB-6210) was used for the load cell, also recorded in MATLAB at 1000 Hz. To study steady-state compensation, all analyses were focused on the final 30 seconds of each condition listed above. Further, this chapter is focused on describing the effects of the stiffness, so only data from conditions NC and p100 are compared here. The following chapter will consider the changes to these compensations throughout “recovery,” or progression from p100 through NC2.

All recorded data were then processed in MATLAB using custom code. Sagittal-plane kinematics (joint angles) and kinetics (net joint moments) of the hip, knee, and ankle joints were computed using inverse kinematics and dynamics of motion and ground-reaction force data. Limb-level function was characterized by the “whole-limb” angle, or the global angle of the toe-to-hip vector from the anterior direction (Fig. 4.2). The contribution from the AFO cylinder was accounted for by calculating the torque produced by the cylinder force about the AFO joint. Specifically, the cross-product was taken between the vector from the AFO joint axis to the cylinder axis, and the cylinder force vector. The dot product between this torque and the AFO joint axis was taken to obtain the torque acting directly about the AFO ankle joint. It was assumed that the sagittal-plane axes of rotation of the biological ankle
and AFO joint were aligned. EMG signal means were zeroed, followed by band-pass filtering (20-250 Hz) using a fourth-order recursive Butterworth filter. The data were then rectified and low-pass filtered (7 Hz, 4th order recursive Butterworth) to produce linear envelopes of the muscle activation signals.

**Gait events**

The following gait events were found within each gait cycle for both limbs, based on Perry [4] and Morris [108], and converted to 0 – 100% of the gait cycle (cf. Fig. 4.3, Sec. 1.1.1):

**IHS1** “Ipsilateral heel strike,” when the ipsilateral limb strikes the ground; signifying the beginning of the current gait cycle for the ipsilateral limb in stance, and the beginning of the loading response phase.

**CTO** “Contralateral toe off,” when the contralateral limb lifts off the ground; signifying the transition from loading response to mid-stance.

**WA** “Weight alignment,” when the center of the pelvis is over the ipsilateral toes; signifying the transition from mid-stance to terminal stance.
Figure 4.3: The eight gait events described in the text separate seven sub-phases of gait during stance: loading response (LR), mid-stance (MS), terminal stance (TS), pre-swing (PS); and swing: initial swing (ISw), mid-swing (MSw), and terminal swing (TSw). Adapted from [108].

CHS “Contralateral heel strike,” when the contralateral limb strikes the ground; signifying the transition from terminal stance to pre-swing.

ITO “Ipsilateral toe off,” when the ipsilateral limb lifts off the ground; signifying the transition from pre-swing to initial swing.

TAA “Toe-ankle alignment,” when the ipsilateral toes align with the contralateral ankle; signifying the transition from initial swing to mid-swing.

KAA “Knee-ankle alignment,” when the ipsilateral knee and ankle are aligned; signifying the transition from mid-swing to terminal swing.

IHS2 “Ipsilateral heel strike,” signifying the end of terminal swing, and the beginning of the next gait cycle.

Additionally, two main phases compose the gait cycle: the limb is in stance between IHS1 and ITO, and in swing between ITO and IHS2. Piecewise linear length normalization (PLLN) was used to align these events and create constant sampling from 0 – 100% of the period for all gait cycles, to facilitate averaging and remove
time variance effects from the amplitudes of the averaged curves. For more details, and a discussion on the benefits of PLLN, see [5, 109]. Original event data were used to assess timing effects of the ankle perturbation using paired $t$ tests between the NC and p100 conditions ($\alpha = 0.05$) across the group (SPSS 20; IBM Corp., Armonk, NY). IHS1 and IHS2 were excluded since they are always at 0% and 100% of the gait cycle, respectively. In order to capture changes to the gait cycle as a whole, the original stride time (in seconds) from IHS1 to IHS2 was also statistically compared.

**Regions of deviation**

As a way to pinpoint areas of the gait cycle that were significantly affected between all subjects, the regions of deviation method was utilized. Regions of deviation have previously been used in gait to determine the effects of artificially-increased joint stiffness of healthy males [8], as well as dogs suffering from ligament deficiencies [9]. This method compares each subject’s behavior against normative behavior. Here, a subject’s average behavior in p100 was compared against his normative data in NC. We previously performed regions of deviation analysis on symmetry data [9], but here we will briefly review the methodology for data associated with a single signal (i.e., angle data from one limb rather than the difference between limbs). Let $x = \{x_i\}_{i=0}^{100}$ and $y = \{y_i\}_{i=0}^{100}$ be sequences of averaged data for one full gait cycle, warped to align gait events using PLLN. Each of the 101 data points correspond to the $i^{th}$ percentage of the cycle, averaged across all cycles from one trial. Here, $x$ denotes data from NC and $y$ from p100. Finally, let $\sigma^{(x)} = \{\sigma^{(x)}_i\}_{i=0}^{100}$ denote the sequence of standard deviations in NC, across all gait cycles for one trial. Then the standard deviation at the $i^{th}$ percentage of the cycle, $\sigma_i^{(x)}$, corresponds to the mean value $x_i$. For each data point $i$ of a given signal in p100 from a particular subject, the deviation from normative behavior [8] is defined as
For a statistical comparison throughout the entire gait cycle, the group’s deviations in p100, $D_{i}^{(y)}$, were compared against zero with a one-sample $t$ test at every point $i$ in the gait cycle ($\alpha = 0.05$). All points proving significantly different were then considered to fall within a region of deviation from the NC condition for the given signal.

Regions of deviation were evaluated for the kinematics and kinetics described above in order to determine significant changes throughout the cycle and provide a clear understanding of the limitations and compensation mechanisms created by the perturbation. During p100, the net left ankle moment is a combination of the biologically-produced muscle moment and the AFO torque. Therefore, the biological left ankle moment was computed as the difference between the net moment and the AFO torque.

**Muscle activations**

The underlying muscle activity was assessed via the EMG linear envelope data. To quantify the activations, the peak value and corresponding percentage of gait cycle were found for the major repeatable bursts of muscle activity in the linear envelope data after aligning with PLLN. The activation peaks were normalized by the average of the maximum peak value during NC for each subject and signal. These peak amplitudes and timings were then compared between NC and p100 using paired two-tailed $t$ tests ($\alpha = 0.05$) for each limb. Significant changes can give a more direct insight to the neuromuscular control system’s commands to compensate for the ankle stiffness. Due to significant noise, only nine of the subjects were included in the EMG
analyses, except for the following muscles: left HAM (two excluded), right VL (three excluded), and right HAM (two excluded).

Movement variance

The subject’s ability to maintain the overall goal of whole-limb motion was tested using the ideas of long-term and short-term variability introduced above.

**Long-term variance** To measure each subject’s overall change in kinematics from NC to p100, the similarity of average steady-state trajectories between conditions was quantified using a normalized cross correlation of zero phase shift [110, 111], given by

$$r_{\{x,y\}} = \frac{\sum_{i=0}^{100} (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=0}^{100} (x_i - \bar{x})^2} \sqrt{\sum_{i=0}^{100} (y_i - \bar{y})^2}} \times 100 \quad (4.2)$$

where $x = \{x_i\}_{i=0}^{100}$ and $y = \{y_i\}_{i=0}^{100}$ are sequences of data for one full gait cycle, previously described for Eq. (4.1). Again, $x$ denotes data from NC and $y$ from p100, and the sequence averages are denoted $\bar{x}$ and $\bar{y}$. The resulting value of $r_{\{x,y\}}$ ranges between 100 and $-100$, corresponding to perfect positive or negative alignment, respectively.

The correlations between NC and p100 were computed for the individual joint angles (hip, knee, ankle), as well as the whole-limb angle (cf. Fig. 4.2), for each subject. Finally, consider a “self-correlation” of the NC condition$^2$, $r_{\{x^{(o)},x^{(e)}\}}$ computed using Eq. (4.2), where $x^{(o)}$ and $x^{(e)}$ are average cycles from the odd and even gait cycles in NC, respectively. This correlation quantifies the baseline similarity for each signal.

To test whether data from p100 were significantly different than NC, $r_{\{x,y\}}$ for each signal were compared against the self-correlation threshold, $r^*_{\{x^{(o)},x^{(e)}\}}$, defined as the lowest group mean value of $r_{\{x^{(o)},x^{(e)}\}}$ across all signals. For this study, the right ankle exhibited the lowest mean self-correlation across the group, resulting in

$^2$Note that the terminology “auto-correlation” is avoided due to its specific definition of the cross-correlation of one signal onto itself.
$r^*_{\{x(o),x(e)\}} = 99.82$. Paired one-tailed $t$ tests ($\alpha = 0.05$) were used to test the null hypothesis that $r_{x,y} \geq r^*_{\{x(o),x(e)\}}$ for each signal in $p100$. Rejection of the null hypothesis would indicate the presence of long-term variability between conditions for that signal. While the analysis of long-term variance was motivated by Chang et al. [69], it should be noted that the quantification was performed in a different manner.

**Short-term variance** To measure each subject’s short-term compensations, the cycle-to-cycle variance of these data were quantified for each subject via a cumulative sum of standard deviations across the gait cycle, similar to [69]:

$$\sigma_{\text{tot}} = \sum_{i=0}^{100} \sigma_i \quad (4.3)$$

where $\sigma_i$ is the standard deviation of a signal at the $i^{\text{th}}$ percentage of the cycle, previously described for Eq. (4.1). This metric was computed for the individual joint angles as well as the whole-limb angles of both conditions, for each subject. To assess condition effects on short-term variability, paired $t$ tests ($\alpha = 0.05$) were performed between NC and $p100$ for each signal of a given limb. To test differences between whole-limb angle variance and individual joint angle variances, repeated-measures ANOVAs were performed *within* each condition ($\alpha = 0.05$) for all signals of a given limb. In cases where a condition showed significance, post-hoc testing determined specific differences between the whole-limb angle and each joint angle via paired $t$ tests ($\alpha = 0.05$).
4.3 Results

Many of the following results are compared pairwise between the unconstrained (NC) and constrained (p100) conditions since the data were repeated by the same subjects. In these cases, the group averages of the differences between the two conditions are also presented (Δ) to get a better sense of the true changes between NC and p100 across the group. To briefly summarize all of the results presented below, this study has looked at gait events normalized to percent gait cycle (%GC), regions of deviation of joint kinematics and kinetics, muscle activation peaks, and movement variance (both long-term and short-term) of joint and whole-limb angles.

4.3.1 Gait events

The increased ankle stiffness during p100 caused several significant timing effects compared to NC (Tables 4.1 and 4.2). Both limbs experienced similar significant increases in stride time (Δ = 0.03 ± 0.02 sec, p = 0.001). Bilateral stride times were identical, most likely coupled by the treadmill speed. In p100 compared to NC, the left limb experienced delayed weight alignment (WA) (Δ = 1.1 ± 0.8%GC, p = 0.002), and delayed knee-ankle alignment (KAA) (Δ = 0.8 ± 0.7%GC, p = 0.005). In p100 compared to NC, the right limb experienced earlier contralateral toe-off (CTO) (Δ = −1.1 ± 0.4%GC, p < 0.001), delayed toe-ankle alignment (TAA) (Δ = 0.9 ± 0.5%GC, p = 0.001), and delayed KAA (Δ = 0.6 ± 0.6%GC, p = 0.019).
Table 4.1: Group averages (±SD) of total stride time for the unconstrained (NC) and constrained (p100) conditions, and their difference (∆). Condition effects were assessed with paired *t* tests, where * denotes statistical significance (α = 0.05). AFO worn on left limb, running shoe on right.

<table>
<thead>
<tr>
<th></th>
<th>Left Stride Time (sec)</th>
<th>Right Stride Time (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NC</td>
<td>1.19 ± 0.06</td>
<td>1.19 ± 0.06</td>
</tr>
<tr>
<td>p100</td>
<td>1.22 ± 0.06</td>
<td>1.22 ± 0.06</td>
</tr>
<tr>
<td>∆</td>
<td>0.03 ± 0.02*</td>
<td>0.03 ± 0.02*</td>
</tr>
</tbody>
</table>

Table 4.2: Group averages (±SD) of the gait events described in Sec. 4.2.2 for the NC and p100 conditions, and their difference (∆). Events are expressed in terms of percentage of the full gait cycle. Condition effects were assessed with paired *t* tests, where * denotes statistical significance (α = 0.05). Note that IHS1 and IHS2 are not displayed since they are 0% and 100% by definition.

<table>
<thead>
<tr>
<th></th>
<th>CTO (%GC)</th>
<th>WA (%GC)</th>
<th>CHS (%GC)</th>
<th>ITO (%GC)</th>
<th>TAA (%GC)</th>
<th>KAA (%GC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>15.0 ± 1.0</td>
<td>39.9 ± 1.3</td>
<td>49.5 ± 0.9</td>
<td>64.4 ± 0.9</td>
<td>78.1 ± 1.0</td>
<td>90.2 ± 1.5</td>
</tr>
<tr>
<td>p100</td>
<td>15.3 ± 1.4</td>
<td>41.0 ± 1.7</td>
<td>49.9 ± 1.2</td>
<td>63.8 ± 1.5</td>
<td>78.6 ± 1.3</td>
<td>91.0 ± 1.5</td>
</tr>
<tr>
<td>∆</td>
<td>0.4 ± 1.2</td>
<td>1.1 ± 0.8*</td>
<td>0.5 ± 1.0</td>
<td>−0.6 ± 1.0</td>
<td>0.5 ± 0.7</td>
<td>0.8 ± 0.7*</td>
</tr>
<tr>
<td>Right:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>14.9 ± 1.0</td>
<td>40.5 ± 1.7</td>
<td>50.5 ± 0.9</td>
<td>65.5 ± 1.1</td>
<td>78.6 ± 0.9</td>
<td>90.1 ± 1.1</td>
</tr>
<tr>
<td>p100</td>
<td>13.8 ± 1.0</td>
<td>40.7 ± 1.3</td>
<td>50.1 ± 1.2</td>
<td>65.4 ± 1.1</td>
<td>79.5 ± 0.9</td>
<td>90.7 ± 0.8</td>
</tr>
<tr>
<td>∆</td>
<td>−1.1 ± 0.4*</td>
<td>0.2 ± 0.9</td>
<td>−0.4 ± 1.0</td>
<td>−0.1 ± 0.7</td>
<td>0.9 ± 0.5*</td>
<td>0.6 ± 0.6*</td>
</tr>
</tbody>
</table>
4.3.2 Joint kinematics and kinetics

Only slight differences are noticeable between conditions for the group average behaviors of the gait kinematics and kinetics (Figs. 4.4–4.7). Here, the data from NC are displayed in black (with dashed lines representing one standard deviation), while data from p100 are in red. Whole-limb angles were largely unchanged between conditions (Fig. 4.4). Further, their minimal group standard deviations throughout the cycle relative to the individual joint kinematics suggest this measure was more repeatable across subjects than others. The largest visible differences in joint kinematics were understandably in the left ankle (Fig. 4.5e). The joint kinetics computed via inverse dynamics then provide further information as to the control effort necessary to overcome the left ankle perturbation (Fig. 4.6). Note that during p100, the net left ankle moment (Fig. 4.6e, red curve) includes contribution from both the ankle-foot orthosis (AFO) torque produced by the cylinder (Fig. 4.7a), as well as the biological net muscle moment at the left ankle (Fig. 4.7b, red curve). Torque produced by the AFO is assumed to be negligible during NC.

