Central pattern generator based control for human lower limb movements with functional electrical stimulation

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Central Pattern Generator Based Control For Human Lower Limb Movements With Functional Electrical Stimulation

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School of Electrical & Electronic Engineering

A thesis submitted to the Nanyang Technological University
in fulfilment of the requirements for the degree of
Doctor of Philosophy

2007

Statement of Originality

I hereby certify that the work embodied in this thesis is the result of original research and has not been submitted for a higher degree to any other University or Institution.

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Date

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Zhang Dingguo

To my parents.

Acknowledgements

Firstly, I would like to show my deepest gratitude to my supervisor, Dr. Zhu Kuanyi, for his valuable advice, warm encouragement, and intelligent guidance. He has given me a great deal of intellectual freedom. Without his patience and help, I would not accomplish this thesis successfully. Moreover, he also supports me much in the daily life as a good mentor.

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Summary

This thesis aims to study central pattern generator (CPG), and model its key mechanisms to design a control system for the human lower limb movement control via functional electrical stimulation (FES). The neural control mechanism of biological motor system is borrowed, which is the kernel of this work.

A full musculoskeletal model for the lower limb movement is adopted, which contains eighteen muscles and seven skeletons. The important properties of muscle such as contraction property, activation dynamics and muscle fatigue, are all considered in this model.

In this thesis, much work is devoted to the neural control mechanism, i.e. the central pattern generator (CPG) and neural oscillator. The CPG is a complex neuronal circuit in the spinal cord of vertebrate, which contributes much to the rhythmic movement. Neural oscillator is a simplified form of CPG, which is used widely in bio-inspired robot control. Simulation is performed to study the properties of the CPG and neural oscillator, which reveals the stable oscillatory behavior, network mechanism, parameter characteristics, sensory feedback effect, etc. In addition, a thorough theoretical analysis is carried out on the neural control mechanism. In the analysis, some mathematical methods, such as Lyapunov stability, piecewise linear analysis, describing function, *poincaré* mapping, are adopted. The analysis will provide firm foundation for the FES control system design.

Another important work is the FES control system design. Here, the neural control mechanism in the biological system is transformed into engineering application. In the control system, the CPG plays an important role and the adaptive mechanism is developed for it. In the meantime, some artificial control techniques such as neural network control and fuzzy logic control are incorporated. Therefore, it can be deemed a hybrid control system. Moreover, several types of sensory feedback are

integrated to endow the control system with adaptivity and robustness.

Three types of lower limb movements, swinging, cycling and walking, are tested on this proposed FES control system.

For the FES swinging movement, this neural network control system has demonstrated the satisfactory tracking performance with less tracking time. Especially, in this control system, the effect of tonic input is added into the CPG, which can endow the control system with robustness and strong adaptive ability, so that a strong resistance against the measurement noise can be ensured and it is able to track the movement with varying speed. This is an important contribution.

For the FES cycling movement, as an evaluation of the control performance, tracking the desired trajectories and maintaining the crank speed are both considered. In the design of the CPG, a fully connected CPG is developed, and mechanical coupling of CPG is built for the coordinated movement of the bilateral limbs.

For the FES walking movement, several important components are combined together to accomplish this delicate and fascinate task. For example, the CPG provides the approximate rhythmic pattern, the regulator and impedance controller aim to reduce the tracking errors, and the posture controller designed is to maintain the body balance during walking. In fact, this complete control system can also be deemed a complex hybrid FES control system reflecting the natural human motor system creditably. New algorithms are developed in the design of RBF neural network for the regulator. A fuzzy logic controller is designed to serve for the posture control. Also several types of sensory feedbacks are integrated for tuning this neural network control system. Simulation study demonstrates that the musculoskeletal system can perform smooth and accurate locomotion and maintain the posture well with this newly developed FES control system.

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Chapter 1

Introduction

1.1 Motivation

Human movement is a magic phenomenon that has attracted the interests of various researchers for a long time. It seems a simple task, but in fact, it combines the knowledge from several areas such as neuroscience, biomechanics, biomedical engineering and control engineering. A better understanding of the human motor system will gestate more good ideas to develop new artificial control systems for human movement.

In this interdisciplinary area, many researchers have made different contributions. Neuroscientists try to find out how the nervous system controls the movement of human body. Biomechanical scientists focus on the coordination of muscles and skeletal dynamics. Control engineers learn from the biological motor control system in order to design desirable human movement control systems and better bio-inspired robot. Biomedical engineers are eager to transfer the theories into clinical applications for the patient with physical disabilities, e.g. “Functional Electrical Stimulation” (FES), introduced by Moe and Post [131], which aims to stimulate

muscles electrically in a controlled way in order to restore as much motor function as possible for the patients with partial paralysis.

At present, in the world, there are thousands of people who lose motor functions due to spinal cord injury (SCI), stroke, traumatic brain injury and so on. For example, in USA, there are about 250,000 SCI patients with 10 times the number of stroke patients. It is an urgent and challenging task to develop a technique for these patients to restore some motor functions. It is known that FES is a promising technique that has been developed for about 40 years. With the help of some traditional artificial control methods, FES is successful in some clinical applications, such as knee joint movement. However, for restoring more complex motor functions, it still has a long way to go. Perhaps, cloning the biological motor control mechanism for FES system is a feasible way or at least a good attempt and it is the main motivation of this work.

Human beings use the lower limbs most frequently such as in standing, swinging, cycling, and walking, and these motor functions are especially important for the paraplegic, therefore, in this thesis, we focus on the FES control of the lower limb movements.

1.2 Synopsis

The biological motor system (CPG) is studied. Based on CPG, a new FES control system will be developed to control three types of the lower limb movements: swinging, cycling and walking. This control system transforms some mature biological theories into engineering practice, and this promises both chances and challenges, because there is not an intelligent machine that can be compared with human motor system in the world at present.

A complete musculoskeletal model of lower limbs is adopted for the simulation study, which contains eighteen muscles and seven skeletons. The muscle model is an improved version of Hill-type muscle model, and the key characteristics are all considered in this model, especially, the muscle fatigue is identified via our own experiments.

The neural control mechanism is mainly derived from the network structure existed in the spinal cord, central pattern generator (CPG). It has the ability to provide rhythmic movement patterns for the invertebrate and vertebrate. However, for the practical application of FES control, we use the simplified CPG model, neural oscillator, to accomplish this task as it is robust and easy to tune the parameters under a controllable configuration.

Some mature artificial control methods are also adopted in this work to assist the FES control system design. They are artificial neural network (ANN) and fuzzy logic control (FLC). The ANN aims to mimic the other neuronal circuit except for the CPG in the spinal cord, while the FLC aims to maintain the posture during walking. An integration of different sensory feedbacks is also incorporated in this FES control system, for example, the overall speed sensor, noise sensor, foot touch sensor and a simplified vestibular system that senses the central of gravity of the segment for the posture control.

Therefore, this FES control system can be deemed a hybrid system which contains both biological and artificial control mechanisms. Simulation studies have demonstrated satisfactory performance with the proposed FES control system and it has the potential for the clinical application in future.

1.3 Contributions

The major contributions of the thesis are listed as follows:

- The neural mechanism of biological central pattern generator (CPG) and neural oscillator is studied. Some subtle new properties of neural oscillator are established, and quantitative analysis through simulation is accomplished, especially the effect of the sensory feedback and tonic input. The sensory feedback of foot touch will entrain the CPG and generate the out-of-phase patterns for two legs. It will also trigger the posture controller during stance phase in walking. The tonic input with high intensity has the ability to resist the noise and generate more robust rhythmic motor patterns.
- Theoretical analysis of CPG and neural oscillator is performed and new results are established. A further exploration of the neural control mechanism based on the engineering perspective is conducted. Some important properties are achieved such as the determination of frequency, conditions of oscillation, local stability, and network property. These theoretical analyses have laid a firm foundation for the FES controller design in my work.
- A hybrid FES control system, which borrows the biological motor control mechanism, is developed. This could be a pioneer work in this area. Auxiliarily, some artificial control methods such as artificial neural network, fuzzy logic control and impedance control are also investigated to mimic the natural human control mechanism. The several types of controller are combined together to accomplish the task of lower limb rhythmic movement control via FES.
- An integration of different sensory feedbacks is incorporated, which mainly provides the adaptive ability for the CPG. This contribution is at the front

edge in this area at present, because such a nice and smart adoption of the integration of different sensory feedbacks for CPG is seldom considered in the previous literatures

- The versatility of this FES control system for knee joint movement, cycling and locomotion control is successfully demonstrated. Based on the simulation study, biomechanical analysis such as the contribution and coordination of individual muscles, is carried out.
- Both angular control and cadence control is considered in the FES cycling design. Both rhythmic control and posture control is considered in the FES walking design.

1.4 Outline of the Thesis

The rest parts of the thesis are organized as follows:

Chapter 2 gives a comprehensive literature review about FES, human motor control system, musculoskeletal model, and control techniques applied in human movement.

Chapter 3 is concerned with construction of a detailed musculoskeletal model of lower limbs, which serves as a plant for the FES control system in the simulation study. Dynamic behaviors of the model are also discussed.

In **Chapter 4**, the mechanism of CPG and neural oscillator is investigated and their parameters and physical meaning are presented.

Chapter 5 provides theoretical analysis for CPG and neural oscillator which are presented in Chapter 4. New theoretical results on CPG about boundness, oscillation, stability and determination of frequency are established.

Chapter 6 presents the FES control system design. The main idea comes from CPG

discussed in Chapter 4 and Chapter 5. The purpose of the FES control system is to help the lower limb paralyzed patients to perform certain movements such as leg swinging, cycling and walking. To verify its effectiveness, simulation study is performed for the lower limb movements.

Finally, the summary, conclusion and future work of my research are given in **Chapter 7**.

Chapter 2

Literature Review

Functional electrical stimulation (FES), model theories and control techniques are combined together in this research area. A comprehensive literature review about them is given in this chapter.

2.1 Functional Electrical Stimulation

People will lose some motor functions due to spinal cord injury, brachial plexus injury, stroke, multiple sclerosis, and traumatic brain injury. Functional Electrical Stimulation (FES) has generally been thought of as a promising technique to stimulate the peripheral nerve of the muscles in order to restore the motor functions of the muscles (or generate desired movements). Therefore, it should not only be deemed a simple method of therapy to prevent the atrophy of muscles. This technique is firstly initiated by Moe [131], and improved much by Kralj [103].

From a macroscopic point of view, FES is not only used to stimulate the skeletal muscle, it can also be applied to the cardiac muscles and smooth muscles of the human inner organs. In fact some FES techniques have been developed to pro-

vide bladder and bowel control, and to improve sexual function in disease or after traumatic injury [92]. However, the term FES is more or less reserved for providing functions for inner organs. Here FES is used as the traditional term that only serves for the skeletal muscles.

FES has many ways to stimulate the peripheral nerves in the muscle according to different interfaces. For example, surface electrodes, intramuscular electrodes, epimysial electrodes, cuff electrodes, intraneural electrodes and so on [145], [199]. The surface stimulation uses the adhesive electrodes that are placed on the surface of the subject's skin (above the location of the desired muscles) [98]. This method has been used for a long time since the early nineteenth century, but it was only for the therapy at the beginning. Compared with the other methods, the surface stimulation is easy to be performed and does not need extensive operation skills, so it is used most widely in the FES control system. However, changes in the surface conditions of the skin and differences in positioning electrodes can lead to variability in stimulation characteristics. Therefore, there are many works today in improving the surface electrodes. Intramuscular electrodes can be injected using a hypodermic needle through the closed skin nonsurgically, or through an open incision. Especially, the implantation is relatively easy with a percutaneous lead that can be inserted with a hypodermic needle. Epimysial electrodes must be placed surgically, which are placed below the skin, on the surface of a muscle. An advantage of epimysial electrodes over intramuscular electrodes is that they are less prone to mechanical failure and less likely to move after the implantation. Cuff electrodes encircle the nerve trunk. They have either tube or spiral configuration. As the cuff electrodes circumvent a nerve and their selectivity aims at fascicles more than at nerve fibers, they can afford appropriate muscular selectivity, power efficiency, and reduce the risk of infection compared to the penetrating stimulation. It is likely that the implanted electrodes will have a good prospect, but some features are desired such as robustness, reliability, chronic implantation, biocompatibility, safety and

low cost [164]. Intraneural electrodes are positioned to penetrate the epineurium around the nerve trunk, thus, the current and charge requirements for stimulation are very low compared with externally placed electrodes. They are not only applied for central nervous system, but also for peripheral system at present.

The application of FES can be divided into two main areas: upper limbs and lower limbs.

With regard to the lower limbs, the earliest work was for correction of the drop foot by Liberson et al. [117] in 1961. They proposed application of FES to the common peroneal nerve to correct the drop foot and a foot-switch was used to synchronize the application of electrical stimulation to the swing phase of gait. The device they developed is subsequently referred to as a peroneal stimulator or drop foot stimulator. This technique is very mature now [123]. The other applications in the lower limbs include knee joint movement [19], [45], [67], [93], [151], cycling [20], [86], [144] standing up [29], [162], keeping body balance [85], and walking [99], [103], [148], [198]. FES has made great progress in the first two applications, and many good results are achieved from real experiments on the subjects [19], [86], [93], [151]. Especially, the two research groups of Hunt and Petrofsky have made much contribution to the FES cycling separately. However, with regard to the later three motor task, most of the work is still at the simulation stage.

The FES application of the upper limb has not been widespread compared with the case of lower limbs. In the application of the upper limb, much attention is paid to the FES hand grasping [4], [26], [70] rather than elbow [149] or arm movement [111], [135].

Various commercial products of FES system have been developed. For the lower limbs: Parastep [13], [147], RGO [172] and the system designed by Kralj, et. al [104]. For the hand grasping: Freehand [70], Handmaster [135], Bionic Glove [154], FES-mate [183] and RPS [146]. More comprehensive introductions about these systems

are found in [100], [146]. However, so far most of the products are not very popular in the market. There are many reasons, and perhaps the cost/benefit reason is the most important one. Reliability, functionality, safety, and convenience of the current products should also be improved.

2.2 Model Theories

A thorough understanding of the structure and function of the complex motor system has challenged biologists and engineers. Yet, this challenge is worth embracing for both biomedical and engineering purpose. Better knowledge of the functional structure of the motor system can help us to effectively treat disease or injuries affecting posture and movement control. Conversely, learning how the motor system controls the complex movements and adapts to changing tasks and environmental perturbations will help engineers design better control systems.

Generally there are two ways about human motor system modeling. The biologists focus on developing new hypotheses and qualitative models for the system based on experimental results; while the engineers aim towards providing quantitative models (i.e. mathematical or analytical models) based on experimental findings and using engineering tools such as system analysis and control theories. These models are useful for testing the hypotheses, posing critical questions and improving our understanding about the motor system, designing new experiments, and predicting motor behavior under a variety of specified or imposed conditions.

According to the direction of my work, the literature review will be focused on three parts: 1. Overview of human motor system; 2. Muscle model; 3. Skeletal dynamics.

2.2.1 Overview of Human Motor System

Human motor control is extremely complex. In a simple word, the central nervous system (CNS) processes sensory information to determine a suitable movement trajectory, and it organizes the individual muscles and skeletons to perform the desired trajectory. We cannot touch every details of the whole motor control system. Here, we only provide a brief introduction. There are three main parts in the motor system: central nervous system (CNS), the sensory feedback and the musculoskeletal system. A simplified neurophysiological model of the human motor system is shown in Figure 2.1. The basic idea is helpful to develop the final FES control system in my work.

The CNS related with the motor control comprises of spinal cord and higher motor centers in brain such as associative cortex, motor cortex, cerebellum, basal ganglia and brainstem. The cerebellum is specialized for supervised learning based on the error signal encoded in the climbing fibers from the inferior olive. The basal ganglia are specialized for reinforcement learning based on the reward signal encoded in the dopaminergic fibers from the substantia nigra. The cortex is specialized for unsupervised learning based on the statistical properties of the input signal [35]. The higher centers are responsible for determining the goal and overall direction of movements as well as providing motor commands for the spinal cord. The spinal cord comprising of neural integration units is a main pathway from higher center to peripheral nerves and also can generate some simple movements by itself.

Among the motor centers, motor cortex, cerebellum, and spinal cord have been studied more extensively. Here, a brief discussion is given as follows.

Motor Cortex: The motor cortex integrates information from different sensory sources and memory, makes decision, and generates motor command. Electrical stimulation of the precentral of the cerebral cortex, the motor cortex, can generate

Figure 2.1: A simplified model of human motor system. Only basic components are given.

movements. This suggests direct involvement of the motor cortex in the control of movements. Primary motor cortex (M1) and somatosensory cortex (S1) are the most important parts in the motor cortex. M1 has direct physical connections that can influence the spinal cord for generating the muscle contraction. S1 is responsible for detecting the presence and magnitude of the sensory feedback. It receives the sensory signal from mechanoreceptors in the skin, muscles and joints.

The experimental results show that the motor cortex is involved in the execution of the movement rather than in its planning [160]. Research evidence [51] also shows

that the motor activity is probably related to two important control parameters of motion: force and direction.

Cerebellum: The cerebellum is an important part of the brain, which contains over half neurons of the brain. The function of the cerebellum has been studied extensively. Generally, it integrates the sensory feedback with the ongoing motor command to correct movement errors and adapt to changes in the motor system. It is also important for the correct timing of movements.

The cerebellum does not involve the initiation of voluntary movements and its processing of sensory feedback is not necessary for conscious perception. So the cerebellum is properly deemed as a modulatory role and its function is for integration and coordination.

Although the cerebellum is not involved in motor activities directly, it plays a very important role for movement co-ordination as well as for “long-term” and “short-term” predictions [84], [180]. It uses the incoming sensory feedback information, makes continual adjustments during the ongoing movement to ensure accuracy and smooth movement. Its acquisition of performance skills is achieved with practice. Some models have been developed for the cerebellum: a comparator for detection of the movement error [180], an internal model of the musculoskeletal system [97], adaptive filter [50], and a regulator for joint stiffness [171].

Spinal Cord: The spinal cord is the main communication link between the brain and the peripheral nerves. It carries the command from the brain resulting the muscle’s contractions to initiate the movements. It also can deal with the sensory feedback from the body organs or external environment.

However, the spinal cord is not just a simple pathway for transmitting motor commands and sensory information; it is in charge of descending control and reflex control. Examples of the basic spinal units are reflex and central pattern generator

(CPG). A reflex is a relatively stereotyped response to a specific sensory stimulus. Spinal reflexes are important for protection of muscles, limbs, and the body against sudden external disturbance, and for coordination of the relevant muscles in different voluntary movements. There are several reflex modalities, and sensory information plays the major role in reflexes. An excellent review is given by A. Prochazka et al. [152]. The central pattern generator (CPG) in the spinal cord can generate the desired motor pattern for rhythmic movement such as locomotion in the absence of both sensory and descending input. Many researchers have proposed CPG models, and the literature review about CPG will be given in Section 2.3.2.

Sensory Feedback: In human motor system, the sensory information is sent back to the CNS to regulate the human movement. The human natural sensors related to movements are cutaneous receptors, proprioceptor, visual and vestibular systems. Cutaneous receptors are important in connecting the body with the environment and objects, for example, the cutaneous receptors of foot can sense the ground reaction force in walking. The proprioceptor is a type of receptor located in the muscles, tendons, ligament and skeletal joints. The proprioceptor is a mechanoreceptor and it is activated by physical distortion. The function of the proprioceptor is to collect information about the position and movement of the limbs and send back this information to the CNS [5]. Generally, proprioceptors in joint capsules and ligaments appear to generate information on joint position and joint movement. While in muscles, they include muscle spindles that are sensitive to muscle length and changes of length, Golgi tendon organs that are sensitive to muscle tension [129]. The visual system provides information about the location and movement of our own body segments with respect to the environment. The vestibular system provides information about the head movement, and it is important for the maintenance of postural stability.

The lowest level of the human motor control system is muscular-skeletal system,

which is studied comprehensively in detail. The muscle modeling is a very active research area and a broad survey is presented in the next section.

2.2.2 Muscle Model

In the area of FES, muscle model serves as an actuator. The muscle model should have the characteristics of comprehension and prediction of the force generated under stimulation. Moreover, the credibility and tractability is desired. Primarily there are three classes of muscle models according to the level of the structure: (1) microscopic models; (2) fiber models; and (3) macroscopic (whole muscle) models.

Microscopic models are further divided into (i) “conventional” cross-bridge models (Huxley-type models) of the type first introduced by A. F. Huxley [89], and later elaborated by T. L. Hill [76] and many other researchers; (ii) “unconventional” cross-bridge models, where the postulation made is significantly different with those in Huxley-type models.

Macroscopic models are further divided into three subcategories:

(i) Hill-type models, which are developed on the prototype model proposed by A. V. Hill in his classic “1938-paper” [75]; Various models are further developed on the Hill-type model [39], [168], [205].

(ii) Viscoelastic models, which consider muscle to be a viscoelastic material.

(iii) System models, which regard muscle as a “black box” or “grey box” [12], [36], [55], and the input and output data are used to determine the inner property without considering the detailed structure or mechanism in the “box”. This technique is used widely in control engineering named as system identification.

In fact there is no definite borderline amongst the models mentioned above. They share some common ground more or less. For example, the system model is so

general that it could subsume the viscoelastic and Hill-type models, and Hill-type model can also be deemed formally as a special class of the viscoelastic model.

So far, the two muscle models, Huxley-type microscopic model and Hill-type macroscopic model, are widely used. The former has superior credibility as it catches many physiological details, while the latter is much more tractable because it generalizes some key features. The distribution moment (DM) model [203] is a bridge between the microscopic and macroscopic models.

2.2.3 Body Dynamics

The dynamics of human body is complex as it contains many muscles and skeletons. However, if the muscle dynamics is not considered, the skeletal dynamics can be computed with the methods used widely in robotics, e.g. Newton-Euler method etc. Most simulations of the human lower limb movement are performed in two dimensions (in the sagittal plane). It is because of the following reasons. Firstly, flexion and extension represent the major movements at most joints, so they contribute most significantly to performance in most motor tasks. Secondly, planar models with hinge joints have fewer degrees of freedom (DOF) than that of three-dimensional models. Therefore, simulations based on planar models are easy to be realized and generally take less computation time than three-dimensional models.

The simplest planar form of body dynamics model is one-segment model that is just like a pendulum. This type of model is used widely for the knee joint control [19], [45], [93], [118], [151]. Generally, in these works the thigh is assumed to be fixed, and the shank can be looked on as the pendulum. One-segment model is also served for the ankle joint control [85], [200].

Two-segment model is a very common form. For example, it is used for arm movement control, in which the two segments are represented as upper arm and fore-

arm [107], [111], [179]. With regard to the leg movement control, they represent the thigh and shank [95], [108], [148]. The two segments can also roughly model the upper body and lower body for the standing control [126].

The most popular model is the three-segment model. This model is used to represent, for example, the human arm (upper arm, forearm and hand) [17], leg (thigh, shank, and foot) [116] and whole body (upper body, thigh and shank) [99], [106], [162] in the two-dimensional plane. In addition, the complex four-segment model is also found in the body balance control [72].

There are some complex models that have more than four DOFs. These models mainly serve for the whole body movement such as walking (locomotion). The classic work is initiated by Hatze [64], [66], and his model contains 17 segments and 42 DOFs. Another important work is Taga's model that contains 8 segments and 20 muscles [182]. Because these models have complex structures and have too many DOFs, it is hard to control them in reality via FES. So far they are only suitable for computer simulation and the experimental test is still faraway. For example, Yamaguchi used 8-DOF model of the body to simulate the full gait cycle [198]; Anderson used a three-dimensional, 10-segment, 23-DOF model of the skeleton to simulate one cycle of normal walking [7].

Regardless of whether the body dynamics is modeled in two or three dimensions, and no matter how many DOFs it has, the relationships between the forces applied to the body and resulting motion of the body segments can always be expressed in this form:

$$M(\Theta)\ddot{\Theta} + C(\Theta)\dot{\Theta}^2 + G(\Theta) + L_{ma}(\Theta)F_m + E(\Theta, \dot{\Theta}) = 0 \quad (2.1)$$

where $\Theta, \dot{\Theta}, \ddot{\Theta}$ are vectors of the generalized coordinates (joint angles), velocities, and accelerations; $M(\Theta)$ is the system mass matrix and $M(\Theta)\ddot{\Theta}$ is a vector of inertial

torques; $C(\Theta)\dot{\Theta}^2$ is a vector of centrifugal and Coriolis torques; $G(\Theta)$ is a vector of gravitational torques; F_m is a vector of muscle forces, $L_{ma}(\Theta)$ is the matrix of muscle moments, and $L_{ma}(\Theta)F_m$ is a vector of muscle torques; $E(\Theta, \dot{\Theta})$ is a vector of external torques applied to the body by the environment.

If the number of DOFs of the skeletal model is very large, a more efficient computing method is desired to handle it. A number of special software packages are available for this, including AUTOLEV by On-Line Dynamics Inc, SD/FAST by Symbolic Dynamics Inc, ADAMS by Mechanical Dynamics Inc and so on.

2.3 Control Methods

In this section the control methods applied in human movement control and FES application will be discussed. Engineers aim to design a controller that can replace certain functions of the biological motor system. In fact, the biological structure represented in Figure 2.1 has been transformed to a hierarchical model by many researchers [72], [102], [110], [111]. A generalized functional three-level motor control scheme is shown in Figure 2.2. This hierarchical control structure has many advantages such as adaptivity, modularity, easiness of application, and possibility of integration into a man-machine system.

2.3.1 Control System Overview

From a control engineering point of view, the musculoskeletal system is deemed a plant to be controlled. The positions and velocities of body segments are the plant states. The muscles that power the system are actuators. The signals driving the actuators (muscles) are controls (e.g. neural pulse trains in intact humans, or electrical stimulus in FES systems). The controller is the process by which the

Figure 2.2: Hierarchical model of motor control system.

controls are generated (e.g. the CNS in the intact human, or the artificial controller in the FES systems). The time history of the plant states in response to the control signals is referred to as the system trajectory (e.g. the joint-angle trajectory).

Controllers can be designed to function without sensors and therefore without knowledge of the actual plant trajectory (open loop control), since the trajectory information is not fed back to the controller. Reference-based open-loop controllers precompute and store (a priori to task execution) the muscle stimulation patterns that will, hopefully, execute the desired motor task. The muscle stimulation patterns were developed by combining clinical experience with trial-and-error, and then stored in the controller. Though it is easy to be applied in experiments [103], the performance of any open-loop control system will probably be inadequate, since disturbance always exists, which will cause performance to deviate significantly from that required for proper task execution. For example, from walking on uneven and rough surface;

from unexpected actuator performance, such as muscle fatigue. Open-loop control is a primitive and simple method. Even in the absence of the disturbance, the open loop control will probably be inadequate, since musculoskeletal properties have never been fully understood or perfectly modelled.

To resist disturbance and remedy muscular modeling mismatch, a feedback controller with ongoing knowledge of the effects of disturbance must be designed. Sensors (e.g., joint-angle sensors) provide measurements from which the current state of the system (e.g., the actual joint angles and velocities) can be estimated. These estimates are fed back to the controller, resulting in a feedback, or closed-loop control system. Closed-loop controllers are sometimes referred to as error driven, since they respond to the trajectory error. Error-driven systems are particularly well suited for accurate and slow movements such as tracking a trajectory [3], [19], [45], [93], [151], [162], moving at a constant velocity [20], [86], or postural control.

The closed-loop control has many advantages for FES systems. (1) A closed-loop control system can automatically compensate for small disturbances and consequent trajectory errors. (2) It can regulate the system behavior more precisely than open-loop control. (3) It lets the designer specify the system response characteristics. In summary, the closed-loop control lets designer develop systems with a level of performance and flexibility better than that with an open-loop control.

To control tasks where trajectories must change rapidly but predictably (such as walking), reference control is sometimes employed in addition to error-driven feedback. These controllers utilize feedforward controller to generate an approximate trajectory [3], [105]. A feedback controller is employed in-parallel to correct for trajectory errors resulting from disturbance and modelling errors. If the body deviates too far from the desired trajectory, however, the stored controls may actually interfere with the recovery being provided by the feedback controller. This situa-

tion can arise because the reference-based controller functions independently from the feedback controller and cannot adjust its stored stimulation patterns during the task.

A more robust system for motor control could be developed with model-based control [45]. The model-based controller utilizes sensors and a dynamic model of the system to continuously recalculate the best trajectory to complete the desired task. During the movement, for the body to follow the desired path, the model-based controller can be designed with the inverse dynamics. In this case, if the body deviates from the desired path, the model-based controller calculates a new path and generates a new muscle stimulation pattern. Though this sounds attractive, the complexity and computational requirements of model-based control continue to limit its application to the real problem.

2.3.2 Advanced Control Technology

The study on human movement and FES engineering expands rapidly in recent years and attracts more and more engineers' attention from the control and system areas. Various control methods, such as model reference control (MRC) [45], [67], model predictive control (MPC) [119], [187], adaptive control [18], [27], fuzzy logic control (FLC) [29], [72], iterative learning control (ILC) [37], H_∞ robust control [85], sliding mode control [93], data-driven control [151], and nonanalytic control (NAC) [186] have been investigated for FES control system design. The excellent review work on this aspect can also be found in [145] and [153].

In fact, there is not a clear classification for these methods in application, and these control methods (or algorithms) are sometimes combined to accomplish one task. Therefore, it is appropriate to call them as the hybrid control methods, for example, proportional-integral-derivative (PID) as an auxiliary method is added to a neural

network control system [3], [19], and to an optimal control system [109].

Particularly, optimization and neural network (NN) control methods are used most widely in the human movement control, and we will discuss them in the following parts.

Optimization Method

Because the number of muscles crossing a joint is greater than the number of degrees of the freedom (DOFs) specifying joint movement, the force developed by each muscle can't be determined uniquely. It's hard to provide each muscle with the appropriate stimulation pattern, so optimization theory is an appropriate choice to solve this problem [7], [87], [106], [143].

There are two optimization methods in terms of the dynamics, inverse and forward optimization [140]. If the dynamic model of human movement is developed, it can be used to determine the quantities that may not be easily measured. For example, if the muscle forces could not be measured noninvasively, they can then be determined using inversive or forward dynamics methods. In the inverse dynamics method, the noninvasive measurements of body motions (position, velocity, and acceleration) and the external forces are used as the inputs to calculate the muscle forces. However, in the forward dynamics method, the muscle excitations (or the muscle activations) is used as the inputs to calculate the corresponding body motions.