Figure 4.4: Group averages of whole-limb angles for NC (black) ±1SD (dashed), and p100 (red). AFO worn on left limb, running shoe on right.
Figure 4.5: Group averages of joint angles for NC (black) ±1SD (dashed), and p100 (red). Positive values indicate joint flexion.
Figure 4.6: Group averages of joint net muscle moments for NC (black) ±1SD (dashed), and p100 (red). Positive values indicate extensor moments.
Figure 4.7: (a): Group average of measured AFO torque for p100 (solid) ±1SD (dashed). (b): Group average of resulting biological ankle moment (net minus AFO) for p100 (red), with corresponding normative data from NC (black) ±1SD (dashed). Positive values indicate extensor moments.

### 4.3.3 Regions of deviation

The group average behaviors for joint angle deviation values, $D$ from Eq. (4.1), along with the significant regions of deviation (shaded, Fig. 4.8), provide insight into the specific locations of altered kinematics throughout the gait cycle due to motion restriction of the left ankle. The shading indicates significant deviations in left hip movement patterns during loading response, pre-swing through initial swing, and terminal swing across the group (Fig. 4.8a). The right hip experienced a significant deviation during late loading response and early mid-stance (Fig. 4.8b). The left knee had significant deviations in a short portion of the loading response, the end of terminal stance through early initial swing, and terminal swing (Fig. 4.8c). Finally, the left ankle movement deviated during loading response, mid-stance (though not necessarily clinically significant due to lower magnitude of difference), and pre-swing through the entire swing phase (Fig. 4.8e).
Figure 4.8: Group averages of joint angle deviations ($D$) from each subject’s NC normative window for p100. Significant regions of deviation (shaded) were found with a one-sample $t$ test against zero ($\alpha = 0.05$) at each percent of the gait cycle.
The group average behaviors for deviation values for joint moment data, along with the significant regions of deviation (shaded, Figs. 4.9 and 4.10), provide further insight into the instances throughout the gait cycle where the subjects’ control strategies changed to compensate for the increased ankle stiffness. The left hip moment experienced significant deviations during pre-swing and intermittently through initial swing (Fig. 4.9a), while the right changed during late loading response and early mid-swing (Fig. 4.9b). The left knee moment was affected during mid-stance (Fig. 4.9c), where the right was mainly affected during late terminal stance and pre-swing (Fig. 4.9d). The left ankle was affected during late loading response through mid-stance, and intermittently throughout swing, though not necessarily clinically significant due to lower magnitude of difference (Fig. 4.9e). The right ankle was also affected during late loading response and terminal swing (Fig. 4.9f). The biological ankle torque was affected during loading response, late mid-stance through early pre-swing, and the entire swing phase (Fig. 4.10).
Figure 4.9: Group averages of net joint moment deviations ($D$) from each subject’s NC normative window for $p100$. Significant regions of deviation (shaded) were found with a one-sample $t$ test against zero ($\alpha = 0.05$) at each percent of the gait cycle.
Figure 4.10: Group average of left biological ankle moment deviations ($D$) from each subject’s NC normative window for p100. Significant regions of deviation (shaded) were found with a one-sample $t$ test against zero ($\alpha = 0.05$) at each percent of the gait cycle.

4.3.4 Muscle activations

The linear envelope peaks common between subjects give a more direct insight to the neuromuscular control system’s commands to compensate for the ankle stiffness (Tables 4.3 and 4.4, Fig. 4.11). In p100 compared to NC, the first left tibialis anterior (TA) peak decreased in amplitude ($\Delta = -18.7 \pm 13.5\%_{NC}$, $p = 0.004$, Table 4.3), and occurred later in the gait cycle ($\Delta = 1.3 \pm 1.2\%_{GC}$, $p = 0.019$, Table 4.4). The left vastus lateralis (VL) peak amplitude increased ($\Delta = 19.8 \pm 19.5\%_{NC}$, $p = 0.021$, Table 4.3), while the left hamstrings (HAM) peak occurred later in the gait cycle ($\Delta = 1.7 \pm 1.6\%_{GC}$, $p < 0.001$, Table 4.4). There were no significant changes to the right limb’s EMG activation peaks.
Table 4.3: Group averages (±SD) of normalized electromyography (EMG) amplitudes for tibialis anterior (TA, two peaks), soleus (SOL), vastus lateralis (VL) and hamstrings (HAM), for the NC and p100 conditions, and their difference (Δ). Peaks were normalized to each subject’s largest average peak for each muscle in NC. Condition effects were assessed with paired t tests, where * denotes significance (α = 0.05).

<table>
<thead>
<tr>
<th></th>
<th>TA₁ (%NC)</th>
<th>TA₂ (%NC)</th>
<th>SOL (%NC)</th>
<th>VL (%NC)</th>
<th>HAM (%NC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td>NC</td>
<td>100 ± 0</td>
<td>49.1 ± 9.0</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>81.3 ± 13.5</td>
<td>46.3 ± 17.9</td>
<td>103.9 ± 25.1</td>
<td>119.8 ± 19.5</td>
</tr>
<tr>
<td></td>
<td>Δ</td>
<td>−18.7 ± 13.5*</td>
<td>−2.8 ± 16.0</td>
<td>3.9 ± 25.1</td>
<td>19.8 ± 19.5*</td>
</tr>
<tr>
<td>Right:</td>
<td>NC</td>
<td>100 ± 0</td>
<td>59.7 ± 18.9</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>100.8 ± 10.9</td>
<td>57.4 ± 17.3</td>
<td>94.4 ± 8.8</td>
<td>100.0 ± 11.7</td>
</tr>
<tr>
<td></td>
<td>Δ</td>
<td>0.8 ± 10.9</td>
<td>−2.3 ± 5.2</td>
<td>−5.6 ± 8.8</td>
<td>0.0 ± 11.7</td>
</tr>
</tbody>
</table>

Table 4.4: Group averages (±SD) of EMG amplitude times (percent gait cycle after PLLN) for TA (two peaks) SOL, VL and HAM, for the NC and p100 conditions, and their difference (Δ). Condition effects were assessed with paired t tests, where * denotes significance (α = 0.05).

<table>
<thead>
<tr>
<th></th>
<th>TA₁ (%GC)</th>
<th>TA₂ (%GC)</th>
<th>SOL (%GC)</th>
<th>VL (%GC)</th>
<th>HAM (%GC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td>NC</td>
<td>0.7 ± 1.3</td>
<td>63.3 ± 2.1</td>
<td>44.5 ± 2.7</td>
<td>6.3 ± 3.3</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>1.9 ± 1.4</td>
<td>64.9 ± 5.3</td>
<td>46.3 ± 3.1</td>
<td>7.4 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>Δ</td>
<td>1.3 ± 1.2*</td>
<td>1.5 ± 4.5</td>
<td>1.7 ± 2.4</td>
<td>1.2 ± 1.8</td>
</tr>
<tr>
<td>Right:</td>
<td>NC</td>
<td>0.7 ± 0.9</td>
<td>66.2 ± 2.5</td>
<td>45.4 ± 3.3</td>
<td>7.0 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>0.6 ± 0.7</td>
<td>66.3 ± 2.5</td>
<td>46.1 ± 3.2</td>
<td>7.1 ± 2.0</td>
</tr>
<tr>
<td></td>
<td>Δ</td>
<td>−0.1 ± 0.6</td>
<td>0.1 ± 0.5</td>
<td>0.7 ± 1.1</td>
<td>0.0 ± 0.9</td>
</tr>
</tbody>
</table>
4.3.5 Movement variance

Long-term variance

In addition to the significant changes in timing, and after accounting for those changes by applying the temporal-normalization process of piecewise linear length normalization (PLLN), there still remained several differences between correlations of NC and p100 kinematics (Table 4.5). The correlations between NC and p100 of the left hip angle ($r_{\{x,y\}}: 99.69 \pm 0.17, p = 0.020$), knee angle ($r_{\{x,y\}}: 99.44 \pm 0.44, p = 0.012$) and ankle angle ($r_{\{x,y\}}: 93.61 \pm 2.48, p < 0.001$) were significantly lower than the
predetermined threshold of $r^*_{\{x^{(o)\prime},x^{(e)\prime}\}} = 99.82$. The right limb also experienced significant changes to the ankle angle trajectory ($r_{\{x,y\}}: 99.03 \pm 0.62, p = 0.002$). The whole-limb angles for both limbs were not significantly affected ($r_{\{x,y\}} \geq 99.94$), nor were the right hip and knee joint angles ($r_{\{x,y\}} \geq 99.78$).

**Short-term variance**

The short-term variances of angular data changed slightly due to the increased stiffness (Table 4.6). After adding the perturbation, the left ankle angle variability decreased significantly ($\Delta = -22.9 \pm 14.6$ deg, $p = 0.001$), and the left whole-limb angle variability increased significantly ($\Delta = 9.9 \pm 9.0$ deg, $p = 0.010$). In the right limb, the ankle angle variability increased significantly ($\Delta = 17.6 \pm 18.6$ deg, $p = 0.02$), as did the knee angle ($\Delta = 12.0 \pm 11.9$ deg, $p = 0.014$). The other comparison of interest was the whole-limb angle versus the individual joint angles. The repeated-measures ANOVAs indicated significant differences between angle variances for all conditions bilaterally ($p \leq 0.001$). Post-hoc $t$ tests found that in all cases except for the left ankle during p100 ($p = 0.063$), the whole-limb angle variances were significantly less than the hip, knee, and ankle joint angle variances ($p < 0.001$).

**Table 4.5: Long-term variance group averages (\(\pm\)SD) of cross correlations between the NC (\(x\)) and p100 (\(y\)) conditions \(r_{\{x,y\}}\) for whole-limb, hip, knee, and ankle angles. Self correlation of NC \(r_{\{x^{(o)\prime},x^{(e)\prime}\}}\) is given as reference. The null hypothesis that the group mean of \(r_{\{x,y\}}\) \(\geq 99.82\) was tested with one-sided $t$ tests, where $^*$ denotes the p100 group is statistically lower than 99.82 ($\alpha = 0.05$).**

<table>
<thead>
<tr>
<th></th>
<th>Whole-limb</th>
<th>Hip</th>
<th>Knee</th>
<th>Ankle</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left:</strong></td>
<td>(r_{{x^{(o)\prime},x^{(e)\prime}}})</td>
<td>99.99 ± 0.00</td>
<td>99.95 ± 0.03</td>
<td>99.96 ± 0.03</td>
</tr>
<tr>
<td></td>
<td>(r_{{x,y}})</td>
<td>99.96 ± 0.03</td>
<td>99.69 ± 0.17$^*$</td>
<td>99.44 ± 0.44$^*$</td>
</tr>
<tr>
<td><strong>Right:</strong></td>
<td>(r_{{x^{(o)\prime},x^{(e)\prime}}})</td>
<td>99.99 ± 0.01</td>
<td>99.96 ± 0.04</td>
<td>99.98 ± 0.02</td>
</tr>
<tr>
<td></td>
<td>(r_{{x,y}})</td>
<td>99.94 ± 0.04</td>
<td>99.78 ± 0.12</td>
<td>99.82 ± 0.13</td>
</tr>
</tbody>
</table>
Table 4.6: Short-term variance group averages (±SD) of summed standard deviations for whole-limb, hip, knee, and ankle angles, for the NC and p100 conditions, and their difference (Δ). For each angle, condition effects were assessed with paired t tests, where * denotes statistical significance (α = 0.05). Within each condition, † denotes joint angle values that are statistically higher than whole-limb angle.

<table>
<thead>
<tr>
<th></th>
<th>Whole-limb (deg)</th>
<th>Hip (deg)</th>
<th>Knee (deg)</th>
<th>Ankle (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>60.4 ± 10.7</td>
<td>111.5 ± 25.6†</td>
<td>145.4 ± 24.5†</td>
<td>105.0 ± 18.5†</td>
</tr>
<tr>
<td>p100</td>
<td>70.3 ± 14.8</td>
<td>116.4 ± 33.2†</td>
<td>154.1 ± 39.2†</td>
<td>82.1 ± 18.0</td>
</tr>
<tr>
<td>Δ</td>
<td>9.9 ± 9.0*</td>
<td>4.9 ± 19.5</td>
<td>8.7 ± 24.4</td>
<td>−22.9 ± 14.6*</td>
</tr>
<tr>
<td>Right:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>58.7 ± 8.5</td>
<td>105.3 ± 29.1†</td>
<td>152.9 ± 43.8†</td>
<td>105.0 ± 28.6†</td>
</tr>
<tr>
<td>p100</td>
<td>61.9 ± 11.7</td>
<td>113.1 ± 25.8†</td>
<td>164.8 ± 45.9†</td>
<td>122.6 ± 36.6†</td>
</tr>
<tr>
<td>Δ</td>
<td>3.2 ± 7.4</td>
<td>7.8 ± 17.6</td>
<td>12.0 ± 11.9*</td>
<td>17.6 ± 18.6*</td>
</tr>
</tbody>
</table>

4.4 Discussion

The results of this study show a clear indication of kinematic and kinetic compensation strategies to maintain successful gait in the presence of increased ankle stiffness. Further, we see that the whole-limb kinematics were preserved across the group, indicating that the hypothesis of maintained limb-level control is confirmed.