In fact, the inverse dynamics (or static optimization) method [45], [69], [163] solves an optimization problem at each instant of the movement, but the forward dynamics (or dynamic optimization) method [7], [143], [155], [198] solves an optimization problem during one complete cycle of the movement. Compared to the dynamic optimization, the static optimization has its own shortcomings. First, it depends on the accurate record of the measurement instruments, specially the measurement of the positions, velocities, and accelerations of the body segment. However, at

present there are many difficulties to get accurate velocity and acceleration from the available instruments. Second, it is hard to include the muscle physiology in the formulation of a static optimization because of the same reason as above. Finally, the static optimization is a descriptive or analysis-based approach, so a model of the motor task cannot be included in the method.

The dynamic optimization is more powerful because of the following reasons. (1) The system equation of muscle physiology is easily incorporated in the formulation of the problem. (2) A model of the motor task can be included because the optimization is done over a cycle of the task. Though the two optimization methods are different in many aspects, they appear to give the same results for relatively low-frequency movements like walking.

Neural Network Control

In the early 1940s the primitive idea of neural network was introduced by some pioneers [73], [128], however, the rapid growth in neural network took place after 1970s with the classic work of Hopfield [80]. Neural network has now become very popular, especially in the control area, mainly due to: (1) its ability of mapping nonlinear systems; (2) its highly parallel structure that can perform a distributed processing; (3) its learning and adaptation ability; (4) its data fusion ability that means it can operate on quantitative and qualitative data simultaneously; (5) its ability in handling multivariable and complex systems.

In some applications, neural network components are used in a standard control system configuration, such as radial basis function (RBF) and multi-layer perceptron (MLP). However, in other applications, neural network components have been used in somewhat *non-standard* control system configurations, such as the configurations based on the structure of neurophysiological control systems, e.g. central pattern generator (CPG) [3]. This *non-standard* neural network is specially suitable for the rhythmic movement control.

Central pattern generator (CPG) is a neuronal network existing in the biological nervous system of vertebrate and invertebrate. When the CPG is activated, it can generate motor patterns for different rhythmic movements such as walking, breathing, flying, and swimming [124].

Many researchers in neuroscience have studied the CPG in recent years. E. Marder et al. studied the pattern generator that models the walk rhythmic pattern and dynamic clamp in the stomatogastric nerves system of the crab and lobster [124], S. L. Hooper's research group also made great contribution on the pattern generators of crustacean [78], V. A. Straub et al. developed the lymnaea feeding pattern generator [175] and S. Grillner et al. developed the swimming pattern generator of the lamprey [58], [59], [61], [63]. Accordingly, some researchers have modified the CPG into a relatively simple mathematical format: the neural oscillator [10], [125]. It is more useful and convenient in engineering applications, such as in robot and biped control [11], [43], [44], [46], [48], [89], [90], [111], [115], [133], [141], [158], [180], [193]. In fact, some hardware and software based on the structure and features of CPG are developed for the practical application [113], [169] and [173]. The *non-standard* network CPG is the kernel of the thesis, and much investigation will be performed in the following chapters.

With regard to the review on the standard artificial neural network (ANN) control, there are too many literatures to list them all. Here, I only focus on the work where ANN is applied into the FES control.

Lan, et al. [109] used neural network to control arm movements, which mapped a set of optimal kinematic trajectories into a set of stimulation parameters. This network utilized a off-line learning to determine the network parameters that can be then used in real-time specification of stimulation signals. This network has the ability to generalize the learned control from a limited set of movements to a broader set of scaled movements. Though this ability may be important for applications, it has

the potential disadvantage due to off-line learning.

Kostov, et al. [102] have used an adaptive logic network to control locomotion in paralyzed human subjects using FES. In their work, the network was used in a multi-state control configuration in which real-time measurements from sensors were used to trigger state transitions. Actually this method was obtained through addition of the feature of flexibility and adaptability into the method presented in the previous work. A shortcoming of this approach is that it does not adjust stimulation levels to fit for particular individuals.

Graupe, et al. [60] designed neural network for FES to facilitate patient-responsive ambulation. The proposed network discriminated above-lesion upper-trunk electromyography (EMG) time series to activate standing and walking functions under FES. As it can control FES stimuli levels using response-EMG signals, it is applicable to an untrained user and allows safe ambulation in the face of system errors.

Chang, et al. [19] used neural network and proportional-integral-derivative (PID) feedback to control the knee joint movement through stimulation of the quadriceps muscle. Here, a multi-layer feedforward time-delay neural network was trained and then was used as an inverse model of the FES induced quadriceps-lower leg system for direct feedforward control.

Tong and Granat [188] designed an ANN controller to clone the expertise of walking with hand-switches. This controller has three layers with five hidden nodes using a set of ten force sensors and another set of 13 “virtual” kinematic sensors. The continuous neural networks offered good FES controllers with significantly fewer oscillations.

Jonic et al. [95] compared three machine learning techniques in the human locomotion control. One of them is a radial basis function (RBF) neural network with orthogonal least squares learning algorithm, and it is combined with an entropy

minimization-type of inductive learning. The simulation results show some advantages of the RBF neural network over the other two.

In addition, an important work was initiated by Abbas [3], where the standard and non-standard neural network are combined. In his work an adaptive neural network was developed to control the leg cyclic movement, the structure of which is based on the hierarchical motor control system. The pattern generator of this system is a network based on the lamprey swimming circuitry to produce oscillatory signals. The pattern shaper is a Hebbian neural network with one layer. Later, Abbas and his research group made some improvement on this neural network system, and experiment was performed to test its performance [1], [156], [157], [174]. However, most of their work is focused on the feedback control (pattern shaper) instead of the pattern generator.

In order to have a clear view, the control techniques adopted recently in the human movement control are summarized in Table 2.1. It can be seen that most control techniques are only practical for the knee joint movement. For more complex movements with multi-joint and multi-degree of freedom, the current techniques seem incompetent and only simulation results are presented so far. This means it still has a long way to reach the stage of real experiments.

Control Methods	Authors	Task	Muscles&Skeletons (Number)	FES Experiment
PID control	Franken et al. [48] Lan et al. [108]	leg swinging hindlimb (cat)	3&2 4&2	✓ ✓
Model reference control	Hartwell et al. [67] Ferrarin et al. [45]	knee joint movement knee joint movement	2&1 1&1	✓ ✓
Model predictive control	Towhidkhah [187] Li et al. [119]	walking elbow joint movement	2&2 2&1	
Adaptive control	Riess et al. [157]	swinging	1&1	✓
Iterative learning control	Dou et al. [37]	knee joint movement	2&1	✓
Sliding mode control	Jezernik et al. [93]	shank movement	2&1	✓
Gain scheduling control	Previdi et al. [150]	knee joint movement	1&1	✓
Data-driven control	Previdi et al. [151] Petrofsky [144]	knee joint movement cycling	1&1 6&4	✓ ✓
H_2 (LQR, LQG) control	He et al. [74] Hunt et al. [88]	standing posture standing posture	10&3 2&2	✓
H_∞ (Robust) control	Hunt et al. [85]	standing posture	2&2	✓
Optimal control	Khang et al. [99] Lan [111] Popovic et al. [148] Anderson [7] & Pandy [140]	standing up arm movement walking walking	13&3 6&2 4&2 54&10	
Fuzzy logic control	Davoodi [29] Chen et al. [20]	standing up cycling	4 & 3 4&4	✓
Machine learning	Jonic et al. [95]	locomotion (walking)	4&2	
Neural network	Abbas et al. [1], [3] Chang et al. [19]	knee joint movement knee joint movement	2&1 1&1	✓ ✓
Central pattern generator	Taga [182] Zhang et al. [211]	locomotion (walking) locomotion (walking)	20&8 18&7	

Table 2.1: Control techniques for the human movement control.

Chapter 3

The Musculoskeletal Model

In this chapter, the musculoskeletal model of human lower limb intended for FES application will be illustrated. It serves as a control plant instead of a real patient. The muscle model is constructed based on the Hill-type muscle model, which contains the fundamental physiological characteristics of the muscle. It has exhibited a good compromise between complexity and tractability. The skeletal dynamics is modeled according to the traditional methods similar with that used in robotics. In the end, simulation study is performed on this musculoskeletal model.

3.1 Theoretical Methods

Before discussion on the muscle model, we should have a brief view about the real human skeletal muscle (Figure 3.1). The skeletal muscle is fixed to the bone by the tendon and fascia. The tendon plays an important role, so it is often combined with the muscle together, named as “musculotendon” [205]. Each muscle is made up of fascicles, and fascicle is made up of muscle fibers. There are different types of mysium (perimysium, epimysium, and endomysium) amongst the fascicles and fibers (Figure 3.1). Each muscle fiber has thousands of myofibril, and every section of the

myofibril is the typical sarcomere. The sarcomere has the famous structure “cross-bridge” (Figure 3.1(c)), which induces many important properties of the muscle [89].

Figure 3.1: Physiological structure of the muscle. In the lower figure: (a) a whole muscle. (b) muscle fiber. (c) myofibril (d) structure of sarcomere, which is made up of myosin and actin filament.

As mentioned before, the two primary functions of the muscle model, comprehension and prediction, are most important. Biological scientists are concerned with understanding how muscle works, whereas the engineers wish to predict its behavior as an element of a large control system. As this research aims to develop a human

musculoskeletal model for the biomedical engineering study (FES application), a simpler and more tractable model is expected.

In fact, the Hill-type model is used widely in the engineering application, and many researchers have made more or less revision on this model [25], [39], [168], [203], [205]. Zajac's version of Hill-type model is the typical one adopted widely by engineers [205]. The attractive feature of his model is “scaling”, which permits the calculation of the mechanical behavior of many different muscles using four simple characteristic quantities. The benefit of such simplicity seems to be worth the cost of some loss of fidelity in the representation of the complex muscle mechanics.

3.1.1 Muscle Contraction Dynamics

Figure 3.2: The typical Hill-type muscle model.

There are three elements in the mechanical muscle model shown in Figure 3.2. A contractile element (CE) that models the muscle's force-length-velocity property, a series element (SE) that models muscle's active stiffness, and a parallel-elastic element (PE) that models the muscle's passive stiffness. The instantaneous length of the actuator is determined by the length of the muscle, the length of the tendon, and pennation angle (omitted here) of the muscle. In this model, the width of the muscle is assumed to remain constant as the muscle length changes.

The active force produced by the muscle, F_a , is affected by four parameters: the peak isometric force F_{max} , the muscle length l , the muscle shortening or lengthening velocity v , and muscle activation a_m . The general form can be written as:

$$F_a = f(F_{max}, l, v, a_m) \quad (3.1)$$

For detailed computation, it is often defined as the product of F_{max} with three dimensionless quantities:

$$F_a = F_{max} * f_{fl} * f_{fv} * a_m \quad (3.2)$$

where f_{fl} is a normalized factor which describes the relationship between the muscle force and muscle length. The muscle active force strongly depends on the muscle length l_m . When l_m changes, it not only affects the maximum peak force produced by the muscle, but also the joint angles at which peak force occurs. Due to the cross-bridge structure of CE, it can be shown that the peak force occurs at the optimal muscle length l_{opt} . It is well known the force-length relationship is parabolic since Zajac's classic work [205]. There are various mathematic equations representing the force-length relationship. Three examples are given below, and equations (3.3), (3.4), (3.5) are adopted from [72], [138], and [162] respectively. It is obvious the relationship shown in (3.3) and (3.4) is not general and only suitable for a specific muscle. The general characteristic is desired for the muscle model, and the parameter identification is a formidable task.

$$f_{fl} = 0.32 + 0.71 \exp[-1.112(\bar{l} - 1)] \sin[3.722(\bar{l} - 0.656)] \quad (3.3)$$

$$f_{fl} = \exp\left[-\frac{|\bar{l}^{2.8286} - 1|^{2.3680}}{0.6182}\right] \quad (3.4)$$

$$f_{fl} = \exp\left[-\left(\frac{\bar{l} - 1}{\varepsilon}\right)^2\right] \quad (3.5)$$

where $\bar{l}$ is the normalized muscle length with respect to the optimal muscle length l_{opt} : $\bar{l} = l_m/l_{opt}$.

In my work, a Gaussian-function-like equation (3.5) is selected to represent the force-length relationship. The main advantage of this format is that it is simple with very few parameters and can be regulated for different muscles by the parameter ε . The identification of ε is achieved in [162].

The force-velocity relationship is described by the function f_{fv} , which also has various representations. Three examples are given in the following equations (3.6), (3.7) and (3.8), which are adopted from [138], [72], and [69] respectively. We can see that none is general. For different muscles, the differences of force-velocity relationship are not significant compared with force-length relationship, and there are nearly no research publications providing the full parameter identification for different muscles. Therefore, the parameter-fixed force-velocity relationship was widely used in the previous research. In my work, equation (3.8) is adopted.

$$f_{fv} = 1 + \tan(3.0\bar{v}) \quad (3.6)$$

$$f_{fv} = \begin{cases} \frac{1+7.3125\bar{v}}{1+4.0625\bar{v}} & \bar{v} > 0 \text{ (stretching)} \\ \frac{1+\bar{v}}{1-2.25\bar{v}} & \bar{v} < 0 \text{ (shortening)} \end{cases} \quad (3.7)$$

$$f_{fv} = 0.54\arctan(5.69\bar{v} + 0.51) + 0.745 \quad (3.8)$$

where $\bar{v}$ is the normalized muscle velocity with respect to the maximum contraction (shortening) velocity v_{max} of the muscle: $\bar{v} = v_m/v_{max}$.

The muscle length (l_m) and velocity v_m can be calculated from the joint angle (θ) and angular velocity ($\dot{\theta}$). In this case, the relationships are defined by [162]

$$l_m = C_m + \sum \int L_{ma}(\theta)d\theta \quad (3.9)$$

$$v_m = \sum \dot{\theta} L_{ma}(\theta) \quad (3.10)$$

where L_{ma} is the moment arm and C_m is constant.

3.1.2 Muscle Activation Dynamics

When the muscle tissue is stimulated by electrical pulses (neural drives or artificial electrical signals), an internal muscle tissue state (a_m , muscle activation) is generated. This process is associated with two factors, the release of calcium at the neuromuscular junction and then through the muscle contraction dynamics. This action energizes the cross-bridges, so that the muscle force is generated. Figure 3.3 shows the qualitative relationship between the electrical pulse and the muscle force generated.

Figure 3.3: Muscle response (force generation) to electrical signal.

The twitch response of a muscle to a single electrical impulse is divided into two parts, contraction and relaxation, with a latent period (time delay) (Figure 3.3(a)).

It can be modeled by a second-order, critically damped mechanical system, which is expressed as [39]:

$$T(t) = te^{-rt} \quad (3.11)$$

where $T(t)$ is the joint torque produced by the electrical impulse, t is the time, r is reciprocal of the time to peak torque. The strength of the twitch increases with the increase in the magnitude of the pulse. Therefore, the strength of the twitch is said to be graded (or incrementally increased). This is due to the recruitment of increased numbers of muscle fibers that are involved in the twitch. If you increase the frequency of the electrical pulses, eventually the second stimulus will be applied before the first muscle twitch is over, then the second stimulus will build upon the previous contraction and add to that response. This is called twitch summation as shown in Figure 3.3(b).