4.4.1 Gait events

Changes to stride time and gait events between the unconstrained (NC) and constrained (p100) conditions provide a good quantification of the overall effects of the left ankle perturbation and resulting neuromuscular control adaptations. They must also be kept in mind when considering the remaining results, as data were warped to align the sub-phases of gait (Fig. 4.3) using piecewise linear length normalization (PLLN). Timing changes within the gait cycle might indicate areas where the chosen adaptations were not able to fully overcome the limitations placed on the left ankle. Alternatively, these changes could be a result of the adaptation strategies themselves.

Increased stride times indicate that subjects may have tried reducing step frequency as a compensation strategy to the added perturbation (Table 4.1). Since the
treadmill speed was fixed, we can only infer that subjects most likely wanted to slow down, as seen in previous ankle-foot orthosis (AFO) studies with healthy adults [e.g., 94]. One might argue whether there is a clinical relevance to this increased stride time, since it only increased by 0.03 seconds, or 2.5% of the cycle time during the NC condition. The constraint placed on gait by the treadmill speed offers some support that the increased stride times are significant, as the kinematics must change to accommodate the same gait speed with decreased stepping rates. Further, there were no outlying subjects with decreased stride time during the p100 condition. Clearly, the longer stride time is a compensation strategy that every subject used to some degree as a result of increased ankle stiffness.

Looking within the phases of the gait cycle, the delayed weight alignment (WA) of the left limb indicates that subjects had difficulty progressing through mid-stance, when the body was rolling over the ankle (Table 4.2). The left ankle attempted to counteract this delay in WA by reducing the biological plantarflexion moment (Fig. 4.10). Left knee-ankle alignment (KAA) was also delayed significantly, indicating a longer mid-swing. Interestingly, the regions of deviation results indicate that both the left hip and knee joints were significantly more flexed during late mid-swing and early terminal swing of p100 (Fig. 4.8a,c). This behavior indicates that subjects may have been less confident that their foot would successfully clear the ground during swing. This change in limb configuration would also put the ankle slightly further behind the knee, causing the delay in KAA.

During the right-limb gait cycle, subjects experienced early contralateral toe-off (CTO), most likely an artifact of slightly late contralateral heel strike (CHS) and early ipsilateral toe-off (ITO) during the left cycle. Notice the associated left-limb values in Table 4.2, and consider that these values define both the left limb pre-swing and the right limb loading response. When the left events are combined, although each not significant, they create a shorter phase by the same amount that the right CTO
was changed. One could argue that the subjects were trying to load their right limb faster as a stability mechanism, but it is equally plausible that the significance of right CTO was caused by the AFO stiffness forcing the left ankle through pre-swing faster. While not statistically significant, it is interesting to note that the right CHS was generally early, and left CTO was generally delayed. This indicates that subjects may have tended to increase their double-limb support time when stepping with the affected (left) foot. Finally, we note that both toe-ankle alignment (TAA) and KAA were significantly delayed in the right limb, indicating an expanded initial swing phase, but a shorter terminal swing. This further supports the evidence above (late left WA) that the left limb was delayed through mid-stance, with the subjects attempting to land the right foot early to compensate during terminal swing.

Similar arguments as those for the stride time changes could be made for or against the clinical significance of these gait event changes. Again, the physical restriction of treadmill speed imposed on the subject increases any significance found in the gait event results. Had the subjects walked overground, it is quite plausible that the left ankle perturbation would cause greater changes, and asymmetry, within the gait events. This result was found by Guillebastre et al. [94] in healthy adults wearing an AFO while walking overground. Discussing effects of the treadmill would be incomplete without considering the similarities or differences between treadmill and overground walking. Altman et al. [112] found that other than step width, lower-limb kinematics were not significantly affected by walking on a split-belt instrumented treadmill with similar gap between belts as in this study. Zeni Jr. and Higginson [113] found that after about five minutes of familiarization, movement variability reached a steady-state value. The current study allowed at least 30 minutes of familiarization time before any data were recorded.
4.4.2 Compensations throughout the gait cycle

Understandably, the left ankle was the joint most affected by the added AFO stiffness (Fig. 4.5e). The regions of deviation within the gait cycle for the kinematic and kinetic data provide further information on what changes were taking place across the group in order to compensate for the ankle perturbation (Figs. 4.8-4.10). Left ankle kinematics were particularly affected in the following ways (Table 4.2 and Fig. 4.8e):

1. Reduced plantarflexion during loading response.

2. Prolonged mid-stance.

3. Reduced plantarflexion during pre-swing (push-off) and throughout swing.

The specific left-limb compensations to each of these restrictions, as well as the corresponding compensations within the right limb, will be detailed below.

Reduced plantarflexion during loading response

The perturbation created reduced ankle plantarflexion and increased net dorsiflexor moment during loading response (Figs. 4.8e and 4.9e). Subjects reduced their biological dorsiflexor moment slightly during this phase (Fig. 4.10), also indicated with reduced tibialis anterior (TA) activation (Table 4.3), but not enough to return normal ankle motion. Left TA activation was also slightly later in the cycle (Table 4.4), indicating subjects’ initial reliance on the AFO stiffness upon heel strike. The remaining kinematic perturbation was then counteracted by increased hip and knee flexion (Fig. 4.8a,c). This strategy allowed subjects to maintain normal timing through loading response even though the AFO was impeding the natural rocker motion of the heel by slowing rotation to foot flat. The hip and knee joint moments were not initially altered until the latter half of the loading response (Fig. 4.9a,c). This suggests subjects found a low-effort kinematic compensation to the stiffness effects during impact,
but later compensated for the more crouched posture with increased knee and hip extensor moments. Left vastus lateralis (VL) activation also increased in the middle of loading response (Table 4.3), confirming this behavior.

**Prolonged mid-stance**

As discussed in Sec. 4.4.1, left WA occurred later during p100, indicating that subjects had difficulty progressing through mid-stance, when the body was rolling over the ankle (Table 4.2). During this time, and through a majority of terminal stance, the biological left ankle moment was significantly decreased (less plantarflexion, Fig. 4.10). This compensation would allow the body to roll over the ankle faster, otherwise there may have been more delay. After accounting for this timing change through PLLN, the decreased left ankle moment successfully maintained normal left-limb kinematics through mid-stance and terminal stance, including a reduction in timing delay at CHS. The knee extensor moment was also significantly higher during mid-stance (Fig. 4.9c). This behavior could be a continued compensation from the altered knee kinematics during loading response, as discussed above, or perhaps another compensation strategy for delayed WA.

**Reduced push-off during pre-swing**

The perturbation also created increased left ankle dorsiflexion throughout pre-swing and the entire swing phase (Fig. 4.8e). The changes during pre-swing affected gait the most, as push-off force from ankle plantarflexion is a key contributor to gait propulsion [84]. While the left ankle angle during a majority of terminal stance was normal, when push-off was most prominent, we saw a significantly decreased biological plantarflexor moment (Fig. 4.10). Here, subjects allowed the AFO stiffness to assist in ankle plantarflexion by relaxing their own moment production. They returned to normal moment production for the pre-swing phase until the AFO resisted
that motion as well, limiting the ankle’s range of motion throughout push off. The net ankle moment (biological plus AFO) was not significantly changed during this time, until later in pre-swing (Fig. 4.9e), which in combination with the kinematic restriction of the AFO stiffness suggests decreased push off. Subjects then adapted by extending their hip and knee joints to compensate for the reduced ankle motion (Fig. 4.8a,c). This compensation occurred earlier than the restriction to ankle plantarflexion, confirming the presence of adapted motor control behavior, rather than just reactive compensation. Interestingly, the left hip moment showed a secondary compensation after the increased hip and knee extension. During the NC condition, we saw a small extensor moment peak near toe-off, which disappeared during p100 (Fig. 4.6a), and was a significant deviation from normal (Fig. 4.9a). This decrease in extensor moment (or increase in flexor moment) may have been the subjects’ attempt to advance the limb into the swing phase faster, and counteract the delay caused by extending the entire limb further through terminal stance and early pre-swing.

While the increased dorsiflexion during swing phase does not affect ambulation, it does present some interesting compensations. The discussion of delayed left KAA in Sec. 4.4.1 mentions that perhaps subjects were unsure of their foot clearance during swing. We also see that the left hamstrings (HAM) activation was significantly delayed during terminal swing (Table 4.4). This delay could be due to the knee being more flexed up until that point, thus delaying the deceleration of the shank before heel strike (Fig. 4.8c). In addition, we see that the biological left ankle moment was significantly increased in plantarflexion. This seems counterintuitive, but the subjects may have simply reduced their dorsiflexion moment as the AFO stiffness provided dorsiflexor torque. Perhaps reducing ankle moments while slightly flexing the hip and knee was an easier strategy for subjects to perform than simply lifting their toes further. This behavior could suggest that subjects were also unsure of their heel clearance with the added ankle stiffness. The latter reasoning is more plausible,
because if subjects were worried about their toes hitting, they most likely would increase their dorsiflexor moment.

**Right limb compensations**

Since the right limb was not directly perturbed in this experiment, changes to this limb were most likely used to compensate for gait timing changes rather than physical restrictions caused by perturbation. We can gain further information about the specific compensations throughout the gait cycle by looking at the right limb’s regions of deviation results. During loading response, right limb kinematics do not deviate significantly for p100 compared to NC (Fig. 4.8b,d,f). However, recall that this phase is shortened by reduced left push-off (early right CTO, Table 4.2). All three right-limb joints can be seen compensating for this lack of propulsion in late loading response via increased hip, and decreased knee and ankle extensor moments (Fig. 4.9b,d,f). The increased hip extensor moment continues through early mid-stance, in order to counteract the increased hip flexion angle (Fig. 4.8b) caused by the reduced propulsion during loading response. These compensations by the right limb allowed subjects to progress through mid-stance normally even though the left ankle perturbation caused reduced propulsion during loading response.

Terminal stance was the only other phase of the right limb’s cycle significantly affected by the left ankle perturbation. Here, subjects exhibited decreased right knee extensor moments (Fig. 4.9d) and right ankle plantarflexor moments (Fig. 4.9f). These compensations would reduce the body’s forward speed and force upon impact of the left limb, perhaps to gain stability at CHS.

### 4.4.3 Movement variance

While the kinematic and kinetic deviations and muscle activation results highlight the individual adaptations and compensation strategies to the increased left ankle
stiffness during p100, the long-term and short-term variability results provide information about the plausibility of a limb-level controller goal during injury. Recall that long-term variability indicates adaptation to maintain successful gait in the presence of the perturbation, while short-term variability indicates less consistency from cycle to cycle, and perhaps less importance to the overall goals of gait. Subjects were able to successfully overcome the decrease in ankle range of motion to maintain a steady and symmetric gait when considering kinematics of the entire limb. The results also show overall adaptations to individual joint behaviors in order to successfully achieve the limb-level goal.

**Long-term variance**

One hypothesis of this study was that the neuromuscular control system attempts to maintain limb-level function in the face of injury or perturbation to a joint. Changes to subjects’ mean kinematic patterns between NC and p100 showed that the long-term compensation strategy was successful at achieving this goal. The cross-correlations for the bilateral whole-limb angles were not affected by the increased ankle stiffness (Table 4.5). In the presence of the simulated ankle injury, subjects were able to maintain the same patterns of whole-limb motion, resulting in similar gross body motion during gait. This limb-level consistency was maintained through compensations to individual joint kinematics, as seen in their deviations discussed above (Fig. 4.8), as well as their own cross-correlation results. The average behaviors of the left ankle, knee and hip, and right ankle experienced changes from NC to p100 based on the cross-correlation results (Table 4.5). These changes to the average behaviors corroborate the deviation results in that these joints show the most kinematic adaptation or compensation (Fig. 4.8).

While the right ankle long-term variance was statistically significant, there were no significant regions of deviation when comparing across the group (Fig. 4.8f). This sug-
gests that compensations to the right ankle kinematics were not consistent across all subjects. Since the right limb was not directly perturbed in this experiment, changes to the right ankle were most likely used to compensate for gait timing changes, and not necessarily for specific changes to the kinematics as a function of the restriction. Therefore, it is plausible that subjects could exhibit both positive and negative changes at any given point of the gait cycle, causing low average deviation values. The discussion of right ankle compensations in Sec. 4.4.2 further supports the claim that right ankle adaptations served the primary function of timing adjustments.

**Short-term variance**

In addition to the overall changes to the kinematic patterns described above, changes in short-term variance provide insight into the repeatability of these signals throughout the trial. Decreased variance across the gait cycle indicates that the kinematic pattern is more consistent throughout the trial. This consistency suggests that the neuromuscular control system is focusing on the variables with lower variance (the goal), while allowing more freedom in the variables with higher variance (the uncontrolled manifold). In conjunction with the long-term variance hypothesis, here it was assumed that the whole-limb angle would have lower variability than the individual joint angles, which was confirmed for NC as well as p100. It was also hypothesized that this variability would not increase for whole-limb angle, but would increase for joint angles, in the presence of increased ankle stiffness. This hypothesis was not confirmed, as discussed below.

While the left whole-limb angle exhibited less short-term variance than the corresponding joint angles, the changes in variance between conditions were different from those expected. Decreased ankle variance in p100 could be due to a higher concentration on maintaining whatever ankle kinematics are allowed during the perturbation (Table 4.6). It is also possible that the physical restriction of added ankle stiffness is
decreasing the cycle-to-cycle variability by limiting the effect that the control efforts could provide to the ankle. Left whole-limb variance significantly increased between conditions, indicating that the added perturbation to the ankle created less repeatable motion when considering the whole limb. However, left whole-limb variance was still significantly lower than the hip and knee angle variances during p100. Therefore, while affected by the perturbation, the whole-limb angle was still the most repeatable kinematic measure across all conditions for that limb. It should also be noted that the group standard deviations for the whole-limb variance metrics are about half of those for the individual joint angles, indicating more consistency throughout the entire group. This occurrence further supports the claim that maintaining whole-limb motion is an important goal during gait, at least for gait on a treadmill.

Cycle-to-cycle variance also increased significantly for the right knee and ankle (Table 4.6). Subjects’ mean behaviors of the knee throughout the trial were unchanged based on the cross-correlation analysis, but the increased short-term variance shows some compensation was still occurring. These results suggest that the neuromuscular control system was less focused on the right knee and ankle during p100 than NC. This could be due to the more demanding task of compensations within the left limb, or that right-limb consistency was less important to the overall goal. The increased variance of the right knee and ankle could also be working together to compensate for the changes to the right ankle mean trajectory. Whole-limb consistency was unchanged for the right limb, showing that the contralateral compensations to the left ankle perturbation were performed in conjunction with maintaining right limb-level function as well.
4.4.4 Summary and further thoughts

Summary of results

The results of this study show a clear indication of active bilateral kinematic and kinetic compensation strategies to maintain successful gait in the presence of increased ankle stiffness. Further, we see that the whole-limb kinematics were preserved across the group, aided by significant changes to individual joint behaviors. These compensatory relationships support our hypothesis that a major goal of gait is the motion of each limb as a whole. Additionally, short-term, or cycle-to-cycle, variability of the whole-limb angle remained significantly lower than the individual joint angles, suggesting that flexibility was exploited in the joint motions to maintain the limb-level behavior. This finding further supports the argument that the limb-level kinematics are the primary focus of the neuromuscular control system, with goal-equivalent compensations carried out at the expense of the individual joint angles’ variability.

Limitations and future work

Unfortunately, very few studies have focused on whole-limb behavior during locomotion, and no studies have been found which report a direct comparison of whole-limb motion during overground and treadmill walking. Further work should be completed to determine whether similar relationships between whole-limb variance (long-term and short-term) exist for overground walking as we found in this study for treadmill walking. Ivanenko et al. [75] found that sagittal-plane kinematics were similar between overground and treadmill walking. In both cases, the data were reduced to a planar space using principal component analysis, where the principal axes tended to correlate with whole-limb orientation and length, supporting our hypothesis of limb-level control. While that study did not directly compare whole-limb kinematics between treadmill and overground gait, the findings seem to support at least the
plausibility that long-term variance (changes to average behavior) were not an artifact of the treadmill. However, Dingwell et al. [114] claimed that treadmill walking reduced kinematic stride-to-stride variability during gait, and may have even suppressed changes for certain conditions. In particular, their results showed a slight decrease in joint angle variability from overground to treadmill walking of the same population, but the decrease was only statistically significant for the ankle. While we may not be able to make declarative statements about the changes (or lack thereof) to short-term variability between conditions in this experiment, we can still conclude that the decreased whole-limb variance supports the hypothesis that whole-limb behavior is an underlying kinematic goal of gait.

Another limitation of this study may be the perceived lack of difference between group averages of NC and p100 data. This work has quantified changes from NC to p100 within each subject, then statistically compared those results across the group. Otherwise, the large variance between subjects (e.g., Fig. 4.5) leads to certain changes between conditions being washed out, including the plotted average kinematics. Even still, within-subject changes from NC to p100 are not large. This may suggest that the neuromuscular control system is well adept at overcoming perturbations to the ankle. Considering that this work supports the idea that the overall locomotion goal is whole-limb function, it would make sense that the body could easily overcome perturbations to the ankle joint, as it is the most distal joint of the lower limb. Another factor contributing to high inter-subject variance could be that subjects may have utilized different strategies to overcome their compensation. An example of this is the right ankle result of statistically significant increases in long-term and short-term variance (Tables 4.5 and 4.6), but no significant regions of deviation (Fig. 4.8f).

There are several areas where further investigation is possible. First, including more subjects would allow for stronger statistical comparison, and a better determination of the clinical significance of the results. In relation to the previous paragraph’s
discussion, more subjects would also allow for a more in-depth study of coexisting compensation strategies between subjects. Also, even though subjects walked for 15 minutes after the addition of the perturbation, they may not have settled onto a true steady-state compensation strategy similar to those which someone who has worn an AFO for years might adopt. As this was part of a larger study, we were only able to allow subjects 15 minutes to acclimate to the increased stiffness. Noble and Prentice [7] suggest that this is sufficient time to reach steady-state kinematics, but their perturbation was not an increased joint stiffness. Only ground-reaction force data were available for every minute of each condition, which displayed steady-state behavior well before the final trials of each condition based on visual inspection.

Finally, this study uses the NC condition as “normal” rather than the base condition, requiring a rather large assumption that the AFO without added stiffness does not affect the subjects’ normal gait. While this assumption may not hold for all of the data considered here, it was a necessary trade-off in order to directly compare effects of only increased stiffness on the subjects’ compensation strategies. Future work is also warranted on the effect of the weight of the AFO on the left limb, and on the effect of the weight of the cylinder and load cell between NC and p100. It is possible that some of the adaptations discussed above could have been caused by the added 0.23 kg from the cylinder and load cell.

4.5 Conclusion

This study provides evidence of active bilateral kinematic and kinetic compensation strategies to maintain successful gait in the presence of a simulated injury via increased ankle stiffness. A limb-level goal equivalent manifold for human gait is also present (at least during treadmill walking), even during the perturbation. The primary effects of the perturbation occurred during loading response and push-off of the
affected (left) limb. At this level of perturbation, subjects were not able to, or simply chose not to, provide more ankle torque to overpower the AFO, as seen by the large effects to ankle motion during these phases of gait (Fig. 4.8e). Instead, they relied on kinematic and kinetic compensations throughout both limbs to help maintain the whole-limb behavior, which was very consistent across subjects.
Chapter 5

Studying the Progression of Compensation Changes During Simulated Recovery from Injury

Abstract

This study is focused on analyzing the manner in which compensations during gait change while recovering from an injury. The injury is simulated as a restriction in ankle function from increased stiffness of an orthotic device. Recovery is simulated by a systematic reduction along discrete stiffness levels. It is hypothesized that adaptations to the perturbation will not change at the same rate as the reduction in stiffness, but rather exhibit relative discontinuous changes or switch between coexisting strategies during recovery. Ten subjects walked without, then with (at three successively smaller values of stiffness), and again without, the added ankle stiffness. Their compensation strategies to the perturbations during recovery were assessed via lower-limb kinematics, kinetics, and muscle activation patterns. The results demonstrate that the compensation strategies developed for the largest ankle stiffness perturbation do not all recover at a comparable rate as the reductions in stiffness. There is evidence that some adaptations change at a different rate than the perturbation, as well as switching between different compensation strategies, between certain perturbation levels. These results suggest that clinicians should consider the possibility that multiple compensation strategies can achieve similar motor goals, and that the adaptations may follow different paths during recovery.
5.1 Introduction

In Chapter 4, an experiment to systematically explore compensations to injury and recovery was introduced. In that work, we showed that subjects successfully maintained a goal of whole-limb motion during gait, even when faced with increased unilateral ankle stiffness. The specific adaptations to the initial perturbation were detailed and analyzed, showing that the control of individual lower-limb degrees-of-freedom compensated to maintain whole-limb motion and minimize its cycle-to-cycle variance.

The current chapter is concerned less with the actual compensations to a particular injury or perturbation, but more with the manner in which these injury compensations change during simulated healing (or decreasing levels of perturbation). As seen in Sec. 4.2.1, the gait experiment has simulated a recovery from injury through controlled changes to ankle stiffness. Analysis of the gait compensations within each condition will be performed similar to Sec. 4.2.2, with focus placed on differences between all of the conditions.

While there is much interest in how patients compensate for particular injuries, much of the focus is specifically placed on the healing process in order to refine rehabilitation methods and track recovery. Certain populations have shown better adoption of successful compensation strategies than others during functional tasks after anterior cruciate ligament repair [24]. This finding suggests that physical restrictions or perhaps impairments to sensory feedback remain for a subset of patients recovering from injuries. Other studies have shown the return of functional tasks like walking before more difficult tasks like hopping [25, 26]. It is also possible that patients will develop compensation strategies, like muscle co-contraction, to increase joint stiffness and stability that remain well after the injury is healed [28]. The aforementioned comparisons of changes during rehabilitation can usually only be made to an individual’s healed state, or relative to a normative control group, which may not
be reflective of the individual’s pre-injury state. Thus, there is a need to more fully understand the mechanisms of control change during rehabilitation from an injury that can be directly and confidently compared to the healthy state prior to injury.

As in the previous chapter, analysis of compensations to the ankle perturbations is motivated by the motor redundancy problem [48] and the uncontrolled manifold hypothesis [49, 50]. Recall that Chapter 4 confirmed the hypothesis that limb-level function was a key goal of gait, seen via consistent whole-limb kinematics attained through changes to individual joint kinematics. This result was similar to findings in a peripheral nerve injury model to cats [69], where the authors examined individual joint behaviors in conjunction with whole-limb behavior via long-term and short-term kinematic variability. Long-term variability was quantified by the change in average kinematic behavior between conditions using linear regression. Short-term variability was then quantified by the amount of cycle-to-cycle variance for each kinematic behavior, summed across the gait cycle. Changes to long-term variability would indicate that those angular kinematics were adapted to maintain successful gait during the perturbation. Changes to short-term variability would indicate that those angular kinematics were less consistent from cycle to cycle, and perhaps were less integral to the overall goals of gait. Quantification of similar variability measures related to whole-limb motion could provide further evidence of limb-level control in human gait.

The underlying goal of this study was to examine the progression of changes to the compensation strategies discussed in Chapter 4 during recovery from the initial perturbation. Recovery from ankle injury was simulated using a custom ankle-foot orthosis (AFO) with adjustable bidirectional resistance to ankle motion. The ankle was chosen due to its importance to gait propulsion [84], and the AFO provided a controlled experimental environment for simulating abnormal gait with known conditions [cf. 8, 107]. Our hypothesis was that some of the compensations employed
during the full perturbation would not change at a comparable rate with the reductions in stiffness; rather they would exhibit relative discontinuities at certain points during reduction in perturbation, or switch between coexisting strategies. We also hypothesized that whole-limb motion would remain the primary goal of gait and be conserved throughout the recovery, in terms of both long-term and short-term variance. These assumptions were based both on the clinical rehabilitation results mentioned above, and discussed in more detail in Sec. 1.1.2, as well as the dynamic systems results of Chapters 2 and 3.

5.2 Methods

Much of the experimental design and data analysis were described in Sec. 4.2. The pertinent details will be reviewed below, along with any changes to the methodology considered in this chapter.

5.2.1 Experimental design

Ten able-bodied male subjects, age 25.5 ± 5.1 years, mass 77.4 ± 11.4 kg, and height 1.77 ± 0.05 m (mean ± SD), participated in the study after giving written informed consent. The protocol was approved by the University of Illinois at Urbana-Champaign Institutional Review Board. A custom AFO was fabricated for the left lower limb of each subject (Fig. 4.1), detailed in Sec. 4.2.1. A linear pneumatic cylinder (DSNU-5/8"-5"-P; Festo Inc., Hauppauge, NY) was attached to the posterior side of the AFO. Ankle motion was restricted by filling both ends of the cylinder with a known air pressure and closing the ports manually with flow-control valves (GR-QB-5/32-U; Festo Inc.). The force produced by the cylinder was measured with a bi-axial load cell (LC202-100; OMEGA Engineering Inc., Stamford, CT). The AFO with all added components weighed 1.44 kg. Some conditions required removal of the cylinder and
load cell, reducing the weight to 1.21 kg.

Subjects were tested on two consecutive days. On the first day, the subject was fitted with the AFO to ensure minimal ankle motion relative to the AFO joint. An eleventh subject (not included) experienced noticeably affected gait while wearing the AFO without resistance, and was excluded from participation. Next, surface electromyography (EMG) sensors were placed over the tibialis anterior (TA), soleus (SOL), vastus lateralis (VL), and hamstrings (HAM) of each leg (Bagnoli-16; Delsys Inc., Natick, MA). The subject was then asked to walk on an instrumented split-belt treadmill (Instrumented Treadmill; Bertec Corp., Columbus, OH) while wearing the AFO without the cylinder, to identify a comfortable walking speed and allow acclimation to the AFO.

On the second day, EMG sensors were reattached and reflective markers were prepared for three-dimensional motion capture via a five-camera system (460 Datastation; Vicon, Oxford, UK). The reflective markers were bilaterally attached over the anterior-superior iliac spine, greater trochanter of the femur, lateral mid-thigh, lateral epicondyle of the femur, tibial tuberosity, lateral malleolus, heel, and the first and fifth metatarsal heads; as well as the L5-S1 sacral junction. Markers were also attached to the treadmill and AFO. While wearing the AFO, markers were removed from the subject’s lateral malleolus and heel. These two positions were approximated using the markers on the AFO, offset with static corrections.

The following assessments were then performed in order for each subject:

**base** To capture his baseline walking behavior, the subject walked on the treadmill in his running shoes for seven minutes, then rested for 10 minutes.

**NC** While wearing the AFO with zero resistance (no cylinder) on the left foot and running shoe on the right, the subject walked for seven minutes to re-acclimate to the AFO, then rested for 10 minutes.
p100 The cylinder was attached and the AFO was adjusted to the “full” perturbation\(^1\), where the cylinder was charged with 100 psig of air. The subject walked for 15 minutes then rested for 15 minutes.

p60 The AFO was adjusted to 60 psig, and the subject walked for seven minutes, then rested for 10 minutes. It was anticipated that the subject would require less time to reach steady state walking patterns since the resistance change is much lower than in p100, and moving toward normal.

p20 Condition p60 was repeated at 20 psig for seven minutes.