If a stream of electrical pulse is applied to the muscle, i.e. the pulse frequency is increased continuously, eventually no relaxation will be allowed and the muscle contraction will increase smoothly up to a point of maximum strength where individual twitches are not observed as they are fused. This is called tetany or tetanic contraction (see Figure 3.3(c)). Tetany is the way by which normal muscle contractions occur. In this situation, a steady muscle force is generated. Do not confuse this term with tetanus, a disease where muscles contract continuously due to bacterial infection and release of tetanus toxin which prevents the neuromuscular synapse from relaxing.

This process can be divided into several parts: IRC, frequency effect, Ca^{++} dynamics, muscle fatigue and time delay.

Isometric Recruitment Curve

In some models, it is assumed that there are two factors affecting a_m in equation (3.2). One is the effect of isometric recruitment curve (IRC), and the other is the

effect of stimulation frequency. The IRC is a very important characteristic of the muscle, which can be defined as the static gain relationship between the stimulus activation level and output force. When the term “isometric” is used in the context of muscle stimulation experiment, it generally means the experiment equipment holds muscle tendon at a fixed length, it does not mean the contractile element of muscle is held isometric [38], [39].

In FES application, when an electrical pulse is applied to a muscle, it activates, or recruits a certain number of motor units in the muscle fibers. When the stimulation intensity (i.e. pulse width or pulse amplitude) is varied, the number of motor units recruited varies, so does the amount of force generated by the muscle. The relationship between the stimulation level and the amount of force produced by the muscle is defined by isometric recruitment curve (IRC). It is typically nonlinear, and exhibits a deadband and a saturation. If the width of the electrical signal is regulated for stimulating the muscle, the IRC can be modeled using a piecewise continuous function with three segments [45]. In this case, the muscle IRC is looked upon as the relationship between the pulse width z_m and neural drive u , which is normalized between 0 and 1. Then, the IRC can be described by

$$u = \begin{cases} 0 & \text{for } z_m \leq W_d \\ \frac{1}{W_s - W_d}(z_m - W_d) & \text{for } W_d < z_m < W_s \\ 1 & \text{for } z_m \geq W_s \end{cases} \quad (3.12)$$

where W_d is a threshold pulse width (recruitment deadband), W_s is a saturation pulse width. Here, W_d is set as 100 μs and W_s is 500 μs .

Calcium Dynamics

It can be seen in Figure 3.3 that the muscle cannot be activated or relaxed instantaneously. The delay between muscle excitation and activation (or the development of muscle force) is mainly due to the time taken for calcium to pump out of the sar-

coplasmic reticulum, to travel down the T-tubule system and bind to troponin [140]. The relationship between muscle excitation $u(t)$ and muscle activation $a(t)$ is usually modeled as a first-order process [143], [205]. In fact, $u(t)$ can also represent the neural drive.

$$\frac{da(t)}{dt} = \frac{1}{\tau_{rise}}[u(t)^2 - u(t)a(t)] + \frac{1}{\tau_{fall}}(u(t) - a(t)) \quad (3.13)$$

where τ_{rise} and τ_{fall} are time constants. Note that in this model the frequency effect and muscle fatigue are not considered.

Property 3.1 The muscle activation $a(t) \in \mathbb{R}$, $\forall t \geq 0$ given in (3.13) is bounded if $u(t) > 0$ and has

$$a(t) \leq \frac{\tau_{fall}u(t)^2 + \tau_{rise}u(t)}{\tau_{rise} + \tau_{fall}u(t)} \quad (3.14)$$

Proof: Re-arranging equation (3.13), we have

$$\frac{da(t)}{dt} = \left[\frac{1}{\tau_{rise}} + \frac{u(t)}{\tau_{rise}} \right] \left[\frac{\tau_{fall}u(t)^2 + \tau_{rise}u(t)}{\tau_{rise} + \tau_{fall}u(t)} - a(t) \right] \quad (3.15)$$

Clearly if $u(t) > 0$, then $\tau_{rise} + \tau_{fall}u(t) > 0$ and $\frac{\tau_{fall}u(t)^2 + \tau_{rise}u(t)}{\tau_{rise} + \tau_{fall}u(t)} > 0$. Let (3.15) be 0, then we can get the maximum value of $a(t)$. Thus, the result (3.14) is obtained.

Property 3.2 Consider the first order differential equation (3.13), where τ_{rise} and τ_{fall} are the time constants. Assume $u(t) = u$ is constant, then the rise time is mainly determined by τ_{rise} , and the relax time is determined by τ_{fall} .

Proof: For a constant $u(t) = u$, we have the solution with the zero initial condition

$$a(t) = \frac{\tau_{fall}u^2 + \tau_{rise}u}{\tau_{rise} + \tau_{fall}u} \left[1 - e^{-\left(\frac{1}{\tau_{fall}} + \frac{u}{\tau_{rise}}\right)t} \right] \quad (3.16)$$

If $u = 1$, the response of $a(t)$ is mainly determined by $\frac{u}{\tau_{rise}}$ as it is higher. Therefore, τ_{rise} determines the rise time, i.e. the response time of $a(t)$ when u is increased.

However, when u is decreased to a very small value, for example, $u = 0$. Then, resolving (3.13) yields

$$a(t) = C_0 e^{-\frac{t}{\tau_{fall}}} \quad (3.17)$$

where C_0 is the initial value. Clearly, the transient behavior of $a(t)$ when it falls from an initial value is mainly determined by τ_{fall} . Therefore, the rising and falling behaviors of $a(t)$ can be separately characterized. In practice, the range is from $12ms$ to $20ms$ for rise time, τ_{rise} , and for relaxation time, τ_{fall} , it is from $24ms$ to $200ms$. The calcium dynamics is often deemed a low-pass filter. Sometimes, it can be determined by a second-order transfer function.

Frequency Effect

When the frequency of stimulation pulse varies, it also affects the force produced by the muscle. This effect is named as the force-frequency characteristic and can be defined by [163]

$$q(t) = \frac{(k_f f)^2}{1 + (k_f f)^2} \quad (3.18)$$

where f is the stimulation frequency, and k_f is the shape factor.

Muscle Fatigue

When stimulating the muscle electrically, the muscle fatigue can cause a drop in the peak muscle torque. It is shown that approximately 50% reduction occurs over 100 seconds [103], [161] under the full stimulation, and it may also cause a shift in the recruitment deadband. Generally, there are two factors that cause fatigue. One is due to the physiological factor, which is a decrease in the ability to produce force over time. The other is due to the psychological factor. When the brain is becoming bored or tired with an action, it no longer allows the body to perform a further action. Here, we only describe the muscle fatigue due to the physiological factor. It is mainly because of the inability to produce force due to a decline in the sensitivity of troponin to calcium. In this case, the strength of the muscle has been

exhausted and the muscle no longer responds to signals from the brain. Moreover, it is also found that the fatigue rate strongly depends on the stimulation frequency [33]. The function $p(t)$ is used to describe this property and it is defined by

$$\frac{dp(t)}{dt} = \frac{(p_{min} - p(t))a(t)\lambda(f)}{\tau_{fat}} + \frac{(1 - p(t))(1 - a(t))\lambda(f)}{\tau_{rec}} \quad (3.19)$$

$$\lambda(f) = 1 - \beta + \beta\left(\frac{f}{100}\right)^2 \quad (3.20)$$

where $a(t)$ is the muscle activation without muscle fatigue. τ_{fat} is the time constant of fatigue, τ_{rec} is the time constant of recovery, and p_{min} is the minimum fitness. As the fatigue is related with the stimulation frequency, the frequency factor $\lambda(f)$ is introduced. Note that the fatigue factor $p(t)$ in equation (3.19) is a variable without dimension, and it is set as a ratio to the normal muscle activation. In a word it is a general factor for the nine muscles in the lower limb in our work.

In conclusion, after the frequency effect and muscle fatigue are incorporated, the final activation of the muscle a_m is modeled as $a_m(t) = a(t)p(t)q(t)$.

Property 3.3 Assume the muscle activation is a constant $a(t) = a$, and the initial condition is $p = 1$ at $t = 0$. Then, solving (3.19) results in

$$p(t) = \frac{\tau_{rec}p_{min} + \tau_{fat}(1 - \frac{1}{a\lambda})}{\tau_{rec} - \tau_{fat}(1 - \frac{1}{a\lambda})} + \frac{\tau_{rec}(p_{min} - 1)}{\tau_{fat}(1 - \frac{1}{a\lambda}) - \tau_{rec}} e^{[a\lambda(\frac{1}{\tau_{rec}} - \frac{1}{\tau_{fat}}) - \frac{1}{\tau_{rec}}]t} \quad (3.21)$$

It can be seen that the muscle fatigue gain $p(t)$ decreases when a large muscle activation a , or a higher stimulation frequency f is applied. It is accordant with the phenomenon occurred during experiment. Furthermore, the simulation results in Section 3.2.1 will also confirm it.

From the above analysis, the full muscle model can be described in the following diagram shown in Figure 3.4.

Figure 3.4: Block diagram of the muscle model.

3.1.3 Body Dynamics

Before deriving the human body dynamics, we should calculate the moment applied to each joint. As we focus on the lower limb dynamics, three joints of each leg will be considered.

$$T_i = T_{a,i} + T_{p,i} + T_{w,i} \quad (3.22)$$

$$T_{a,i} = \sum_j F_{a,j} \times L_{ma,j} \quad (3.23)$$

$$T_{p,i} = T_{e,i} + T_{v,i} \quad (3.24)$$

where $i = 1, 2, 3$ indicates hip, knee and ankle joint. j indicates the index of the muscles acting on the specific joint. T_a is the torque produced by the active element of the muscle. T_p is the torque produced by passive element, i.e. connective tissue such as ligaments and passive muscles, which includes the viscous torque T_v and the elastic torque T_e . T_w is the torque produced by the muscle weight, however, it

is very small and often ignored in the literatures. L_{ma} is the moment arm for each muscle.

The viscous torque is given by a simple linear function:

$$T_{v,i} = K_{v,i}\dot{\theta}_i \quad (3.25)$$

where $i = 1, 2, 3$ indicates hip, knee and ankle joints respectively.

While the elastic torque is determined by a so-called double-exponential function [40],

$$T_{e,i}(t) = \exp(c_1 + c_2\theta_{distal} + c_3\theta_i + c_4\theta_{proximal}) - \exp(c_5 + c_6\theta_{distal} + c_7\theta_i + c_8\theta_{proximal}) + c_9 + T_k \quad (3.26)$$

where $i = 1, 2, 3$ indicates hip, knee and ankle joints. θ is the joint angle being investigated. $\theta_{proximal}$ is the angle of immediately proximal joint. θ_{distal} is the angle of immediately distal joint. Note that $T_k = \exp(c_{10} + c_{11}\theta)$ is not considered for hip and ankle joints, and it is added to the function when computing the knee angle. $c_1, c_2, \dots, c_{11}$ are elastic parameters. This equation is used to describe the three joints of leg: ankle, knee and hip, and the coefficients are given in Table 3.4.

Skeletal Dynamics

A typical musculoskeletal model of the lower limb is shown in Figure 3.5. It has four segments and nine muscles. In order to perform the different movements of the lower limbs, one-segment, three-segment and four-segment leg model are developed in the study. For example, the shank swimming movement can be deemed one-segment movement as only the shank moves in this case. The knee joint control is needed in this simple case. One-segment skeleton dynamics can be easily described as

$$T_{tot} = \left(\frac{1}{8}ml^2 + \frac{1}{2}I\right)\ddot{\theta} + \frac{1}{2}mgl\sin\theta \quad (3.27)$$

where T_{tot} is the total torque acted on the skeleton, m is the skeleton mass, I is the moment of inertia, θ is the joint angle, and g is the gravitational constant.

Figure 3.5: Musculoskeletal model of lower limb. The names of muscles are abbreviated in the contents. Gluteus maximus: GMAX, iliopsoas: ILI, vastus group: VAS, biceps femoris: BF, tibialis anterior: TA, soleus: SOL, rectus femoris: RF, hamstrings: HAM, gastrocnemius, GAS.

As an interim, we derive the two-segment model comprises two skeletons, thigh and shank, via Newton-Euler method [126], [148]. However, in my work two-segment dynamics is not used in the simulation study.

$$\begin{bmatrix} T_1 \\ T_2 \end{bmatrix} = \begin{bmatrix} H_{11} & H_{12} \\ H_{21} & H_{22} \end{bmatrix} \begin{bmatrix} \ddot{\theta}_1 \\ \ddot{\theta}_2 \end{bmatrix} + \begin{bmatrix} -h\dot{\theta}_2 & h\dot{\theta}_2 \\ h\dot{\theta}_1 & 0 \end{bmatrix} \begin{bmatrix} \dot{\theta}_1 \\ \dot{\theta}_2 \end{bmatrix} + \begin{bmatrix} G_1 \\ G_2 \end{bmatrix} \quad (3.28)$$

where: $H_{11} = I_2 + m_2 l_{c2}^2 + m_2 l_1 l_{c2} \cos(\theta_1 - \theta_2)$

$H_{12} = I_1 + m_1 l_{c1}^2 + m_2 l_1^2 + m_2 l_1 l_{c2} \cos(\theta_1 - \theta_2)$

$H_{21} = I_2 + m_2 l_{c2}^2$

$H_{22} = m_2 l_1 l_{c2} \cos(\theta_1 - \theta_2)$

$$h = m_2 l_1 l_{c2} \sin(\theta_1 - \theta_2)$$

$$G_1 = m_2 g l_{c2} \sin \theta_1 + (m_1 g l_{c1} + m_2 g l_1) \sin \theta_2$$

$$G_2 = m_2 g l_{c2} \sin \theta_1$$

T_1, T_2 : torques acted on the thigh and shank

θ_1, θ_2 : hip and knee joint angles respectively

l_1, l_2 : thigh and shank lengths respectively

l_{c1}, l_{c2} : the distances from (hip, knee) joints to the centers of gravity position

m_1, m_2 : thigh and shank masses respectively

I_1, I_2 : thigh and shank moments of inertia respectively.

Similarly, the dynamic equation of the three-segment (or more) can also be derived by Newton-Euler method. The detail is referred to Appendix. Such complex skeletal dynamics aims to serve for the simulation study of cycling and walking in Chapter 6.

3.2 Model Simulation

In this section, we will perform simulation on the musculoskeletal model. It is known that the experimental data for the musculoskeletal model are abundant in the literature [138], [155], [162], [193]. Anthropometric data for the body segment and muscles are listed in Table 3.1 and Table 3.2 respectively. The muscle parameters relating the activation and contraction property are shown in Table 3.3 ([31], [45], [82], [161], [162]), [163], particularly the parameters about muscle fatigue (τ_{fat} and τ_{rec}) are adjusted to match our own experiments. The coefficients relating the passive moment are listed in Table 3.4 ([40]).

Table 3.1: Anthropometric data for the body segment.

segment	length [m]	center of gravity [m]	mass [kg]	moment of inertia [kgm ²]
HAT	0.80	0.469	47.4	2.6967
thigh	0.50	0.244	10.0	0.2431
shank	0.45	0.279	3.5	0.0476
foot	0.20	0.15	0.83	0.012

Table 3.2: Anthropometric data for the nine muscles of lower limb. Notice: H=hip, K=knee, A=ankle, L_{ma} =moment arm.

muscles	$F_{max}[N]$	$l_{opt}[m]$	$v_{max}[m/s]$	$L_{ma}[m]$
GMAX	2370	0.11	0.54	0.092 (H)
IL	1850	0.146	0.73	0.132 (H)
VAS	5200	0.086	0.48	0.04 (K)
BF	400	0.173	0.69	0.049 (K)
TA	1100	0.099	0.36	0.023 (A)
SOL	3600	0.033	0.10	0.036 (A)
RF	1000	0.086	0.51	0.049 (H)
				0.025 (K)
HAM	2190	0.121	0.48	0.054 (H)
				0.049 (K)
GAS	1600	0.054	0.32	0.050 (K)
				0.040 (A)

3.2.1 Activation Dynamics

In FES application, there are two ways to modulate the intensity of the electrical pulse in the stimulation for the muscle. One way is to change the amplitude of the electrical pulse [19], [93]; the other is to vary the width of the electrical pulse [45], [85], [150]. The later method is adopted in this work. In this section, simulation study on activation dynamics is performed, and arbitrarily rectus femoris (RF) is selected. Note that without any other specific illustrations on the muscle, it indicates RF in this section. In fact, activation dynamics is designed in a general form in the work, and it has the same values for all the muscles concerned.

It is known that the muscle has no response to generate force if the pulse width is too small, and the muscle's response will keep stable if the pulse width reaches an

Table 3.3: The parameter values of muscle activation dynamics.

$\tau_{fat}[s]$	$\tau_{rec}[s]$	p_{min}	$u_d[us]$	$u_s[us]$	k_f	β	time delay [s]
18	30	0.2	100	500	0.1	0.6	0.025

Table 3.4: The coefficient values of muscle passive element.

joint	viscous	elastic								
	K_{vis}	c_1	c_2	c_3	c_4	c_5	c_6	c_7	c_8	c_9
	[Nms/deg]									
ankle	0.011	2.35	0	-0.085	-0.021	-7.8	0	0.19	0	-1.6
knee	0.018	1.3	-0.06	-0.06	0.0477	0.5	0	0.017	-0.005	3.9
hip	0.035	1.0	-0.003	-0.09	0	0.9	-0.033	0.04	0	8

upper limit. If the pulse width keeps increasing, it would do harm to the muscle. Experiments show that the acceptable range for the pulse width is between 100 *us* and 500 *us* [45], [85], [150]. The pulse width within this range is used to stimulate the muscle. The amplitude of the electrical pulse is fixed 25 *mA* and frequency is fixed at 20*Hz*. The result is shown in Figure 3.6. Note that the activation level normalized to 100% is increased when the pulse width (*PW*) becomes large. As fatigue property is not considered, there is no drop of the activation level after it reaches the saturation area (tetanic contraction). Each curve is accordant with the qualitative properties of the muscle, such as latent period (time delay), IRC and so on.

The stimulation frequency f also has effect on the muscle activation level, which is defined as pulses per second (*pps*). The contribution of pulse width and pulse frequency to the muscle activation is shown in Figure 3.7, where the fatigue factor is considered. Without considering the fatigue, a high stimulation frequency can make the muscle produce a large force. However, a high frequency can also cause a strong muscle fatigue. So the stimulation frequency should not be too high. The suitable range for the frequency is within 20 *pps* and 40 *pps*.

The important property, muscle fatigue, has been incorporated into the muscle

Figure 3.6: Muscle activation with different stimulation intensity (pulse width). The pulse amplitude is fixed at 25 mA , and the pulse frequency is fixed at 20 Hz .

Figure 3.7: Effect of pulse frequency and pulse width on the muscle activation.

model. It is actually combined with the muscle activation, and consequently the force produced by the muscle will decrease as time goes on. The experiment has shown when the stimulation frequency is increased, the force produced by the muscle will drop severely due to the muscle fatigue [33]. This property is well fitted by $p(t)$ in equation (3.19). The simulation results of the muscle activation level with the fatigue are shown in Figure 3.8. In this simulation, the pulse width is held at 500 μs . We can see that the drop rate of activation level is related with the stimulation frequency. Within the time range, 0-40 sec , the activation level of 40 pps is above 20 pps all along, which is due to the fact that high stimulation frequency can produce high activation level at the beginning. However, if time moves on furthermore, the activation level of 40 pps will be lower than that of 20 pps sooner or later.

Figure 3.8: Muscle fatigue property with regard to stimulation frequency.

Although the muscle under continuous electrical stimulation will be exhausted due to the fatigue, it can recover after resting for certain time. Actually, this time should be long enough [34]. First, the simulation study is performed to test the muscle's recovery property. In this case, a constant electrical signal is used to stimulate the

RF muscle. As the fatigue factor is set to a general ratio in equation (3.19), it is also suitable for the other eight muscles. The stimulation pattern is set as pulse width of 400 μs , stimulation frequency of 40 pps that is indicated by the thick bar in Figure 3.9, and pulse amplitude of 35 mA . The stimulation period is 1.5 sec , while the muscle rests for 1.5 sec . From Figure 3.9, we can see that the activation level keeps on dropping, that means the resting time is too short for the muscle to recovery.

In the second case, the same electrical signal is used to stimulate the muscle for 40 sec , and then the muscle rests for 120 sec . After that, we re-stimulate the muscle, and the result on fatigue is shown in Figure 3.10. In this case, the muscle can get a full recovery, and its high activation level can be achieved again. It is very similar with the weight lifting. The lifter's muscle is very strong at the beginning. However, after lifting for continuous repetitions, the muscle's force becomes weaker, and finally exhausted due to the fatigue as more repetitions are performed. If the weight lifter rests for some time between the sets of lifting, then the muscle will recover from its fatigue, and the same weight can once again be lifted.

Figure 3.9: Simulation result. No muscle recovery in the case with short resting time. Stimulation time (horizontal thick bar): 1.5 sec , and resting time: 1.5 sec .

Figure 3.10: Simulation result. Muscle recovery in the case with long resting time. Stimulation time (horizontal thick bar): 40 sec, and resting time: 120 sec.

Figure 3.11: Experiment on healthy subject to measure data with regard to muscle fatigue. RF muscle is stimulated via “Compex Motion” stimulator.

Experiment is performed on a healthy subject in BioRobotics Lab to verify the simulation results of muscle fatigue property. “Compex Motion” (Switzerland) stimulator is used [98], and RF muscle is stimulated. Force gauge is used to measure the force generated by RF. The setup of experiment is shown in Figure 3.11. The first experiment is performed to test the simulation result shown in Figure 3.9. The stimulation pattern is set as pulse width of 400 *us*, stimulation frequency of 40 *pps*, and pulse amplitude of 35 *mA*. The stimulation period is 1.5 *sec*, rest time is 1.5 *sec*, and the stimulation is repeated for 40 *sec*. The data of force is plotted in Figure 3.12. Comparing Figure 3.9 with Figure 3.12, we find the simulation result shows good agreement with the experimental data.

The second experiment is also conducted on RF muscle of the same subject in the second day after the first experiment. It aims to eliminate the effect of the first experiment. The objective is to verify the results on muscle recovery performance of the fatigue factor shown in Figure 3.10. The stimulation pattern is the same as that in the first experiment: pulse width of 400 *us*, stimulation frequency of 40 *pps* and pulse amplitude of 35 *mA*. The stimulation period is 40 *sec* and rest time is 120 *sec*. Repetitive stimulation is performed continuously for three times. The force data is measured and the experimental result is plotted in Figure 3.13. We find the good match is shown between Figure 3.10 and Figure 3.13. The credibility of the muscle fatigue model is verified.

3.2.2 Contraction Dynamics

The muscle contraction dynamics means force-length, and force-velocity properties. However, it is a quite formidable task to get the data via experiment, especially for the muscle length and velocity. Generally, surgical experiment should be performed on animals. However, for the human subject, a good and possible choice is to use noninvasive method. The length-force relationship can be expressed in terms of the

Figure 3.12: Experimental result. No muscle recovery in the case with short resting time. Stimulation time (short thick bar): 1.5 sec, and resting time: 1.5 sec.

Figure 3.13: Experimental result. Muscle recovery in the case with long resting time. Stimulation time (short thick bar): 40 sec, and resting time: 120 sec

torque (moment) and joint angle [82], [148], [168]. With some common equipments such as the goniometer and torque gauge, the data can be acquired, which needs not any surgery or equipment implanted in the human body.

Figure 3.5 shows the musculoskeletal structure of lower limb. The simulation is performed on the lower limb, where nine muscles and three joints are involved for each leg. In the simulation, the electrical pulse with the maximum pulse width is used to stimulate the muscles. In this case, the muscle activation reaches the saturation level and the maximum moment can be produced. Here, the optimal stimulation frequency is fixed at 20 *Hz*. This strategy ensures that the activation of the muscle is stable, because the effect of muscle fatigue is acceptable at this level. Therefore, the moment-angle relationship can be effectively evaluated.

The procedure for the simulation study is discussed as follows. For the hip joint, the extensors are hamstrings (HAM) and gluteus maximus (GMAX); the flexors are iliopsoas (IL) and rectus femoris (RF). When the extensors (flexors) are stimulated by the electrical pulse, the hip angle and knee angle vary between 60° and 180°. First, the hip angle is held at 60°, and then the knee angle increases from 60° to 180° with the step of 20°. The moment at each step is then recorded. When the hip angle is added by 20°, another set of data is achieved. This procedure is repeated to obtain the full data. The moment-angle relationship for the hip joint is shown in Figures 3.14– 3.16.

Hip Joint

The curve in Figure 3.14 shows the relationship between the total moments and joint angles (hip and knee), which is normalized to 100%. All flexors shown in Figure 3.14(a) are activated maximumly, and all extensors shown in Figure 3.14(b) are activated maximumly. Figures 3.15 and 3.16 show the contributions of each muscle relating the hip joint. IL and GMAX are uniarticular muscles, so the moments produced by them are only related with the hip angles. From the simulation results

shown in Figure 3.15(a) and 3.16(a), we can find that the surfaces are smooth and there is no fluctuation along the x -axis (knee angle).

Figure 3.14: (a) Relationship between the total normalized moment and angle of hip flexion. (b) Relationship between the total normalized moment and angle of hip extension.

(a)

(b)

Figure 3.15: (a) Moment-angle relationship of IL muscle. (b) Moment-angle relationship of RF muscle.

HAM and RF are the biarticular muscles, and both hip and knee angle affect the moment. It can be seen from Figures 3.15(b) and 3.16(b) that the surfaces are a little uneven in this case.

Note that the biarticular muscles are too short to fully flex, or fully extend simultaneously all over the two joints. For HAM, it can extend the hip joint and flex the knee joint, but these actions cannot be performed maximally at the same time. In fact, the hip extension is normally associated with the knee extension, and the hip flexion is normally associated with the knee flexion in cycling, or walking. In this way, the length of the muscle within the normal working range is about 100% to 130% of the rest length. The main advantage of the biarticular muscle is that the tension is produced in one muscle rather than two, or more, which can conserve energy. Accordingly, working within the 100% to 130% range allows the maximum flexibility in the tension generation.

Knee Joint

There are five muscle groups relating the knee joint movement. The extensors include vastus (VAS), RF; the flexors include HAM, biceps femoris (BF), and gastrocnemius (GAS). The location of BF is deep under the skin surface, and it can't be stimulated with a surface electrode. In fact, the tension produced by BF is very small compared to the other muscle groups. Therefore, its effect on the knee joint is not considered here.

The total moment-angle relationship of knee joint is shown in Figure 3.17 which is normalized to 100%. All flexors shown in Figure 3.17(a) are activated maximally, and all extensors shown in Figure 3.17(b) are activated maximally.

The moment-angle relationship of the individual muscles for the knee flexion is illustrated in Figure 3.18, and they are both the biarticular muscles. HAM crosses over the knee joint and hip joint, while GAS cross over knee joint and ankle joint.

Figure 3.16: (a) Moment-angle relationship of GMAX muscle. (b) Moment-angle relationship of HAM muscle.

Figure 3.17: (a) Relationship between the total normalized moment and angle of knee flexion. (b) Relationship between the total normalized moment and angle of knee extension.

However, in this simulation study, hip joint and knee joint are combined to evaluate the performance. The effect of ankle joint on GAS has not shown here, so the moment surface of GAS is a smooth lean plane. Note that muscle can produce the maximum force when it is at the optimal length. For HAM, the optimal length is determined by both hip and knee angles together. In order to make HAM hold at the optimal length, if the hip angle increases (decreases), the knee angle should also increase (decrease) accordingly. So the maximum force (moment) also follows the direction change as shown in Figure 3.18(a).

The moment-angle relationship of the individual muscles for knee extension is illustrated in Figure 3.18. Note that the moment produced by RF is very small compared with VAS.

Ankle Joint

There are three muscles acting on the ankle joint. They are tibialis anterior (TA, dorsiflexor), soleus (SOL, planar flexor), and GAS (biarticular planar flexor). The total moment-angle relationship of the ankle joint is shown in Figure 3.20, which is normalized to 100%. Here, only the contribution of planar flexors is considered. As the motion range for the ankle is relatively smaller, the upper limit is set by 150° . The moment-angle relationship of the individual muscles for ankle planar flexion is illustrated in Figure 3.21. The moment produced by SOL is significantly large. In fact, during human locomotion, it makes the major contribution at the stance phase.

3.3 Summary

In this chapter, a musculoskeletal model of human lower limb is constructed. Much work is focused on the muscle model, which is actually considered as an improved

Figure 3.18: (a) The moment-angle relationship of HAM muscle.(b) The moment-angle relationship of GAS muscle.

Figure 3.19: (a) Moment-angle relationship of VAS muscle. (b) Moment-angle relationship of RF muscle.

Hill-type model based on the experimental data from recent literatures. The key properties of the muscle are all considered in this model, which contain the force-length-velocity relationship, activation dynamics, passive element, muscle fatigue, frequency effect and so on. Body dynamics in the cases of one-segment to four-

Figure 3.20: Relationship between the total normalized moment and angle of ankle planar flexion.

segment is achieved using a method similar with that used in robotics. Simulation study shows that the model has a good agreement with experimental results published in the literature. This musculoskeletal model will be adopted as the control plant instead of a real patient in the design of the FES control system.

Figure 3.21: (a) Moment-angle relationship of SOL muscle. (b) Moment-angle relationship of GAS muscle.

Chapter 4

Neural Control Mechanism

Research in neuroscience has revealed that some rhythmic movements of the human lower limbs are not related very much with the higher level of the motor system. The rhythmic movement pattern appears to be constructed in advance. The spinal cord itself has the capability to generate the rhythmic activity for the lower limb movements such as cycling and walking. This is mainly due to the central pattern generator (CPG) in it.

As we know that the CPG is a network of neurons (or even a single neuron) that is able to exhibit rhythmic behavior in the absence of sensory input and descending control. For vertebrate, CPG is located in the spinal cord. When CPG is activated, it can generate rhythmic motor patterns for different movements such as walking, breathing, flying, and swimming. Generally, both CPG and neural oscillator can produce oscillatory curves, and the neural oscillator can be deemed a simplified version of CPG. However, they serve in two different areas: neuroscience and engineering. In this chapter, the CPG and neural oscillator are constructed and the simulation study will be carried out.