NC2 Condition NC was repeated for 15 minutes.

5.2.2 Data collection and analysis

During each of the assessments described above, motion capture marker locations, ground reaction forces, EMG signals, and axial force on the AFO cylinder (for p100 through p20; LC202-100, OMEGA Engineering Inc.; Stamford, CT) were recorded for 30-second trials at each minute (more detail in Sec. 4.2.2). To study steady-state compensation, all analyses were focused on the final 30 seconds of each condition listed above. The data were then processed in MATLAB using custom code. Sagittal-plane kinematics (joint angles) and kinetics (net joint moments) of the hip, knee, and ankle joints were computed, including the torque produced by the AFO cylinder. Limb-level function was characterized by the “whole-limb” angle, or the global angle of the hip-to-toe vector from the anterior direction (cf. Fig. 4.2). EMG signal means were zeroed, band-pass filtered (20-250 Hz 4\(^{th}\)-order recursive Butterworth), rectified, and low-pass filtered (7 Hz 4\(^{th}\)-order recursive Butterworth) to produce linear envelopes of the muscle activation signals.

\(^1\)Note that full perturbation refers to the maximum cylinder pressure used, and not a full restriction of ankle motion.
Gait events

The following gait events were found within each gait cycle for both limbs and converted to 0 – 100% of the gait cycle [4, 108] (cf. Secs. 1.1.1 and 4.2.2, Fig. 1.1): Ipsilateral heel strike (IHS1), contralateral toe off (CTO), weight alignment (WA), contralateral heel strike (CHS), ipsilateral toe off (ITO), toe-ankle alignment (TAA), knee-ankle alignment (KAA), and the second ipsilateral heel strike (IHS2). Piecewise linear length normalization (PLLN) was used to align these events and create constant sampling from 0 – 100% of the period for all gait cycles [5, 109]. To assess condition effects, repeated-measures ANOVAs were performed across all conditions for each event (α = 0.05). For events showing significance across conditions, post-hoc testing determined specific differences between individual conditions via paired t tests (α = 0.05). In order to capture changes to the gait cycle as a whole, the original stride time (in seconds) from IHS1 to IHS2 was also statistically compared.

Regions of deviation

Each subject’s average behaviors in conditions p100 through NC2 were compared against his normative data in NC throughout the gait cycle using the regions of deviation method described in Sec. 4.2.2 [8, 9]. Let \( x = \{x_i\}_{i=0}^{100} \) and \( y = \{y_i\}_{i=0}^{100} \) be sequences of data for one full gait cycle, warped to align gait events using PLLN. Each of the 101 data points correspond to the \( i^{th} \) percentage of the cycle, averaged across all cycles from one trial. Here, \( x \) denotes data from NC and \( y \) from one of conditions p100 through NC2. Finally, let \( \sigma^{(x)} = \{\sigma_i^{(x)}\}_{i=0}^{100} \) denote the sequence of standard deviations in NC, across all gait cycles for one trial. Recall the calculation for \( D_i^{(y)} \), the deviation from normal for signal \( y \) at each point \( i \) of the gait cycle, given in Eq. 4.1 and repeated here.
\( D_i^{(y)} = \begin{cases} 
    y_i - (x_i + \sigma_i^{(x)}), & y_i > (x_i + \sigma_i^{(x)}) \\
    0, & (x_i - \sigma_i^{(x)}) \leq y_i \leq (x_i + \sigma_i^{(x)}) \\
    y_i - (x_i - \sigma_i^{(x)}), & y_i < (x_i - \sigma_i^{(x)}) 
\end{cases} \) (5.1)

For a statistical comparison throughout the entire gait cycle, the group’s deviations from each condition (p100 through NC2) were compared against zero with a one-sample \( t \) test at every point \( i \) in the gait cycle \((\alpha = 0.05)\). All points proving significantly different were then considered regions of deviation from the NC condition for the group.

Regions of deviation were evaluated for the joint angles and moments described above in order to provide a clear understanding of the limitations and compensation mechanisms created by the perturbation throughout the gait cycle. During conditions p100 through p20, the net left ankle moment is a combination of the biologically-produced muscle moment and the AFO torque. Therefore, the biological left ankle moment was computed as the difference between the net moment and AFO torque.

**Muscle activations**

Peak values were found from the EMG linear envelopes to assess the underlying muscle activations during each condition. The activation peaks were normalized by the average of the maximum peak value during NC for each subject and signal. To assess condition effects on the peak amplitudes and timings, repeated-measures ANOVAs were performed across all conditions of a given limb \((\alpha = 0.05)\) for each measure. For values showing significance across conditions, post-hoc testing determined specific differences between individual conditions via paired \( t \) tests \((\alpha = 0.05)\). Significant changes would give a more direct insight to the neuromuscular control system’s commands to compensate for the ankle stiffness. Due to significant noise, only nine of the subjects were included in the EMG analyses, except for the following muscles:
left HAM (two excluded), right VL (three excluded), and right HAM (two excluded).

**Movement variance**

The subject’s ability to maintain the overall goal of whole-limb motion was tested using the ideas of long-term and short-term variability introduced above.

**Long-term variance**  Recall in Chapter 4 that we quantified each subject’s overall change in kinematics between NC and p100 using the normalized cross-correlation function with zero phase shift [110, 111], given by Eq. (4.2) and repeated here

\[
 r_{\{x,y\}} = \frac{\sum_{i=0}^{100} (x_i - \bar{x})(y_i - \bar{y})}{\sqrt{\sum_{i=0}^{100} (x_i - \bar{x})^2} \sqrt{\sum_{i=0}^{100} (y_i - \bar{y})^2}} \times 100
\]  

(5.2)

where \( x = \{x_i\}_{i=0}^{100} \) and \( y = \{y_i\}_{i=0}^{100} \) are sequences of data for one full gait cycle, previously described for Eq. (5.1). Again, \( x \) denotes data from NC and \( y \) from the condition being compared (one of p100 through NC2), and the sequence averages are denoted \( \bar{x} \) and \( \bar{y} \). The correlations were computed for the *individual* joint angles (hip, knee, ankle), as well as the *whole-limb* angle (cf. Fig. 4.2), for each subject. Finally, we considered a “self-correlation” of the NC condition\(^2\), \( r_{\{x^{(o)},x^{(e)}\}} \) computed using Eq. (5.2), where \( x^{(o)} \) and \( x^{(e)} \) are average cycles from the *odd* and *even* gait cycles in NC, respectively.

To test whether each condition changed from NC for each angle, \( r_{\{x,y\}} \) from p100 through NC2 were compared to the self-correlation threshold, \( r_{\{x^{(o)},x^{(e)}\}}^* \), defined as the lowest group mean value of \( r_{\{x^{(o)},x^{(e)}\}} \) across all signals. The right ankle exhibited the lowest mean self-correlation across the group, resulting in \( r_{\{x^{(o)},x^{(e)}\}}^* = 99.82 \). Paired one-tailed \( t \) tests (\( \alpha = 0.05 \)) were used to test the null hypothesis that \( r_{\{x,y\}} \geq r_{\{x^{(o)},x^{(e)}\}}^* \) for each signal in conditions p100 through NC2. Rejection of the null hypothesis would indicate the presence of long-term variability between conditions.

\(^2\)Note that the terminology “auto-correlation” is avoided due to its specific definition of the cross-correlation of one signal onto itself.
for that signal\(^3\).

**Short-term variance**  Again following the previous chapter, and the analysis of Chang et al. [69], cycle-to-cycle variance for the whole-limb and individual joint angles were quantified by cumulatively summing the standard deviations across the gait cycle for each subject:

\[
\sigma_{\text{tot}} = \sum_{i=0}^{100} \sigma_i
\]

where \(\sigma_i\) is the standard deviation of a signal at the \(i^{\text{th}}\) percentage of the cycle, previously described for Eq. (5.1). To assess condition effects on short-term variability, repeated-measures ANOVAs were performed across all conditions of a given limb (\(\alpha = 0.05\)). For signals showing significance across conditions, post-hoc testing determined specific differences between individual conditions via paired \(t\) tests (\(\alpha = 0.05\)). To test differences between whole-limb angle variance and individual joint angle variances, repeated-measures ANOVAs were performed *within* each condition (\(\alpha = 0.05\)) for all signals of a given limb. In cases where a condition showed significance, post-hoc testing determined specific differences between the whole-limb angle and each joint angle via paired \(t\) tests (\(\alpha = 0.05\)).

\(^3\)While the analysis of long-term variance was motivated by Chang et al. [69], it should be noted that our quantification was performed in a different manner.
5.3 Results

To briefly summarize all of the results presented below, this study has looked at gait events normalized to percent gait cycle (\%GC), regions of deviation of joint kinematics and kinetics, muscle activation peaks, and movement variance (both long-term and short-term) of joint and whole-limb angles. These measures were analyzed during the initial perturbation of increased left ankle stiffness (p100), and throughout recovery from this perturbation (p60 through NC2), compared to the initial unperturbed state (NC).

5.3.1 Gait events

Test condition had a significant effect on left and right stride time \((p = 0.001)\), left weight alignment \((WA, p = 0.008)\), left knee-ankle alignment \((KAA, p = 0.026)\), right contralateral toe-off \((CTO, p < 0.001)\), and right toe-ankle alignment \((TAA, p < 0.001)\), based on the repeated-measures ANOVAs. Post-hoc \(t\) tests determined that bilateral stride time was significantly longer than NC for p100 \((p = 0.001)\) and p20 \((p \leq 0.048)\) (Table 5.1). Also, p100 exhibited later left WA \((p = 0.002)\) and KAA \((p = 0.005)\), while all three perturbation conditions \((p100 \text{ through } p20)\) exhibited earlier right CTO \((p \leq 0.001)\), as well as later right TAA \((p \leq 0.015)\) (Table 5.2).

Table 5.1: Group averages (±SD) of total stride time for all conditions. Condition effects were assessed with repeated-measures ANOVAs and post-hoc \(t\) tests, where * denotes statistical difference from NC \((\alpha = 0.05)\). AFO worn on left limb, running shoe on right.

<table>
<thead>
<tr>
<th></th>
<th>Left Stride Time (sec)</th>
<th>Right Stride Time (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NC</td>
<td>1.19 ± 0.06</td>
<td>1.19 ± 0.06</td>
</tr>
<tr>
<td>p100</td>
<td>1.22 ± 0.06*</td>
<td>1.22 ± 0.06*</td>
</tr>
<tr>
<td>p60</td>
<td>1.20 ± 0.06</td>
<td>1.20 ± 0.06</td>
</tr>
<tr>
<td>p20</td>
<td>1.20 ± 0.05*</td>
<td>1.20 ± 0.05*</td>
</tr>
<tr>
<td>NC2</td>
<td>1.20 ± 0.05</td>
<td>1.20 ± 0.05</td>
</tr>
</tbody>
</table>
Note that during conditions p100 to the control effort necessary to overcome the left ankle perturbation (Fig. 4.6). Joint kinetics computed via inverse dynamics then provided further information as the largest visible differences in joint kinematics were in the left ankle (Fig. 4.5e). The whole-limb angles were largely unchanged between conditions (Fig. 4.4), while the group average behaviors of the gait kinematics and kinetics (Figs. 4.4–4.7). Events are expressed in terms of percentage of the full gait cycle. Condition effects were assessed with repeated-measures ANOVAs and post-hoc t tests, where * denotes statistical difference from NC (α = 0.05). Note that IHS1 and IHS2 are not displayed since they are 0% and 100% by definition.

<table>
<thead>
<tr>
<th></th>
<th>CTO (%GC)</th>
<th>WA (%GC)</th>
<th>CHS (%GC)</th>
<th>ITO (%GC)</th>
<th>TAA (%GC)</th>
<th>KAA (%GC)</th>
</tr>
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<tbody>
<tr>
<td>Left:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>15.0 ± 1.0</td>
<td>39.9 ± 1.3</td>
<td>49.5 ± 0.9</td>
<td>64.4 ± 0.9</td>
<td>78.1 ± 1.0</td>
<td>90.2 ± 1.5</td>
</tr>
<tr>
<td>p100</td>
<td>15.3 ± 1.4</td>
<td>41.0 ± 1.7*</td>
<td>49.9 ± 1.2</td>
<td>63.8 ± 1.5</td>
<td>78.6 ± 1.3</td>
<td>91.0 ± 1.5*</td>
</tr>
<tr>
<td>p60</td>
<td>15.5 ± 1.4</td>
<td>40.4 ± 1.4</td>
<td>49.6 ± 1.1</td>
<td>63.9 ± 1.5</td>
<td>78.4 ± 1.6</td>
<td>90.8 ± 2.0</td>
</tr>
<tr>
<td>p20</td>
<td>15.3 ± 1.3</td>
<td>40.1 ± 1.5</td>
<td>49.6 ± 0.9</td>
<td>63.8 ± 1.3</td>
<td>78.3 ± 1.2</td>
<td>90.6 ± 1.5</td>
</tr>
<tr>
<td>NC2</td>
<td>15.0 ± 1.3</td>
<td>40.0 ± 1.2</td>
<td>49.4 ± 0.8</td>
<td>64.4 ± 1.2</td>
<td>78.2 ± 1.4</td>
<td>90.1 ± 1.1</td>
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<tr>
<td>Right:</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>NC</td>
<td>14.9 ± 1.0</td>
<td>40.5 ± 1.7</td>
<td>50.5 ± 0.9</td>
<td>65.5 ± 1.1</td>
<td>78.6 ± 0.9</td>
<td>90.1 ± 1.1</td>
</tr>
<tr>
<td>p100</td>
<td>13.8 ± 1.0*</td>
<td>40.7 ± 1.3</td>
<td>50.1 ± 1.2</td>
<td>65.4 ± 1.1</td>
<td>79.5 ± 0.9*</td>
<td>90.7 ± 0.8</td>
</tr>
<tr>
<td>p60</td>
<td>14.3 ± 1.0*</td>
<td>41.0 ± 2.1</td>
<td>50.4 ± 1.1</td>
<td>65.8 ± 1.2</td>
<td>79.3 ± 0.9*</td>
<td>90.6 ± 1.1</td>
</tr>
<tr>
<td>p20</td>
<td>14.2 ± 1.0*</td>
<td>40.8 ± 1.8</td>
<td>50.4 ± 0.9</td>
<td>65.7 ± 1.0</td>
<td>79.3 ± 0.9*</td>
<td>90.6 ± 0.8</td>
</tr>
<tr>
<td>NC2</td>
<td>15.0 ± 1.1</td>
<td>40.5 ± 1.4</td>
<td>50.6 ± 0.8</td>
<td>65.6 ± 1.1</td>
<td>79.1 ± 1.0</td>
<td>90.5 ± 0.8</td>
</tr>
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### 5.3.2 Joint kinematics and kinetics