4.1 Central Pattern Generator

In this section we aim to construct the biological CPG model in the neuronal circuit of lamprey based on Grillner's work [58] (see Figure 4.1). The neurons in CPG can produce a set of oscillatory signal curves, i.e, the variation of the membrane potential of the neuron. Generally, there are two mechanisms contributing to this phenomenon.

The first mechanism is due to the intrinsic oscillatory membrane property of an individual neuron. This type of neuron can generate oscillatory output even it is isolated from the other neurons in the network, and it is usually named as "pacemaker" in some literatures. The pacemaker property is related with the membrane channels with voltage- and time-dependent ionic conductance. The second mechanism is named as network mechanism, and it is due to the interactions amongst the neurons in the neuronal circuit, none of which has specialized membrane pacemaker properties. The network structure of CPG is shown in Figure 4.1.

Research has shown that in some neuronal circuits the first mechanism is dominant at certain frequencies and the second mechanism is dominant at other frequencies. In this work the two mechanisms are both considered in the CPG design. The CPG circuit designed here consists of a set of coupled CPG units, and each CPG unit contains four neurons. The rhythmic pattern generated by the CPG is determined by the properties of the individual neurons, the interactions amongst the neurons in an individual CPG, the interactions amongst the CPGs in the network, and the afferent inputs (i.e. sensory feedback signal) provided by the external environment.

Figure 4.1: Segmental network for locomotion in the nervous system of lamprey. Modified from [58].

4.1.1 Neuron Model (Intrinsic Property)

The mechanism for each neuron of the CPG circuit can be described by the following equations:

$$C_m \frac{dV_m}{dt} = (\sum I_{syn} + I_p + I_{in} - \frac{V_m}{R_m}) \quad (4.1)$$

$$y_{out} = \max[\frac{2}{1 + \exp(-0.25V_m)} - 1, 0] \quad (4.2)$$

where V_m , R_m , and C_m are the membrane voltage, resistance, and capacitance of the neuron respectively, I_{syn} is the synaptic current input, I_p is the pacemaker current, I_{in} is the externally injected current which can excite the neuron and increase the membrane voltage, y_{out} is the output of the neuron. The membrane voltage V_m is converted into a continuous value between 0 and 1 by the modified sigmoid function in equation (4.2).

Arbitrarily, we select the motor neuron (Mn) in the CPG to analyze the intrinsic property. Actually, the other neurons have the similar mechanism, and the only difference is that the values of some parameters are different. The membrane voltage of Mn, V_m , is show in Figure 4.2. There are two items in equation (4.1) that should

Figure 4.2: Membrane voltage of the motor neuron.

be emphasized. One is the synaptic current I_{syn} , the other is the pacemaker current I_p . The synaptic current flowing from one neuron into another neuron is given by

$$I_{syn} = \sum y_{out} G_{syn} (E_{syn} - V_m) \quad (4.3)$$

where G_{syn} and E_{syn} are the conductance and the equilibrium potential for synapse between the different interneurons. The current I_{syn} is shown in Figure 4.3.

Figure 4.3: Synaptic current flowing into the motor neurons.

The pacemaker current I_p provides a single neuron in isolation with the ability to generate oscillatory behavior. The model used for the pacemaker currents is described by

$$I_p = I_{na} + I_k + I_{nmda} + I_{kca} \quad (4.4)$$

where I_{na} is the voltage-dependent sodium current, I_k is the voltage-dependent potassium current, I_{nmda} is the current through N-methyl-D-aspartate (NMDA) receptor channel, I_{kca} is the calcium-dependent potassium current. The experimental and simulation results presented in [14] and [42] demonstrate that the current I_{na} mainly contributes to the action potential and is not a key current for generation of oscillatory membrane potential. In fact, a smoother oscillatory membrane potential for single neuron can be achieved with blockade of I_{na} . As the motor neurons will receive the smoothened rhythmic action potentials due to low-pass filtering char-

acteristics of membrane when it receive external signals, we can directly achieve the smoother rhythmic oscillatory action potentials by blocking I_{na} , i.e. by setting $I_{na} = 0$. It was also shown in [14] and [42] that I_k may affect the frequency and the amplitude of oscillatory membrane potential. Therefore, we can also select $I_k = 0$ and then the frequency and the amplitude are adjusted by setting the conductance of the remained current channels. A similar choice of the CPG structure was also presented in the work [2], [53], [54]. This kind of simplification aims to focus on the key ionic channels concerned. It is also a preparation for the later CPG network design, because the extremely complex structure of CPG network contains hundreds of ionic channels and synapses, which is beyond the practical control in application. However, in the theoretical analysis in Chapter 5, the properties of CPG including the currents I_{na} and I_k will be discussed, which is actually in Section 5.1.3.

For the current I_{nmda} [192],

$$I_{nmda} = G_{nmda}(E_{nmda} - V_m)p \quad (4.5)$$

$$\frac{dp}{dt} = A_a e^{(V_m - E_r)/C} (1 - p) - A_b e^{(E_r - V_m)/C} p \quad (4.6)$$

where G_{nmda} is the conductance of the NMDA receptor channel, E_{nmda} is the equilibrium potential for the NMDA receptor channel, p is the fraction of NMDA channel in the open state, A_a and A_b are constants for opening and closing the NMDA channel, E_r is the resting potential. The activation of the NMDA channel will give rise to the membrane potential and the closure of the NMDA channel will lower the membrane voltage. This mechanism is shown in (4.6), where the first term means closing and the second means opening. The curve of the current I_{nmda} is shown in Figure 4.4.

Figure 4.4: Current of the NMDA channel in the motor neuron.

For the current I_{kca} [184]:

$$I_{kca} = G_{kca}(Ca_{nmda} + Ca_{ap})(E_{kca} - V_m) \quad (4.7)$$

$$\frac{dCa_{nmda}}{dt} = \rho_{nmda}(E_{canmda} - V_m)p - \delta_{nmda}Ca_{nmda} \quad (4.8)$$

$$\frac{dCa_{ap}}{dt} = q^5 \rho_{ap}(E_{ca} - V_m) - \delta_{ap}Ca_{ap} \quad (4.9)$$

where G_{kca} is the conductance of calcium-dependent potassium channels. Ca_{nmda} is the calcium contraction due to currents through the NMDA channel and Ca_{ap} is the calcium contraction due to currents through voltage-gated Ca^{++} (divalent cation of calcium) channel. E_{canmda} and E_{kca} are the equilibrium potentials for the NMDA receptor channels for calcium and the calcium-dependent channels, respectively. ρ and δ are the rates for calcium accumulation and depletion. q is to model the open and closure of $K(Ca)$ channel (Refer to [14] for more details). The current I_{kca} lowers the membrane potential. When Ca^{++} flows through the NMDA channels

and as it accumulates inside the cell, it opens K^+ channels which are sensitive to the local Ca^{++} concentration. The rate of change of Ca^{++} concentration is given by (4.8), and Ca^{++} concentration determines the I_{kca} as given in (4.7). The curve of current I_{kca} is shown in Figure 4.5.

Figure 4.5: Calcium-dependent potassium current I_{kca} in the motor neuron.

There are two components, I_{nmda} and I_{kca} , in equation (4.4) should be elaborated. I_{nmda} raises the membrane potential and I_{kca} lowers the membrane potential. The NMDA-receptor-mediated current provides the driving force for the oscillatory curve in the neuron of the CPG. They may behavior as oscillatory currents due to voltage- and time-dependent processes, which are responsible for opening and closing the NMDA channels. Equation (4.6) describes the rate of change in the fraction of NMDA channels. When the NMDA channels open, the current flows into the neuron, thus the membrane potential is increased.

In conclusion, there are three intrinsic mechanisms that contribute to the oscillatory curves of the neurons. The first mechanism is determined by the NMDA channels.

The second mechanism is due to the calcium-dependent potassium channels. The third mechanism is due to the sensory feedback signals and it will be discussed in the following section of this chapter. Besides the intrinsic mechanisms, the network mechanism also makes contribution on the generation of rhythmic patterns. All the mechanisms contributed to the oscillatory membrane voltage of the CPG are shown in Figure 4.6.

Figure 4.6: (a) Ionic currents related to a neuron in the CPG. (b) The mechanisms contributing to the oscillatory curves. SR-E and SR-I is the sensory excitatory current and inhibitory current separately.

4.1.2 Network Mechanism

From Figure 4.6(b) we can see that not only the intrinsic property but also the network mechanism contribute to the rhythmic patterns, and recent research has shown that polysynaptic connections (networks) are predominant in mammalian [15]. The network structure of the CPG will be analyzed in this section.

The architecture of the CPG network is developed based upon the model of the neuronal circuitry responsible for generating locomotion in the lamprey, which consists

of two coupled central pattern generators (Figure 4.1). Each CPG has a set of four interconnected neurons. The intrinsic property of the neuron has been discussed in Section 4.1.1, and we know each neuron in the CPG serves as a specific function in the generation of oscillatory activity.

The network architecture and the signs of the synaptic interconnections for one pair of CPGs are shown in Figure 4.7. The excitatory interneuron, En, provides the excitatory drive to the other three neurons; the inhibitory glycinergic interneuron, In, provides the recurrent inhibition to contribute to the termination of the excitatory burst for the neurons in another CPG unit; and the lateral interneuron, Ln, inhibits the In interneuron and motor neuron (Mn). The sensory feedback signals are of two types: one (SR-E) excites the CPG and the other (SR-I) inhibits it. The interaction between the four neurons is described by equation (4.3).

Figure 4.7: Network structure of CPG. Each CPG unit contains four interacting neurons: En, In, Ln and Mn. Two CPG units are also coupled with inhibitory synapses.

4.1.3 Simulation Results

A simulation study is performed in Matlab/Simulink based on equations (4.1)-(4.9). The parameters of the CPG are adopted from [14] and given in Tables 4.1 and 4.2. The simulation results are shown in Figure 4.8, which represent the outputs of the four neurons respectively. The rhythmic pattern of each neuron is at the frequency of 1 Hz. The amplitude of the output is normalized to 1.0.

Figure 4.8: (a) is the output of the En interneuron, (b) is the output of In interneuron, (c) is the output of Ln interneuron, (d) is the output of motor neuron (Mn).

Table 4.1: Parameters of coupled neurons in CPG

name	$R_m(M)$	$C_m(pF)$	$V_o(mV)$	synaptic neuron	$G_{syn} (uS)$	$E_{syn}(mV)$
En_1	60	0.04	25	In_2	2.0	-30
Ln_1	50	0.20	25	En_1	0.05	40
				In_2	0.10	-40
In_1	10	0.02	10	En_1	0.20	20
				In_2	0.10	-20
				Ln_1	0.10	-20
Mn_1	50	0.02	25	En_1	0.05	40
				In_2	0.10	-40

Table 4.2: Parameters of coupled neurons in CPG

G_{nmda}	5 uS	G_{kca}	0.04 uS
E_{nmda}	70 mV	E_{canmda}	90 mV
E_{kca}	10 mV	E_r	10 mV
A_a	1400 s^{-1}	A_b	131151 s^{-1}
C	17 mV	I_{inj}	0
ρ_{nmda}	5 $mV^{-1}s^{-1}$	δ_{nmda}	2 s^{-1}
ρ_{ap}	4 $mV^{-1}s^{-1}$	δ_{ap}	20 s^{-1}

4.2 Neural Oscillator

From the analysis on the biological CPG in Section 4.1, we know that there are two mechanisms contributing to the rhythmic patterns of CPG. One is the intrinsic mechanism due to the ionic channels in the neuron, the other is the network mechanism due to the interactions amongst the neurons. With a mathematical point of view, even a single neuron could contain many ODEs (ordinary differential equation), then a network of CPG may contains hundreds of ODEs and numerous parameters. The extremely complex structure of the CPG is beyond control and its application is limited in the engineering practice, e.g. biped and FES locomotion where rhythmic movements are required. Therefore, a trade-off must be made between complexity and controllability, i.e. an practical neural oscillator is needed. In this section, a neural oscillator inspired and simplified from the biological CPG is presented.

The CPG discussed above is modeled from the biological perspective. In fact many simplified CPG models have been designed for the engineering purpose, especially for bio-inspired robot [11], [43], [44], [46], [48], [89], [90], [111], [115], [133], [141], [158], [180], [193]. The basic mechanism is similar with that of the biological CPG, but the structure is relatively simple, and the number of variables or parameters is appropriate. In the mathematical point of view, it means the number of ODEs has been significantly reduced in comparison with that of CPG nervous system in the biology. The simplified CPG is also named as “neural oscillator” in some research. Currently, there are three popular types of neural oscillators widely used to mimic the biological CPG. The first type is recurrent neural oscillator [8], [44], [90], [125], [182], [194]. The second type is lamprey (phase) oscillator [11], [43], [91], [134]. The third type is Van der Pol neural oscillator [10], [116]. The recurrent neural oscillator model is utilized to provide the simulation study in this work. In the neural oscillator model, the biological CPG unit can be simplified by two coupled neurons. The structure of both are illustrated in Figure 4.9.

Here, we give some qualitative analysis of the biological CPG and Matsuoka neural oscillator and discuss their relationship. The four coupled ionic channels of the intrinsic mechanism in CPG, $NMDA$, $K(Ca)$, K^+ , and Na^+ , can be transformed into the self-inhibited routine of the neural oscillator. The parameters of these channels such as ρ , δ , A_a , A_b , C are related to intrinsic inhibitory ionic channels. It can be characterized by only one parameter β in equation (4.13), which indicates the inhibitory connection.

$$\beta \sim f_{\beta}(\rho, \delta, A_a, A_b, C) \quad (4.10)$$

The conductances of the ionic channels such as G_{nmda} , G_{kca} , G_{na} , G_k and the membrane capacitance of the neuron are transformed into the time constant T of the

neural oscillator that appears in equation (4.14):

$$T \sim f_T(G_{nmda}, G_{kca}, G_{na}, G_k, C_m) \quad (4.11)$$

The membrane resistance R_m and capacitance C_m of the neuron is defined by the time constant τ_1 of the neural oscillator that appears in equation (4.13):

$$\tau \sim R_m C_m \quad (4.12)$$

Note that the important parameter p in equation (4.6) plays as a gate for the oscillation behavior. Similarly, there is also such a parameter in Matsuoka neural oscillator, i.e. $f(x_i) = \max(x_i, 0)$ in equation (4.15). The four neurons with interaction in one biological CPG (see Figure 4.7) can be simplified by two coupled neurons.

In summary, the whole process of generalization discussed above is shown in Figure 4.9. A similar process as our work has appeared in [83]. However, all the relationships given above are qualitative descriptions, as at present we can only roughly derive them from the simulation results. More accurate formats in detail need to be further investigated.

4.2.1 Mathematical Model

The recurrent neural oscillator can be described by the following coupled ordinary differential equations.

$$\tau \dot{x}_i = -x_i - \beta v_i - \sum_{j=1}^n w_{ij} y_j + r_i \quad (4.13)$$

$$T \dot{v}_i = -v_i + y_i \quad (4.14)$$

Figure 4.9: Qualitative relationship between biological CPG and neural oscillator. The numerous parameters of ionnic channels and complex relationships in CPG are generalized into the simple format of neural oscillator.

$$y_i = f(x_i - x_\theta), f(x) = \max(0, x) \quad (4.15)$$

where i indicates the i th neuron in the neural oscillator, x_i is the membrane potential, y_i is the neuron output, v_i is the membrane current of the slow recovery component, $f(x)$ is a nonlinear output function of the neuron, and x_θ is the membrane potential threshold. τ and T are the time constants, w_{ij} is the weight for connection with other neurons, β is the coefficient associated with the slow current events, and r_i is the tonic input to the neuron.

For a detailed case, the four-dimensional neural oscillator model is adopted that consists of two neurons arranged in mutual inhibition, and the structure is shown in Figure 4.10(a). The dynamics of Figure 4.10(a) can be represented by the following differential equations (4.16)-(4.21). The diagram in Figure 4.10(b) clearly shows the dynamic relationship, which is helpful for understanding the relationship. As the

Figure 4.10: (a) Network structure of neural oscillator. It is composed of two coupled neurons with self-inhibition. Black circles correspond to inhibitory connections, open to excitatory ones. (b) Schematic diagram shows the intrinsic dynamics of the neuron 1 in the coupled neural oscillator.

two neurons are symmetric, only neuron 1 is plotted.

$$\tau_1 \dot{x}_1 = -x_1 - \beta v_1 - h y_2 + L e + r_1 \quad (4.16)$$

$$\tau_2 \dot{v}_1 = -v_1 + y_1 \quad (4.17)$$

$$\tau_1 \dot{x}_2 = -x_2 - \beta v_2 - h y_1 - L e + r_2 \quad (4.18)$$

$$\tau_2 \dot{v}_2 = -v_2 + y_2 \quad (4.19)$$

$$y_i = f(x_i), i = 1, 2 \quad (4.20)$$

$$y_{out} = c_1 y_1 - c_2 y_2, c_1 - 1, c_2 > 0 \quad (4.21)$$

where x_i is the variable similar with the membrane voltage in a neuron, v_i is the membrane current of the slow recovery component, $f(x_i)$ is a nonlinear function which has a unit gain for x_i when the input is non-negative and zero otherwise, y_i is the neuronal output, y_{out} is the CPG output, h is the gain for the interaction between the two neurons, β is the adaptive gain for the self-inhibition of the neuron, r is the tonic input, e is the sensory feedback and L is the feedback gain.

The amplitude of the oscillator is regulated by the tonic input r , and it is proportional to r . The frequency of the oscillator is determined by the time constants τ_1 and τ_2 . Roughly, the frequency of the oscillator output varies inversely as τ_1 . However, τ_2 also has effect on the oscillatory frequency, so the more exact relationship should be $f_{out} = f(\tau_1, \tau_2)$. The detailed relationship will be proved in Chapter 5. The error sensory feedback e entrains the oscillator with the gain L . When L is zero, the feedback information is ignored and the system functions identically to a pure feedforward system. When L is very large, the neural oscillator is immediately entrained to the feedback signal. However, if the sensory feedback e is eliminated, the oscillator will produce invariant rhythmic pattern, regardless of the magnitude of L .

4.2.2 Simulation Results

Based on the equations (4.16)-(4.21), the simulation study is performed. It can be seen from Figure 4.11 that the output of the oscillator is at the rhythmic pattern of 1.1 Hz. This is considered as a normal rhythmic pattern. Here, the parameters are selected as $\tau_1 = 0.1$, $\tau_2 = 0.2$, $\beta = 2.5$, $h = 2.5$, $c_1 = c_2 = 1.0$, the tonic input $r_1 = r_2 = 1.0$ and sensory feedback $e = 0.1$.

Figure 4.11: A general simulation. Four states of the neural oscillator: x_1 , x_2 , v_1 , v_2 . Output of the neural oscillator: y_{out} .

Effect of tonic input

Tonic inputs affect the behavior of CPG significantly. However, this effect also brings confusion. For example, some literature claimed the tonic input can change the output frequency [11]; while the others found it would not change the output

frequency, but output amplitude [114]. Perhaps this controversy is due to the different structure of CPG models that they used. For Matsuoka neural oscillator, the previous theoretical analysis has proved the output frequency is not related with tonic input [8], [212]. A proper explanation is that the variation of other parameters can change the equilibrium of the neural oscillator, and in this case the tonic input should be changed to guarantee the stable oscillation [133]. However, we could not say the tonic input changes the frequency. This work will not focus on this property although it can be indicated by the phenomena as shown in Figures 4.12(c) and (d), where the output frequency is the same under different tonic inputs. Here, a new property is explored to study the effect of tonic input on resisting the noise of sensory feedback.

The noise along with the afferent signal (sensory feedback signal) affects the oscillatory behavior of the neural oscillator, however, the tonic input plays an important role to resist against the noise disturbance. In the simulation, a white noise with power 0.5 (Figure 4.12(a)) is applied to the sensory feedback signal to illustrate this case. If the tonic input is relatively small, the neural oscillator cannot generate the regular rhythmic signals, which can be seen from Figure 4.12(b), as in this case the added noise has disturbed the normal rhythm. However, if the tonic input increases, the influence of the disturbance can effectively be reduced and an improved oscillatory pattern can be ensured as shown in Figure 4.12(c). Clearly, it can be seen from Figure 4.12 (d) that when the tonic input is large enough, the influence of the noise can be well eliminated and a satisfactory oscillatory pattern can be achieved. Therefore, the robustness of the neural oscillator can be enhanced via an increase of the tonic input. In general, a large tonic input to the neural oscillator is desirable for the neural oscillator to resist against the disturbance from the sensory feedback.

This result is very useful for the CPG design in FES control system of Chapter 6. It is also meaningful for robotics or other control application. As we know CPG

mostly functions as a feedforward controller in a control system [3], [105], and it is good at dealing with measurement noise but sensitive to unexpected disturbance. However, the application of the pure feedforward controller is very limited. The adaptive ability is desired for CPG design [22], [112], [159]. Therefore, sensory feedback should be incorporated to regulate CPG, but the noise along with sensory feedback may bring trouble for CPG to generate proper patterns. Our result has indicated a possible way to resolve this problem.

Figure 4.12: Robustness of the neural oscillator against the noise along with sensory feedback. (a) is the white noise. (b) is the output of neural oscillator y_{out} with $r=1.0$. (c) is y_{out} with $r=10$. (d) is y_{out} with $r=100$.

Effect of sensory feedback

It is well known that the CPG can independently generate a successful rhythmic pattern in the absence of sensory or descending inputs, but it doesn't exclude the

fact that the sensory feedback plays an important role to affect the normal rhythmic pattern, especially for the mammals [32], [206]. In fact, there is not a single type of sensory feedback, and the integration of different sensory feedbacks affects the CPG altogether such as force-feedback, limb loading and joint angles [206]. We have discussed the basic mechanisms of CPG in Section 4.1, however, due to various types of feedbacks and their complexities, it is hard to determine how the sensory feedback works on the CPG. In this section, we can get some clear results through simulation study on the simplified CPG, neural oscillator.

In the neural oscillator, if the sensory feedback is zero and the tonic inputs of the two neurons are identical, then the two neurons are completely symmetric. In this case, the neural oscillator cannot produce the oscillation signals, i.e. the output is actually zero. However, a very small influence can make the system states change significantly. For example, even the sensory feedback is as small as $e = 10^{-8}$ in Figure 4.13(a), the neural oscillator may still generate an oscillatory pattern. This interesting phenomenon is somewhat like the “butterfly effect” and it is firstly reported in this work according to our comprehensive review. Also, due to the different intensity of the sensory feedback signal, the time delay for the oscillation varies. It can be seen from Figure 4.13 that sensory feedback of a large value can make neural oscillator use less time in reaching stable oscillatory state. Figure 4.13 shows that the time delays are $t = 2.6sec$, $t = 1.9sec$, $t = 0.7sec$, respectively, with regard to the corresponding intensities of the feedback signal: $e = 10^{-8}$, $e = 10^{-6}$, $e = 10^{-4}$. It verifies that a very weak sensory feedback could not initiate a rhythmic movement immediately.

It is interesting to discuss why the neural oscillator is so apt to generate the oscillation in this case. We give some explanation here. The parameters selected here, such as τ_1 , τ_2 , β and h , can guarantee an oscillation, then the oscillation is locally stable in a large area [125]. The key characteristics is that it exhibits a limit cycle in

Figure 4.13: Time delay due to the different intensity of sensory feedback e . (a) Time delay is 2.6 sec if $e = 10^{-8}$. (b) Time delay is 1.9 sec if $e = 10^{-6}$. (c) Time delay is 0.7 sec if $e = 10^{-3}$.

x_1 vs. x_2 plane [212], and Section 5.2.4 in Chapter 5 also provides the elaboration. The limit cycle is plotted in Figure 4.14, which indicates the stable oscillation. In the plane, any initial conditions of x_1, x_2 will converge to the limit cycle except $x_1 = x_2$. If the initial conditions are selected as $x_1 = x_2$, the neural oscillator will not generate oscillation because the two neurons are totally symmetric. We can see that the line $x_1 = x_2$ is surrounded by the vast area of the stable oscillation. Even a very weak sensory feedback can make the initial conditions of x_1 and x_2 deviate from the line $x_1 = x_2$. It can be observed why the oscillation is easily generated from Figure 4.14.

Figure 4.14: Limit cycle of neural oscillator in x_1 vs. x_2 plane. Initial conditions in all the area will converge to the limit cycle except the line $x_1 = x_2$.

However, with a constant tonic input, in order to produce the stable oscillation, the feedback signal cannot exceed a certain range. Otherwise, the output will reach a non-zero constant value, and the oscillation will cease, which can be seen from the dash-dot lines in Figure 4.15(a) and (b). For the case as shown in Figure 4.15(a), where the tonic input $r_1 = r_2 = 1.0$ is applied and the sensory feedback e increases from 0.10 to 0.17, note that the amplitude of the oscillatory curves

decreases gradually, but the frequency is not affected, which always keeps a constant value, and finally the oscillation stops at the amplitude of 0.32 (see the dash-dot curve).

If the tonic input r is varied, the range of the sensory feedback for stable oscillation is also varied accordingly. If the tonic input becomes large, the upper limit of the range also becomes large. It can be seen from Figure 4.15 that when the tonic is 1.0, the sensory signal for stable oscillation can reach the upper limit: $e = 0.17$ in Figure 4.15(a); if the tonic increases 10 times: $r_1 = r_2 = 10$, the upper limit of the sensory signal also increases 10 times, that is $e = 1.7$ in Figure 4.15(b).

Comparing Figure 4.15(a) with Figure 4.15(b), we also find the frequency is not changed although the tonic inputs are different. It is accordant with the result we have drawn in the previous section.

Entrainment of sensory feedback

If the sensory feedback is a series of rhythmic signals, it can entrain the neural oscillator and will cause the phase difference, which is shown in Figure 4.16. It indicates that the phase shift is related to the amplitude of the sensory feedback. It can be seen from Figure 4.16(c) that if the amplitude is high enough and still within the oscillatory region, then the phase of the neural oscillator will completely follow the sensory feedback.

However, if the frequency of sensory feedback is different with that of CPG, it will also entrain CPG to follow its frequency under the condition that its amplitude exceeds certain value (critical amplitude). Similar result was reported in [127] where a respiratory CPG model was studied. Although its structure is different with that of Matsuoka oscillator, they have similar entrainment property. That means when the amplitude of the sensory feedback is high enough, it can entrain the CPG to follow the driving frequency completely. The critical amplitude is related to the

(a)

(b)

Figure 4.15: Influences of sensory feedback on the stable oscillation of the neural oscillator. (a) The output y_{out} with different sensory feedbacks “ e ”, where tonic input $r=1.0$. (b) is the output where tonic input $r=10$.

Figure 4.16: Phase difference due to the sensory feedback. (a) is the normal output without the effect of the sensory feedback. In (b) and (c), the sensory feedback of a series of square pulse is applied to the neural oscillator. The amplitude of sensory feedback is 1 in (b), and 5 in (c).

DC part that indicates the tonic input r here. Two examples show the relationship in Figure 4.17: $f = 0.5$ in one case and $f = 1.0$ in another case, where f is the natural frequency of CPG. The relationship is nearly linear. We find that the natural frequency of CPG is almost monotonously increasing with the critical amplitude of sensory feedback.

Figure 4.17: Relationship between critical amplitude of sensory feedback and tonic input r to CPG. $f = 1.0$ and $f = 0.5$ represents the natural output frequency of CPG.

This property is very important for human bilateral rhythmic movement control, because the motor patterns to the symmetric muscles of two contralateral limbs should have the out-of-phase (phase difference) relationship. Generally, this phenomenon is realized through two ways: one is neural interaction between the bilateral CPG units [182], and the other is the mechanical coupling from sensory feedback [185], [206]. Our simulation demonstrates the later case, and it has been applied in CPG design for FES control system [211], where the mechanical sensory information from foot touch is used to entrain the two sets of CPG for lower limbs. Also, this mechanism will be used in Chapter 6.

4.3 Summary

In this chapter, the biological mechanism of the central pattern generator (CPG) is presented. A simplified CPG and neural oscillator, which are commonly used in engineering practice, are introduced.

For the CPG, the intrinsic mechanism and network mechanism are both studied. The mathematical equations describing the ionic channels in the neuron such as NMDA, Ca^{++} are introduced. The interactions amongst the neurons in a CPG unit and the networks with multiple CPGs are also established. Simulation studies are performed to test these mechanisms.

New subtle properties of the neural oscillator are also addressed and analyzed in the simulation study. The effects of the sensory feedback and tonic input on the rhythmic patterns are emphasized. From the simulation study, we can see that although the structure of the biological CPG is more complex than the neural oscillator, the functions and the oscillatory patterns generated from both are very similar. It implies that we can borrow some useful ideas from the biological science, so that certain engineering problems can be effectively handled. Finally, it is worthy to be noted that this neural mechanism presented in this chapter aims to serve for the FES control system in Chapter 6, where typical rhythmic movements of lower limbs will be controlled.

Chapter 5

Theoretical Analysis

This chapter provides some theoretical (mathematical) analysis on CPG and neural oscillator from the engineering perspective. Some criteria such as parameter selection, determination of frequency, and conditions of stable oscillation, are established. The analysis will further strengthen the foundation of CPG theory, and it will significantly enhance the effective application of CPG into the FES control systems.

5.1 CPG

The CPG model has been developed in Chapter 4. Here, we focus on the analysis of intrinsic property of the CPG, which is due to the interactions amongst the ionic channels in a single neuron. Therefore, the current from other neurons I_{syn} is ignored. I_{in} is the external injection current, which only exists in the experiment situation. In the natural situation of CPG, it does not exist. The ionic channels of *NMDA* and Ca^{2+} are important and have specific functions in the neuron of CPG, and firstly most analysis will be performed on these channels. Other channels such as Na^+ and K^+ are the basic and common channels in all the neurons, and they

have been thoroughly investigated since the classic work of Hodgkin and Huxley, 1952 [79]. However, as a further investigation, in Section 5.1.3, a more complete model including all the above currents will be analyzed.