Recall from Sec. 4.3.2 that slight differences were noticeable between NC and p100 for the group average behaviors of the gait kinematics and kinetics (Figs. 4.4–4.7). Whole-limb angles were largely unchanged between conditions (Fig. 4.4), while the largest visible differences in joint kinematics were in the left ankle (Fig. 4.5e). The joint kinetics computed via inverse dynamics then provided further information as to the control effort necessary to overcome the left ankle perturbation (Fig. 4.6). Note that during conditions p100 through p20, the net left ankle moment (Fig. 4.6e, red curve) included contribution from both the ankle-foot orthosis (AFO) torque produced by the cylinder (Fig. 4.7a), as well as the biological net muscle moment at the left ankle (Fig. 4.7b, red curve). Stiffness produced by the AFO is assumed to be negligible during NC and NC2. While not plotted here, the average behaviors trended back toward normal (NC) during recovery from the perturbation (p60 through NC2), as will be detailed in the chosen metrics below.
5.3.3 Regions of deviation

The group average behaviors for joint angle deviation values, $D$ from Eq. (5.1), along with the significant regions of deviation (shaded, Fig. 5.1), provide insight into the locations of altered kinematics throughout the gait cycle due to motion restriction of the left ankle, and how these altered kinematics change during recovery. Here, only the left knee (Fig. 5.1a,c,e) and left ankle (Fig. 5.1b,d,f) are shown for conditions $p_{100}$ through $p_{20}$. Clinically relevant regions of deviation were not found for the remaining joint angle kinematics after $p_{100}$. Recall the original deviations for all joint angles during $p_{100}$ for those not displayed here (Fig. 4.8). In general, the deviations found in $p_{100}$ reduce throughout the protocol, and no significant regions of deviation were identified during NC2.

The group average behaviors for deviation values for joint moment data, along with the significant regions of deviation (shaded, Figs. 5.2–5.4), provide further insight into the instances throughout the gait cycle where the subjects’ control strategies changed to compensate for the increased ankle stiffness. In all cases we see that the deviations found in $p_{100}$ reduce throughout the protocol. Right ankle data are not shown as the regions of deviation vanish after $p_{100}$ (cf. Fig. 4.9f), and in all cases, joint moment deviations were almost nonexistent during NC2.
Figure 5.1: Group averages of joint angle deviations ($D$) from each subject’s NC normative window for p100-p20. Significant regions of deviation (shaded) were found with a one-sample $t$ test against zero ($\alpha = 0.05$) at each percent of the gait cycle. AFO worn on left limb, running shoe on right.
Figure 5.2: Group averages of joint moment deviations ($D$) from each subject’s NC normative window for p100-p20 for the hips. Significant regions of deviation (shaded) were found with a one-sample t test against zero ($\alpha = 0.05$) at each percent of the gait cycle.
Figure 5.3: Group averages of joint moment deviations ($D$) from each subject’s NC normative window for p100-p20 for the knees. Significant regions of deviation (shaded) were found with a one-sample $t$ test against zero ($\alpha = 0.05$) at each percent of the gait cycle.
Figure 5.4: Group averages of joint moment deviations (D) from each subject’s NC normative window for p100-p20 for the left ankle. Significant regions of deviation (shaded) were found with a one-sample t test against zero (\(\alpha = 0.05\)) at each percent of the gait cycle.
5.3.4 Muscle activations

The electromyography (EMG) peak amplitudes and timings give a more direct insight to the neuromuscular control system’s commands to compensate for the ankle stiffness (Tables 5.3 and 5.4). Recall that the linear envelope peaks that were analyzed are indicated on plots of the group average normalized EMG envelopes (Fig. 4.11). The repeated-measures ANOVAs indicated that the peak amplitudes of left tibialis anterior (TA) (first peak, \( p = 0.002 \)) and left vastus lateralis (VL) \( (p = 0.032) \), as well as the peak timings of left TA (first peak, \( p = 0.015 \)) and left hamstrings (HAM) \( (p = 0.014) \) were significantly affected by condition. Post-hoc \( t \) tests determined that the left TA peak was statistically lower than NC for \( p_{100} \) through \( p_{20} \) \( (p \leq 0.025) \), and delayed for \( p_{100} \) \( (p = 0.019) \). Also, left VL increased significantly for \( p_{100} \) \( (p = 0.021) \), and left HAM was delayed for \( p_{100} \) \( (p < 0.001) \) and \( p_{20} \) \( (p = 0.047) \).

There were no significant changes to the right limb’s EMG activation peaks.

Table 5.3: Group averages (±SD) of normalized EMG amplitudes for tibialis anterior (TA, two peaks), soleus (SOL), vastus lateralis (VL) and hamstrings (HAM) for all conditions. Peaks were normalized to each subject’s largest average peak for each muscle in NC. Condition effects were assessed with repeated-measures ANOVAs and post-hoc \( t \) tests, where \( \ast \) denotes statistical difference from NC \( (\alpha = 0.05) \).

<table>
<thead>
<tr>
<th></th>
<th>TA1 (%NC)</th>
<th>TA2 (%NC)</th>
<th>SOL (%NC)</th>
<th>VL (%NC)</th>
<th>HAM (%NC)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Left:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>100 ± 0</td>
<td>49.1 ± 9.0</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td>p100</td>
<td>81.3 ± 13.5( \ast )</td>
<td>46.3 ± 17.9</td>
<td>103.9 ± 25.1</td>
<td>119.8 ± 19.5( \ast )</td>
<td>104.2 ± 21.2</td>
</tr>
<tr>
<td>p60</td>
<td>84.2 ± 15.3( \ast )</td>
<td>52.3 ± 18.9</td>
<td>99.3 ± 9.8</td>
<td>105.0 ± 10.8</td>
<td>87.9 ± 13.5</td>
</tr>
<tr>
<td>p20</td>
<td>88.7 ± 11.6( \ast )</td>
<td>53.5 ± 12.1</td>
<td>94.5 ± 12.2</td>
<td>104.8 ± 12.5</td>
<td>101.7 ± 29.9</td>
</tr>
<tr>
<td>NC2</td>
<td>104.4 ± 10.2</td>
<td>52.0 ± 8.7</td>
<td>102.3 ± 16.0</td>
<td>105.9 ± 14.3</td>
<td>98.0 ± 27.6</td>
</tr>
<tr>
<td><strong>Right:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>100 ± 0</td>
<td>59.7 ± 18.9</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
<td>100 ± 0</td>
</tr>
<tr>
<td>p100</td>
<td>100.8 ± 10.9</td>
<td>57.4 ± 17.3</td>
<td>94.4 ± 8.8</td>
<td>100.0 ± 11.7</td>
<td>116.7 ± 26.0</td>
</tr>
<tr>
<td>p60</td>
<td>99.5 ± 10.6</td>
<td>60.4 ± 20.0</td>
<td>99.8 ± 11.5</td>
<td>103.5 ± 10.9</td>
<td>111.2 ± 31.2</td>
</tr>
<tr>
<td>p20</td>
<td>106.3 ± 9.4</td>
<td>59.2 ± 20.5</td>
<td>96.5 ± 8.2</td>
<td>110.1 ± 15.3</td>
<td>107.6 ± 18.8</td>
</tr>
<tr>
<td>NC2</td>
<td>100.1 ± 16.7</td>
<td>57.3 ± 20.6</td>
<td>95.7 ± 8.8</td>
<td>102.4 ± 16.6</td>
<td>108.5 ± 33.4</td>
</tr>
</tbody>
</table>
Table 5.4: Group averages (±SD) of EMG amplitude times (percent gait cycle after PLLN) for TA (two peaks) SOL, VL and HAM, for all conditions. Condition effects were assessed with repeated-measures ANOVAs and post-hoc t tests, where * denotes statistical difference from NC (α = 0.05).

<table>
<thead>
<tr>
<th></th>
<th>TA1 (%GC)</th>
<th>TA2 (%GC)</th>
<th>SOL (%GC)</th>
<th>VL (%GC)</th>
<th>HAM (%GC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td>NC</td>
<td>0.7 ± 1.3</td>
<td>63.3 ± 2.1</td>
<td>44.5 ± 2.7</td>
<td>6.3 ± 3.3</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>1.9 ± 1.4*</td>
<td>64.9 ± 5.3</td>
<td>46.3 ± 3.1</td>
<td>7.4 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>p60</td>
<td>1.4 ± 1.2</td>
<td>65.2 ± 5.7</td>
<td>45.1 ± 3.0</td>
<td>6.9 ± 2.9</td>
</tr>
<tr>
<td></td>
<td>p20</td>
<td>0.8 ± 1.6</td>
<td>64.6 ± 5.1</td>
<td>45.4 ± 2.5</td>
<td>6.9 ± 2.8</td>
</tr>
<tr>
<td></td>
<td>NC2</td>
<td>0.5 ± 1.4</td>
<td>63.6 ± 2.3</td>
<td>44.6 ± 3.0</td>
<td>6.4 ± 3.0</td>
</tr>
<tr>
<td>Right:</td>
<td>NC</td>
<td>0.7 ± 0.9</td>
<td>66.2 ± 2.5</td>
<td>45.4 ± 3.3</td>
<td>7.0 ± 1.9</td>
</tr>
<tr>
<td></td>
<td>p100</td>
<td>0.6 ± 0.7</td>
<td>66.3 ± 2.5</td>
<td>46.1 ± 3.2</td>
<td>7.1 ± 2.0</td>
</tr>
<tr>
<td></td>
<td>p60</td>
<td>0.7 ± 1.0</td>
<td>66.7 ± 2.1</td>
<td>46.4 ± 2.6</td>
<td>7.2 ± 2.3</td>
</tr>
<tr>
<td></td>
<td>p20</td>
<td>0.6 ± 0.7</td>
<td>66.4 ± 2.3</td>
<td>46.2 ± 2.7</td>
<td>6.8 ± 2.1</td>
</tr>
<tr>
<td></td>
<td>NC2</td>
<td>0.5 ± 0.9</td>
<td>66.3 ± 2.3</td>
<td>45.7 ± 2.7</td>
<td>7.4 ± 1.7</td>
</tr>
</tbody>
</table>

5.3.5 Movement variance

Long-term variance

In addition to the significant changes in timing, and after accounting for those changes by applying piecewise linear length normalization, there still remained several differences between correlations of angular kinematics from NC and the other conditions (Table 5.5). The left hip was significantly different than NC during p100 (p = 0.020), while the left knee was significant for both p100 (p = 0.012) and p20 (p = 0.019). Both the left (p ≤ 0.004) and right (p ≤ 0.009) ankles were significantly affected for all perturbation conditions (p100 through p20) and the open, “recovered,” condition (NC2). The whole-limb angles for both limbs were not significantly affected, nor were the right hip and knee joint angles.

Short-term variance

The short-term variances of angular data changed slightly due to the increased stiffness (Table 5.6). Test conditions had a significant effect on the left whole-limb angle (p = 0.048) and left ankle (p = 0.005), based on the repeated-measures ANOVAs.
The effects to the right limb that we saw in Chapter 4 when only comparing NC to p100 were not significant when considering all conditions together. Post-hoc tests determined that the left whole-limb angle variance was significantly increased during p100 \((p = 0.010)\), while the ankle was significantly lower for p100 \((p = 0.001)\) and p60 \((p = 0.032)\). The other comparison of interest was the whole-limb angle versus the individual joint angles within each condition. The repeated-measures ANOVAs indicated significant differences between angle variances for all conditions bilaterally \((p \leq 0.001)\). Post-hoc \(t\) tests found that in all cases except for the left ankle during p100 \((p = 0.063)\), the whole-limb angle variances were significantly less than the hip, knee, and ankle joint angle variances \((p < 0.001)\).

Table 5.5: Long-term variance group averages (±SD) of cross correlations between NC and p100, p60, p20, and NC2 \(r_{\{x,y\}}\) for whole-limb, hip, knee, and ankle angles. Self correlation of NC \(r_{\{x(o),x(e)\}}\) is given as reference. The null hypothesis that the group mean of \(r_{\{x,y\}} \geq 99.82\) was tested with one-sided \(t\) tests, where * denotes the result is significantly lower than 99.82 \((\alpha = 0.05)\).

<table>
<thead>
<tr>
<th></th>
<th>Whole-limb</th>
<th>Hip</th>
<th>Knee</th>
<th>Ankle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>p100</td>
<td>99.96 ± 0.03</td>
<td>99.69 ± 0.17*</td>
<td>99.44 ± 0.44*</td>
<td>93.61 ± 2.48*</td>
</tr>
<tr>
<td>p60</td>
<td>99.96 ± 0.02</td>
<td>99.83 ± 0.09</td>
<td>99.68 ± 0.32</td>
<td>95.60 ± 2.32*</td>
</tr>
<tr>
<td>p20</td>
<td>99.97 ± 0.02</td>
<td>99.78 ± 0.09</td>
<td>99.70 ± 0.16*</td>
<td>97.06 ± 1.85*</td>
</tr>
<tr>
<td>NC2</td>
<td>99.98 ± 0.01</td>
<td>99.92 ± 0.08</td>
<td>99.83 ± 0.09</td>
<td>99.18 ± 0.06*</td>
</tr>
<tr>
<td>(r_{{x(o),x(e)}})</td>
<td>99.99 ± 0.00</td>
<td>99.95 ± 0.03</td>
<td>99.96 ± 0.03</td>
<td>99.83 ± 0.14</td>
</tr>
<tr>
<td>Right:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>p100</td>
<td>99.94 ± 0.04</td>
<td>99.78 ± 0.12</td>
<td>99.82 ± 0.13</td>
<td>99.03 ± 0.62*</td>
</tr>
<tr>
<td>p60</td>
<td>99.96 ± 0.04</td>
<td>99.87 ± 0.13</td>
<td>99.85 ± 0.10</td>
<td>99.19 ± 0.68*</td>
</tr>
<tr>
<td>p20</td>
<td>99.95 ± 0.04</td>
<td>99.84 ± 0.15</td>
<td>99.85 ± 0.08</td>
<td>99.35 ± 0.41*</td>
</tr>
<tr>
<td>NC2</td>
<td>99.98 ± 0.01</td>
<td>99.89 ± 0.13</td>
<td>99.84 ± 0.13</td>
<td>99.36 ± 0.27*</td>
</tr>
<tr>
<td>(r_{{x(o),x(e)}})</td>
<td>99.99 ± 0.01</td>
<td>99.96 ± 0.04</td>
<td>99.98 ± 0.02</td>
<td>99.82 ± 0.18</td>
</tr>
</tbody>
</table>
Table 5.6: Short-term variance group averages (±SD) of summed standard deviations for whole-limb, hip, knee, and ankle angles for all conditions. For each angle, condition effects were assessed with repeated-measures ANOVAs and post-hoc t tests, where * denotes statistical difference from NC (α = 0.05). Within each condition, † denotes joint angle values that are statistically higher than whole-limb angle.

<table>
<thead>
<tr>
<th></th>
<th>Whole-limb (deg)</th>
<th>Hip (deg)</th>
<th>Knee (deg)</th>
<th>Ankle (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>60.4 ± 11.3</td>
<td>111.5 ± 27.0†</td>
<td>145.4 ± 25.9†</td>
<td>105.0 ± 19.5†</td>
</tr>
<tr>
<td>p100</td>
<td>70.3 ± 15.6*</td>
<td>116.4 ± 35.0†</td>
<td>154.1 ± 41.3†</td>
<td>82.1 ± 19.0*</td>
</tr>
<tr>
<td>p60</td>
<td>62.1 ± 10.6</td>
<td>107.2 ± 28.7†</td>
<td>149.5 ± 29.4†</td>
<td>91.0 ± 21.4*†</td>
</tr>
<tr>
<td>p20</td>
<td>65.1 ± 15.8</td>
<td>105.1 ± 32.5†</td>
<td>150.5 ± 38.4†</td>
<td>104.3 ± 42.2†</td>
</tr>
<tr>
<td>NC2</td>
<td>60.1 ± 8.6</td>
<td>108.3 ± 23.9†</td>
<td>152.6 ± 25.8†</td>
<td>109.9 ± 28.1†</td>
</tr>
<tr>
<td>Right:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NC</td>
<td>58.7 ± 8.9</td>
<td>105.3 ± 30.7†</td>
<td>152.9 ± 46.2†</td>
<td>105.0 ± 30.1†</td>
</tr>
<tr>
<td>p100</td>
<td>61.9 ± 12.3</td>
<td>113.1 ± 27.2†</td>
<td>164.8 ± 48.4†</td>
<td>122.6 ± 38.6†</td>
</tr>
<tr>
<td>p60</td>
<td>59.8 ± 11.7</td>
<td>108.7 ± 27.3†</td>
<td>154.3 ± 38.2†</td>
<td>109.5 ± 23.7†</td>
</tr>
<tr>
<td>p20</td>
<td>60.1 ± 17.0</td>
<td>107.6 ± 25.5†</td>
<td>156.7 ± 43.5†</td>
<td>116.0 ± 53.7†</td>
</tr>
<tr>
<td>NC2</td>
<td>59.4 ± 10.3</td>
<td>110.5 ± 26.9†</td>
<td>159.5 ± 41.1†</td>
<td>113.0 ± 27.0†</td>
</tr>
</tbody>
</table>

5.4 Discussion

The results of this study indicate that the kinematic and kinetic compensation strategies during the full perturbation condition (p100) generally return to normal as the ankle stiffness perturbation is reduced (p60 through NC2). Various compensations throughout the gait cycle return to normal at different rates, with certain minor differences remaining through removal of all stiffness (NC2). Further, we see that the hypothesis of maintained limb-level control is confirmed throughout recovery from the ankle perturbation. Please refer to the corresponding subsections of Sec. 4.4 for expanded discussion of the perturbation effects during p100. The current section details the changes to these compensations during recovery, transitioning from the full perturbation (p100) back to normal (NC2).
5.4.1 Gait events

Stride time significantly increased from NC to p100, where subjects most likely tried reducing step frequency as a compensation to the added stiffness (cf. Sec. 4.4.1). After the initial perturbation, we see that stride time approached the NC value (Table 5.1), indicating subjects were able to overcome the perturbation enough to maintain their normal step patterns after the first reduction in stiffness. In all cases, stride time was symmetric between limbs, but this can be an artifact of treadmill walking. It should be noted that the statistical significance in p20 was close to the limit of $\alpha = 0.05$, and clinically was not much different than p60, except for a smaller group standard deviation.

Left weight alignment ($WA$) and knee-ankle alignment ($KAA$) were significantly delayed during the p100 condition (Table 5.2), indicating subjects had difficulty progressing through mid-stance and mid-swing, respectively (cf. Sec. 4.4.1). Delayed $WA$ suggests the AFO stiffness prevented the body from rotating over the ankle normally. Delayed $KAA$ was most likely caused by increased hip and knee flexion (Fig. 4.8ac), indicating subjects were not confident in their foot clearance during swing. Both events returned close to normal at p60. The overall restriction to ankle kinematics was about halved between p100 and p60 during loading response, as indicated by reduced deviation values (Fig. 5.1d). This reduction in perturbation allowed the body to enter mid-stance in the normal configuration, reducing the delay during this phase, which ends at $WA$. The return of left $KAA$ to normal indicated that subjects were no longer unsure of their foot clearance after reaching the p60 perturbation. This finding suggests that the delay, and corresponding changes in kinematics, were a result of subjects not being able to move their ankle during swing. The sensation of being able to move it again (even slightly) may have given subjects more confidence in their foot clearance.
The right limb experienced early contralateral toe-off (CTO). Based on the discussion in Sec. 4.4.1, it seems subjects were loading their right limb faster, or that the ankle plantarflexion restriction during push-off did not allow the full range of left ankle motion, causing it to lift early. This behavior persisted through all three perturbation levels, returning to normal at NC2. The fact that this behavior lasted longer than the timing effects seen in the left limb suggests that the perturbation to the left limb’s loading response was easier to compensate for than the lack of full push-off motion. Finally, we note that toe-ankle alignment (TAA) was significantly delayed in the right limb through all perturbation levels as well, indicating an expanded initial swing phase, but a shorter mid-swing. In the previous chapter, we postulated that delayed right TAA further supports the evidence above that the left limb was delayed through mid-stance (expanded right initial swing), with the subjects attempting to land the right foot early to compensate during terminal swing. This behavior for the right limb lasted longer than the corresponding left-limb behavior based on the statistical results, but could be due to the larger standard deviations across the group for the left limb WA.

5.4.2 Compensations throughout the gait cycle

As discussed in the previous chapter, the left ankle was the joint most affected by the added AFO stiffness (cf. Fig. 4.5e), particularly in the following ways during p100 (Table 5.2 and Fig. 5.1b):

1. Reduced plantarflexion during loading response.

2. Prolonged mid-stance.

3. Reduced plantarflexion during pre-swing (push-off) and throughout swing.

The specific compensations to each of these restrictions during p100 were detailed in Sec. 4.4.2, and will be discussed below in terms of changes to their regions of deviation.
results during reduction in ankle stiffness (Figs. 5.1–5.4).

**Reduced plantarflexion during loading response**

To summarize the perturbation’s effects on loading response during p100 (cf. Sec. 4.4.2), subjects exhibited increased ankle dorsiflexion (Fig. 5.1b) and dorsiflexor moment (Fig. 5.4a), which they tried to counteract with decreased biological dorsiflexor moment (Fig. 5.4b) and decreased (and delayed) TA activation (Tables 5.3 and 5.4). The biological ankle moment compensation could not fully counteract the perturbation at p100. Additional compensation was made by increased hip (Fig. 4.8a) and knee (Fig. 5.1a) flexion, allowing normal timing through loading response. Hip and knee extensor moments were significantly higher in the latter half of loading response (Figs. 5.2a and 5.3a), suggesting kinematic compensation during impact, followed by kinetic compensation for the more crouched posture. Left VL activation also increased (Table 4.3), confirming this behavior. During reduction in stiffness, we see that the left ankle kinematics (Fig. 5.1bdf) and kinetics (Fig. 5.4) return gradually, but do not reach normal levels until NC2. Conversely, the ipsilateral compensations at the hip and knee generally disappear by p60 (Figs. 4.8a, 5.1ace, 5.2ace, and 5.3ace). This result aligns with the finding that left WA returns to normal by p60. Clearly, the stiffness in p100 creates a limitation in ankle plantarflexion that the subjects chose not to (or were not physically able to) overcome solely with modified ankle moments. Instead, they chose a kinematic strategy that helped absorb the impact at heel strike and in early loading response. Then, after the first reduction in stiffness, subjects stopped employing the hip and knee strategies during this phase. This is a clear indication that changes in neuromuscular adaptation are occurring at different rates throughout the limbs, based on the level of perturbation.
**Prolonged mid-stance**

A majority of mid-stance was unaffected kinematically during p100, but kinetic compensations were present, attempting to correct the delay (cf. Sec. 4.4.2). In particular, the biological left ankle moment showed significantly less plantarflexion (Fig. 5.4b), and the left knee exhibited a significantly higher extensor moment (Fig. 5.3a) during mid-stance. The ankle adaptation was a response to delayed left $WA$, as less plantarflexion would allow the limb to roll over the ankle quicker. The knee adaptation was perhaps also a compensation for delayed $WA$, or to correct the increased knee flexion during loading response. Throughout recovery, the biological ankle moment regions of deviation became gradually narrower, but the knee moment showed no regions of deviation at $p60$ or later. This finding parallels with the discussion in Sec. 5.4.1 that the effects to loading response into mid-stance were overcome with only ankle adaptations after p100.

**Reduced push-off during pre-swing**

Push-off during pre-swing was also affected by the added ankle stiffness, where the AFO limited the full range of ankle plantarflexion, shortening the time the ankle could propel the body (cf. Sec. 4.4.2). Subjects then adapted their hip and knee joints to compensate for the reduced ankle motion before the restriction actually occurred (Figs. 4.8a and 5.1a), confirming the presence of adapted motor behavior rather than just reactive compensation. After this compensation, a second adaptation occurred, where the left hip extensor moment decreased just before ipsilateral toe-off ($ITO$) to help advance the limb into swing more quickly. Throughout the recovery, the plantarflexion restriction continually reduced (Fig. 5.1bdf). The increased extension of the knee immediately previous to this restriction remained, but based on deviation values it only changed between $p100 \rightarrow p60$ and $p20 \rightarrow NC2$. Alternatively, the corresponding hip extension adaptation returned to normal by $p60$. The pre-$ITO$ decrease
in hip extensor moment remained significant throughout the reductions, decreasing gradually in a similar manner to the ankle push-off restrictions. Like the loading response adaptations, we saw variable rates of change between different compensations for the restricted push-off, including evidence of discontinuity in the knee adaptation relative to the changes in perturbation.

After push-off, the left ankle remained dorsiflexed greater than NC throughout the entire swing phase during p100 (Fig. 5.1b). Subjects exhibited increased biological plantarflexion moments during this time (Fig. 5.4b), suggesting they may have relaxed their ankle, allowing the AFO stiffness to hold the foot in place. Moving from p100 to NC2, the increased dorsiflexion angle diminished, but remained significant until NC2 (Fig. 5.1bdf). The compensations seen in the biological ankle moment also diminished gradually. However the moments returned to normal during mid-swing at p60, indicating that perhaps subjects could not rely on the AFO stiffness to ensure toe clearance at this point (Fig. 5.4bdf).

**Right limb compensations**

While the right ankle was significantly affected during p100, regions of deviation were not present, which was discussed in Sec. 4.4.2. However, the changes in kinetics of the right limb give insight into the adaptations necessary to maintain right whole-limb motion. The right ankle moment decreased in late loading response (Fig. 4.9f), helping the limb roll forward over the ankle, perhaps compensating for the reduced push off and early toe-off of the left limb. This compensation was aided by an increased extensor moment at the right hip, further helping progression through early stance (Fig. 5.2b). The right ankle plantarflexor moment was also reduced during terminal stance, which would slow forward speed upon left limb impact (Fig. 4.9f). Concurrently, the right knee experienced a decreased extensor moment, aiding in the reduction of speed at left impact (Fig. 5.3b). As the subjects recovered from the per-
turbation, the right ankle moment returned to normal at p60, while the hip moment adaptations diminished smoothly in loading response. While not mentioned in the previous chapter due to limited regions of deviation during this phase, the knee extensor moment decreased in loading response, actually having more deviations in p60 than p100 (Fig. 5.3b,d). This behavior suggests that the compensations shifted from the ankle to the knee during p60. In terminal stance, the knee moment adaptations were not significant during p60, but may have returned for p20 (Fig. 5.3d,f), again providing evidence of discontinuous control adaptations during recovery.

5.4.3 Movement variance

Long-term variance

In Chapter 4 we observed that the neuromuscular control system successfully maintained the same patterns of whole-limb motion between NC and p100, resulting in similar gross body motion during gait (cf. Sec. 4.4.3). This limb-level consistency was achieved through compensations to the individual joint angles, as detailed in Sec. 4.4.2 and reviewed above. Further, whole-limb motion was maintained throughout the recovery from initial perturbation (Table 5.5). The hip kinematics were statistically indistinguishable from NC after p100, which agrees with the regions of deviation results during those conditions. However, the knee kinematics were indistinguishable only for p60 and NC2, but not for p20. This path of return to normal knee kinematics is also seen slightly in the regions of deviation, where deviations in pre-swing and terminal swing are greater for p20 (Fig. 5.1e) than p60 (Fig. 5.1c). Interestingly, once all other joint kinematics returned to normal, there were still significant differences in the left and right ankles. As seen in many of the specific compensation strategies detailed in Sec. 5.4.2, after a certain amount of stiffness reduction, subjects may be able to utilize kinetic adaptations and/or ankle-only strategies to overcome
the perturbation.

The evidence in this study suggests that by most accounts, subjects returned to normal gait behaviors during NC2. However, bilateral long-term variance based on the cross-correlation results still remained in the ankle kinematics (Table 5.5). No significant regions of deviation remain to explain the residual long-term ankle variance during NC2. Similar to the right ankle during p100 (cf. Sec. 4.4.3), this result suggests that residual compensations to the bilateral ankle kinematics during NC2 were not consistent across all subjects. Perhaps another explanation could be that the ankle least affects whole-limb motion since it is the most distal joint. Therefore, the full return of normal ankle kinematics may be a low priority for the CNS. In either case, the result shows some form of remaining bilateral compensation after full recovery from the left ankle perturbation.

**Short-term variance**

In addition to the overall changes to the kinematic patterns described above, changes in short-term variance provide insight into the repeatability of these signals throughout the conditions (Table 5.6). Decreased variance across the gait cycle indicates that the kinematic pattern is more consistent throughout the trial. This consistency suggests that the neuromuscular control system is focusing on the variables with lower variance (the goal), while allowing more freedom in the variables with higher variance (the uncontrolled manifold). In conjunction with the long-term hypothesis, here it was assumed that the whole-limb angle would have lower variability than the individual joint angles, which was confirmed throughout all conditions. It was also hypothesized that this variability would not increase for whole-limb angle, but would increase for joint angles, in the presence of increased ankle stiffness. This hypothesis was not confirmed, as discussed below.

While the left whole-limb angle exhibited less short-term variance than the cor-
responding joint angles, the changes in variance between conditions differed from the stated expectation. As discussed in the previous chapter, the decrease in left ankle stride-to-stride variance at $p_{100}$ was most likely due to the physical restriction of the added ankle stiffness. This resulted in an increased variance for the left limb. However, this value was still significantly lower than left hip and knee variances. In all other cases, whole-limb variance was significantly lower than all individual joint angles. The effect on left whole-limb variance subsided before $p_{60}$, whereas the decreased ankle variance lasted through $p_{60}$. Either the subjects were able to compensate for the decreased ankle variance during $p_{60}$ elsewhere in the left limb, or it had increased enough until it no longer affected whole-limb variance. Also discussed in Sec. 4.4.3 were the increased right knee and ankle variances. However, when comparing all conditions with ANOVAs, these differences were no longer statistically significant.

5.4.4 Summary and further thoughts

The results of this study show a clear indication of active bilateral kinematic and kinetic compensation strategies to maintain successful gait in the presence of increased ankle stiffness. Further, we see that the whole-limb kinematics were preserved across the group. Significant changes to the average left hip and knee, and right ankle patterns provided the long-term compensation for subjects to maintain the correct limb-level behavior. These compensatory relationships support our hypothesis that a major goal of gait is the motion of each limb as a whole. Further, that the cycle-to-cycle variance is minimized for the whole-limb behavior versus individual joint angles supports the argument that the goal-equivalent compensations are carried out at the expense of the individual joint angles. Please refer to Sec. 4.4.4 for further discussion on the overall compensations during $p_{100}$.

The above discussion focused on the changes in compensation strategies through-
out the simulated recovery from injury (p100→NC2). Overall, there were several left ankle restrictions apparent during p100 which led to various compensation strategies throughout recovery:

1. Reduced plantarflexion function during initial contact and loading response, resulting in kinematic and kinetic compensations. Here, the restrictions to ankle motion and net moment gradually recovered throughout the experiment, along with compensations to biological ankle moment and TA activation levels. However, TA activation timing and kinematic compensations to the hip and knee were only present during p100. Between p100 and p60 subjects recovered enough function to abandon the kinematic adaptations they developed during loading response, relying solely on modified ankle kinetics from that point forward.

2. Decreased biological ankle moment during mid-stance, resulting in kinetic compensations. We saw that the ankle moment recovered gradually, while the corresponding increase in knee moment was only present for p100. Early disappearance of the knee adaptation was most likely also related to the recovery of normal WA timing by p60, while the ankle adaptation was required throughout recovery to aid in stance progression of the left limb.

3. Reduced plantarflexion and push-off function during pre-swing, resulting in kinematic and kinetic compensations. Here, the reduced ankle plantarflexion also gradually recovered until NC2, as did the corresponding increase to knee extension, and the following hip extensor moment compensation to aid in lift-off. However, the increased hip extension during push-off was only present during p100. Combined with the left WA event returning to normal by p60, this suggests that the added hip extension was not needed to gain momentum for the swing phase after p100.
4. Mostly-unchanged right limb kinematics, due to altered kinetic patterns. During loading response, the ankle and hip adaptations gradually returned to normal during recovery, whereas the knee moment adaptation actually increased at \( p_{60} \). Also, terminal stance exhibited adaptations to ankle and knee moments which disappeared before \( p_{60} \).

In general, it seems that left-limb timing was only affected through \( p_{100} \), and the resulting adaptations to these effects disappeared in subsequent conditions. The remaining effects tended to remain until full recovery at \( NC_2 \). While there were no remaining significant regions of deviation at \( NC_2 \), the cross-correlation results did show overall changes to bilateral ankle behavior. This could indicate that subjects arrived at different residual compensation behaviors once the physical restrictions to left ankle motion were completely removed. Clearly the steady-state adaptations developed by the subjects did not all change at rates comparable to the changes in ankle perturbation. This finding validates our original hypothesis regarding changes in injury compensations to gait during recovery, at least to the extent that this protocol has replicated “continuous” recovery.

Further work could certainly be performed to solidify these findings. For instance, it is possible that certain subjects rely on different compensation strategies than others, which could be masking some of the compensations when assessing the group behaviors. Many more subjects would be needed to fully carry out a more detailed analysis of different compensation strategies. Also, this chapter centered around the hypothesis that “discontinuous” changes to injury compensations were possible during continuous healing of an injury. Here we only considered three discrete “healing” steps due to limitations on protocol length. A study comprised of a more continuous perturbation recovery would place much greater stress on the subjects, as well as much more data processing and analysis, and is unlikely to be successfully completed. Finally, there may be issue with the amount of time subjects were allowed to
acclimate to each perturbation level. Short of having subjects wear the AFO for several days, we must satisfy ourselves with the assumption that subjects indeed settled onto an at-least short-term steady-state strategy that would not change much with further exposure time. Please refer to Sec. 4.4.4 for further discussion on some of the limitations and future work of the experiment.

5.5 Conclusion

This study extends the analysis and discussion in Chapter 4 by considering the changes to the compensations during reduction of the initial perturbation. Overall limb behavior was maintained throughout the recovery, but not all compensations to the various ankle motion restrictions returned to normal at a rate comparable to the stiffness changes. In general, timing effects diminished after initial reduction in ankle stiffness. Other compensations, centered around ankle function and maintaining this return to normal gait timing, recover gradually along with the perturbation. The results in this chapter suggest that changes in injury compensations do not necessarily return to normal continuously as the injury heals, and may not return to the healthy condition.
Chapter 6

Conclusions

6.1 Conclusions

When faced with acute musculoskeletal injuries, the human neuromuscular control system adapts to the imposed restrictions on motion. In some cases, this adaptation results in alternate compensation strategies that linger after the injury heals. In general, there is a lack of understanding of how the altered kinematic and kinetic patterns due to injury are correlated with the underlying neuromuscular control compensations creating them. This dissertation was focused on exploring and explaining these control adaptations to injury during gait, and the potential discontinuous and coexisting longitudinal neuromuscular control paths throughout recovery from injury, through a combination of dynamical-systems modeling and clinical experimentation. More specifically, the origins of such nonlinear phenomena were analyzed within the context of broken symmetry in simple dynamical-system models, while the clinical progression of adaptive changes in neuromuscular control were examined throughout incremental recovery from a simulated ankle injury.

In Chapter 2, the characteristics of corrective open-loop harmonic excitation schemes in response to symmetry faults, and in the presence of small nonlinearities during self-healing of such faults, were analyzed via numerical methods and perturbation analyses. The analyses demonstrated the existence of unique excitation conditions for a class of linear systems that ensured a response with particular symmetry properties and desirable response and excitation amplitudes, even in the
case of a symmetry-breaking fault. In the special case of the two-degree-of-freedom coupled oscillator, the analysis further documented the deterioration of performance that resulted with the introduction of nonlinearity. The sources of this deterioration of performance were investigated, including the coexistence of distinct branches of control strategies and a jump from one branch to the other while healing from a symmetry fault.

Chapter 3 extended the linear theory presented in Chapter 2 to a more general class of periodic excitations, determined how this excitation affected the symmetry-compensation strategy, and discussed the implications of its performance on human gait and motor control. It was found that unless the system satisfied certain symmetry properties, square-wave excitation (or any general periodic signal except for single-harmonic sinusoids) could not produce symmetric oscillations. However, choosing the lowest-order symmetry-compensation strategy minimized asymmetry just as effectively as an optimization routine for the given numerical example. In the context of clinical studies, it seems quite plausible that adjustments to CPG timing, phasing, and amplitude could be the primary mechanism for reducing asymmetry during prolonged injury or perturbation in gait.

Chapters 2 and 3 focused on modeling and analysis of dynamical systems with key characteristics found in the control of gait. Specifically, compensations to symmetry degradation of the oscillatory behavior of the models in those chapters were formulated and analyzed under the effects of nonlinearity and multi-harmonic periodic excitation. However, the simplicity of the chosen models leaves only an abstract connection to human gait. Chapter 4 was concerned with discovering and analyzing compensations to injury during human gait, through a simulated recovery from ankle injury, in the context of neuromuscular controller goals. Overall, the hypothesized goal that limb-level kinematics are preferentially conserved during gait was confirmed, even after addition of the ankle perturbation. Individual joint angle kinematics and
kinetics adapted to keep the whole-limb angle consistent, and more repeatable than the joint kinematics themselves.

Chapter 5 extended the analysis and discussion from Chapter 4 by considering the changes to the compensations during reduction of the initial perturbation. Overall limb behavior was maintained throughout the recovery, but not all compensations to the various ankle motion restrictions returned to normal at a comparable rate to the stiffness changes. In general, timing effects diminished after initial reduction in ankle stiffness. Other compensations, centered around ankle function and maintaining this return to normal gait timing, recovered gradually along with the perturbation. The results of Chapter 5 suggest that changes in injury compensations do not necessarily return to normal continuously as the injury heals.

The modeling aspects of this dissertation provided in-depth analyses of broken symmetry in mechanical oscillators, bringing to light the effects of small nonlinearities and multi-harmonic excitations on compensation strategies during recovery from the symmetry fault. Further, the experimental studies extended these abstract findings into clinical relevance by detailing compensations during an experimentally simulated recovery from injury. In each case, results suggest clinicians should consider the possibility that multiple compensation strategies can achieve the same motor goals, and further that these compensations can follow different paths during recovery.

6.2 Future Work

In regard to the dynamic systems modeling work from Chapters 2 and 3, there are several areas to extend beyond the coverage in this dissertation. The analysis methods in Chapter 2, numerical continuation and multiple-scales perturbation analysis, are limited in their effectiveness with physically more realistic systems. However, considering fault adaptations of more realistic systems, perhaps even modeling ac-
tual functional tasks affected by musculoskeletal injuries, would provide a wealth of knowledge directly applicable to clinicians. More realistic models of the central nervous system would enhance these results as well. It would also be interesting to use a local optimization routine to solve the nonlinear system’s control problem (similar to Chapter 3), rather than rely on the linear symmetry conditions.

Although the results of Chapter 3 had significant implications when considering central pattern generators and their role in maintaining gait symmetry, this work could be extended to consider nonlinearities as in Chapter 2. More thought would have to be placed on how exactly to replicate the full analysis, as there are no true “linear symmetry conditions” when considering the bang-bang excitation. In this case, perturbation analysis may prove extremely difficult or impossible to complete. However, numerical continuation could be used on the nonlinear system with bang-bang excitation in a similar manner as the single-frequency excitation. Further work might also consider yet other periodic signals than the square wave used in Chapter 3, with more degrees of freedom to satisfy controller goals. Finally, addition of feedback terms to the control strategy would be interesting to explore as well. Feedback would most likely return symmetry to the asymmetric square-wave system, and might provide further insight into how the CPG interacts with sensory feedback mechanisms in the body.

In regard to the experimental work from Chapters 4 and 5, there are several things that could enhance this dissertation’s findings, along with areas to expand these ideas outside of a simulated recovery from injury. Perhaps the largest area for further work is in expanding the current studies by adding additional subjects. Enhancing the subject population would allow for more definitive statistical results, as well as an increased ability to detect multiple compensation strategies between subjects. An expanded study with more “recovery” steps would increase the ability to assess control changes during a more “continuous” injury recovery, rather than the
three steps used in this dissertation due to limitations on experimental protocol time.

Outside of the current simulated injury protocol, similar experiments could be performed to test other aspects of compensation and rehabilitation. Many of the adaptation studies listed in Sec. 1.3 could be repeated with a similar systematic reduction in perturbation as that performed here. This would allow a more complete understanding of neuromuscular compensations to a range of different perturbations. Work could also be extended to populations suffering from neuromuscular disorders. Rather than testing compensations to injury, this work would test the adaptability of impaired central nervous systems. Those results, in combination with the results presented here for a healthy population, could provide key insights into the underlying mechanisms of neuromuscular disorders. Finally, all of the discussed experimental analyses could be repeated to study the real-time adaptations immediately after changes in perturbation. This would provide more understanding on the abilities to develop the compensation strategies discussed in this dissertation.
References