Firstly, the related equations mentioned in Chapter 4 can be summarized as follows

$$\frac{dV_m(t)}{dt} = [I_{nmda}(t) + I_{kca}(t) - \frac{V_m(t)}{R_m}] / C_m \quad (5.1)$$

$$I_{nmda}(t) = G_{nmda}[E_{nmda} - V_m(t)]p(t) \quad (5.2)$$

$$\frac{dp(t)}{dt} = A_a e^{[V_m(t) - E_r]/C} [1 - p(t)] - A_b e^{[E_r - V_m(t)]/C} p(t) \quad (5.3)$$

$$I_{kca}(t) = G_{kca} C a_{nmda} [E_{kca} - V_m(t)] \quad (5.4)$$

$$\frac{dC a_{nmda}(t)}{dt} = \rho_{nmda} [E_{canmda} - V_m(t)] p(t) - \delta_{nmda} C a_{nmda}(t) \quad (5.5)$$

5.1.1 CPG Properties

Lemma 5.1 *A differential equation is given by*

$$\frac{dy(t)}{dt} = \eta(t)[M_1 - y(t)] + \gamma(t)[M_2 - y(t)] \quad (5.6)$$

where $M_1 \geq M_2$ are constants and $y(t) \in \mathbf{R}$. $0 < \eta(t) < \infty (\in \mathbf{R})$ and $0 < \gamma(t) < \infty (\in \mathbf{R})$ are continuous for $t \geq 0$. Then, it has

$$M_2 \leq y(t) \leq M_1, \forall t \geq 0 \quad (5.7)$$

for $\forall y(0) \in [M_2, M_1]$.

Proof: Equation (5.6) can be written as

$$\frac{dy(t)}{dt} = [\eta(t) + \gamma(t)] \left[\frac{M_1 \eta(t) + M_2 \gamma(t)}{\eta(t) + \gamma(t)} - y(t) \right] \quad (5.8)$$

Since $M_2 \leq \frac{M_1\eta(t)+M_2\gamma(t)}{\eta(t)+\gamma(t)} \leq M_1$, it has

$$M_2 - y(t) \leq \frac{M_1\eta(t) + M_2\gamma(t)}{\eta(t) + \gamma(t)} - y(t) \leq M_1 - y(t) \quad (5.9)$$

Then by multiplying $\eta(t) + \gamma(t)$, which is positive, into (5.9) we obtain

$$[\eta(t) + \gamma(t)][M_2 - y(t)] \leq \frac{dy(t)}{dt} \leq [\eta(t) + \gamma(t)][M_1 - y(t)] \quad (5.10)$$

From (5.10) it results in

$$\frac{d[y(t) - M_2]}{dt} + [\eta(t) + \gamma(t)][y(t) - M_2] \geq 0 \quad (5.11)$$

$$\frac{d[M_1 - y(t)]}{dt} + [\eta(t) + \gamma(t)][M_1 - y(t)] \geq 0 \quad (5.12)$$

Define

$$\frac{dy_1(t)}{dt} + \lambda_m y_1(t) = 0 \quad (5.13)$$

where $\lambda_m = \max\{\eta(t) + \gamma(t)\}$. Then

$$y_1(t) = y_1(0)e^{-\lambda_m t} \quad (5.14)$$

which exponentially converges to 0. We have from the contraction theorem that $y(t)$ in (5.11) converges exponentially and obviously it satisfies $y(t) - M_2 \geq y_1(t)$ for $y_1(0) = y(0) - M_2$, where $y(0) \in [M_2, M_1]$. Therefore,

$$y(t) - M_2 \geq [y(0) - M_2]e^{-\lambda_m t} \quad (5.15)$$

and

$$y(t) \geq M_2 + [y(0) - M_2]e^{-\lambda_m t} \geq M_2 \quad (5.16)$$

In a similar way, for (5.12) we obtain

$$M_1 - y(t) \geq [M_1 - y(0)]e^{-\lambda_m t} \quad (5.17)$$

or

$$y(t) \leq M_1 - [M_1 - y(0)]e^{-\lambda_m t} \leq M_1 \quad (5.18)$$

From (5.16) and (5.18), it can be concluded that (5.7) is true for $\forall y(0) \in [M_2, M_1]$.

This has completed the proof.

Lemma 5.1 shows that $y(t)$ in the differential equation (5.6) will remain in $[M_2, M_1]$ if its initial conditions $y(0) \in [M_2, M_1]$. Here, $M_1 \geq M_2$ can be positive or negative constants. It is useful in the proof of the following propositions.

Proposition 5.1 *For $p(t)$ given in (5.3) it has*

- $\lim_{V_m(t) \rightarrow \infty} p(t) = 1$
- $\lim_{V_m(t) \rightarrow -\infty} p(t) = 0$
- $0 < p(t) < 1$ with $0 < p(0) < 1$ for $-\infty < V_m(t) < \infty$

Proof: With regard to equation (5.3), we can rewrite it as

$$\frac{dp(t)}{dt} + [A_a e^{[V_m(t) - E_r]/C} + A_b e^{[E_r - V_m(t)]/C}]p(t) = A_a e^{[V_m(t) - E_r]/C} \quad (5.19)$$

For the differential equation (5.19), $V_m(t)$ is a function, but it can be assumed $V_m(t) \in [-\infty, \infty]$. In this case, we verify the boundness of $p(t)$. Here, $A_a, A_b, C > 0$ are constants and selected based on the membrane potential range. We have the following result

$$p(t) = r(t)[q(t) + C_p] \quad (5.20)$$

with

$$\begin{aligned}
 r(t) &= e^{-\int [A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}] dt} \\
 q(t) &= \int A_a e^{\int [A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}] dt} e^{[V_m(t)-E_r]/C} dt \\
 C_p &= \text{constant}
 \end{aligned} \tag{5.21}$$

Then

$$p(t) = \frac{A_a \int e^{\int [A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}] dt} e^{[V_m(t)-E_r]/C} dt + C_p}{e^{\int [A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}] dt}} \tag{5.22}$$

To investigate $p(t)$ when $V_m(t)$ changes, firstly we consider it for $V_m(t) \rightarrow \infty$. In this case,

$$\lim_{V_m(t) \rightarrow \infty} p(t) = \lim_{V_m(t) \rightarrow \infty} \frac{A_a \int e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{\int A_b e^{[E_r-V_m(t)]/C} dt} e^{[V_m(t)-E_r]/C} dt + C_p}{e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{\int A_b e^{[E_r-V_m(t)]/C} dt}} \tag{5.23}$$

It can be seen that

$$\lim_{V_m(t) \rightarrow \infty} e^{\int A_b e^{[E_r-V_m(t)]/C} dt} = 1 \tag{5.24}$$

So that equation (5.23) becomes

$$\lim_{V_m(t) \rightarrow \infty} p(t) = \lim_{V_m(t) \rightarrow \infty} \frac{A_a \int e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{[V_m(t)-E_r]/C} dt + C_p}{e^{\int A_a e^{[V_m(t)-E_r]/C} dt}} \tag{5.25}$$

Clearly, both the numerator and denominator of (5.25) approach infinity when $V_m(t) \rightarrow \infty$. Thus, taking derivative yields

$$\lim_{V_m(t) \rightarrow \infty} p(t) = \lim_{V_m(t) \rightarrow \infty} \frac{A_a e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{[V_m(t)-E_r]/C}}{A_a e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{[V_m(t)-E_r]/C}} = 1 \tag{5.26}$$

Now, we verify $p(t)$ when $V_m(t) \rightarrow -\infty$. Since

$$\lim_{V_m(t) \rightarrow -\infty} e^{\int A_a e^{[V_m(t)-E_r]/C} dt} = 1 \quad (5.27)$$

we have

$$\lim_{V_m(t) \rightarrow -\infty} p(t) = \lim_{V_m(t) \rightarrow -\infty} \frac{A_b \int e^{\int A_a e^{[E_r-V_m(t)]/C} dt} e^{[V_m(t)-E_r]/C} dt + C_p}{e^{\int A_b e^{[E_r-V_m(t)]/C} dt}} \quad (5.28)$$

Taking derivative for the numerator and denominator simultaneously results in

$$\lim_{V_m(t) \rightarrow -\infty} p(t) = \lim_{V_m(t) \rightarrow -\infty} e^{2[V_m(t)-E_r]/C} = 0 \quad (5.29)$$

In the above the boundness of $p(t)$ when $V_m(t) \rightarrow \infty$ and $V_m(t) \rightarrow -\infty$, has been proved. Next we will show that $p(t)$ is also bounded for $-\infty < V_m(t) < \infty$. It can be obtained from (5.3) that

$$\frac{dp(t)}{dt} = \alpha(t)[\beta(t) - p(t)] \quad (5.30)$$

where

$$\alpha(t) = A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}$$

$$\beta(t) = \frac{A_a e^{[V_m(t)-E_r]/C}}{\alpha(t)}$$

Note that $\alpha(t)$ and $\beta(t)$ are positive and continuous and it can be seen easily that $0 < \beta(t) < 1$ for $-\infty < V_m(t) < \infty$. Therefore, it has

$$0 < \frac{dp(t)}{dt} + \alpha(t)p(t) < \alpha(t) \quad (5.31)$$

In the similar way used in the proof of Lemma 1, we obtain that $0 < p(t) < 1$ with $-\infty < V_m(t) < \infty$ for $0 < p(0) < 1$. This has completed the proof.

Remark 5.1: Proposition 5.1 has shown that $p(t)$ in equation (5.3) should be bounded. Moreover, when $V_m(t)$ increases, $p(t)$ rapidly approaches its steady-state. In fact, the coefficients A_a , A_b and C will determine the steady-state values of $p(t)$ with respect to $V_m(t)$. Figure 5.1 also confirms this result, where $p(t)$ versus $V_m(t)$ is plotted and the parameters given in Table 5.1 are adopted.

Figure 5.1: Boundness of $p(t)$ for any $V_m(t)$.

Proposition 5.2 Equation (5.20) is a continuous function and it has

$$\lim_{|V_m(t)| \rightarrow \infty} \frac{dp(t)}{dt} = 0 \quad (5.32)$$

and $\max\{\frac{dp(t)}{dt}\}$ with respect to $V_m(t)$ occurs with

$$V_m(t) = E_r + \frac{1}{2} \ln\left(\frac{A_b}{A_a}\right)C \quad (5.33)$$

Proof: The proof is given as follows. For $p(t)$ given in (5.20), its differential is

denoted as

$$\frac{dp(t)}{dt} = -r(t)[A_a e^{[V_m(t)-E_r]/C} + A_b e^{[E_r-V_m(t)]/C}][q(t) + C_p] + A_a e^{[V_m(t)-E_r]/C} \quad (5.34)$$

For $V_m(t) \rightarrow \infty$, $A_b e^{[E_r-V_m(t)]/C} \rightarrow 0$, so equation (5.34) can be transformed into

$$\lim_{|V_m(t)| \rightarrow \infty} \frac{A_a e^{[V_m(t)-E_r]/C} [e^{\int A_a e^{[V_m(t)-E_r]/C} dt} - \int e^{\int A_a e^{[V_m(t)-E_r]/C} dt} e^{[V_m(t)-E_r]/C} dt + C_p]}{e^{\int A_a e^{[V_m(t)-E_r]/C} dt}} \quad (5.35)$$

After a lengthy, but simple manipulation, it gives

$$\lim_{V_m(t) \rightarrow \infty} \frac{dp(t)}{dt} = 0 \quad (5.36)$$

Similarly, we can also show

$$\lim_{V_m(t) \rightarrow -\infty} \frac{dp(t)}{dt} = 0 \quad (5.37)$$

Since we are interested in the maximum of $\frac{dp(t)}{dt}$ with respect to $V_m(t)$, a fixed membrane potential is considered when computing $\max\{\frac{dp(t)}{dt}\}$. Thus, it is assumed that $V_m(t) = V_m$ is constant. Then, we have

$$\frac{dp(t)}{dt} = -r(t)[A_a e^{(V_m-E_r)/C} + A_b e^{(E_r-V_m)/C}][q(t) + C_p] + A_a e^{(V_m-E_r)/C} \quad (5.38)$$

where

$$\begin{aligned} r(t) &= e^{-[A_a e^{(V_m-E_r)/C} + A_b e^{(E_r-V_m)/C}]t} \\ q(t) &= \frac{A_a e^{(V_m-E_r)/C}}{A_a e^{(V_m-E_r)/C} + A_b e^{(E_r-V_m)/C}} e^{[A_a e^{(V_m-E_r)/C} + A_b e^{(E_r-V_m)/C}]t} \\ C_p &= \text{constant} \end{aligned} \quad (5.39)$$

Thus,

$$\frac{dp(t)}{dt} = -C_p[A_a e^{(V_m - E_r)/C} + A_b e^{(E_r - V_m)/C}]r(t) \quad (5.40)$$

Let $\dot{p} = \frac{dp(t)}{dt}$, it results in

$$\frac{d\dot{p}}{dV_m} = -C_p r(t) \left[\frac{A_a}{C} e^{(V_m - E_r)/C} - \frac{A_b}{C} e^{(E_r - V_m)/C} \right] [1 + A_a e^{(V_m - E_r)/C} + A_b e^{(E_r - V_m)/C}] = 0 \quad (5.41)$$

To satisfy equation (5.41), it should have

$$\frac{A_a}{C} e^{(V_m - E_r)/C} - \frac{A_b}{C} e^{(E_r - V_m)/C} = 0 \quad (5.42)$$

Finally, it results in

$$V_m = E_r + \frac{1}{2} \ln\left(\frac{A_b}{A_a}\right)C \quad (5.43)$$

With V_m in (5.43), the maximum of $\frac{dp(t)}{dt}$ at time t can be achieved. This has completed the proof.

Figure 5.2 shows that $\max\{\frac{dp(t)}{dt}\}$ occurs at about $V_m = 46mV$ when the parameters in Table 5.1 are used.

Proposition 5.3 *For the CPG in (5.1)-(5.5), its equilibrium points are determined by*

$$a_0 V_m^2(\infty) + a_1 V_m(\infty) + a_2 = 0 \quad (5.44)$$

$$p(\infty) = \frac{A_a e^{[V_m(\infty) - E_r]/C}}{A_a e^{[V_m(\infty) - E_r]/C} + A_b e^{[E_r - V_m(\infty)]/C}} \quad (5.45)$$

$$C a_{nmda}(\infty) = \frac{\rho_{nmda}}{\delta_{nmda}} [E_{canmda} - V_m(\infty)] p(\infty) \quad (5.46)$$

where

$$a_0 = \frac{\rho_{nmda}}{G_{kca}} \delta_{nmda} \quad (5.47)$$

Figure 5.2: Relationship between $dp(t)/dt$ and $V_m(t)$.

$$a_1 = -G_{nmda} - \frac{\rho_{nmda} G_{kca} (E_{canmda} + E_{kca})}{\delta_{nmda}} - \frac{A_a e^{[V_m(\infty) - E_r]/C}}{A_a e^{[V_m(\infty) - E_r]/C} + A_b e^{[E_r - V_m(\infty)]/C}} \quad (5.48)$$

$$a_2 = G_{nmda} E_{nmda} + \frac{\rho_{nmda} G_{kca} E_{canmda} E_{kca}}{\delta_{nmda}} \quad (5.49)$$

Proof: It is obtained by setting zeros for all derivatives in (5.1)-(5.5).

Remark 5.2: With the parameters in Table 5.1, the equilibrium points are obtained by solving (5.44)-(5.46). There are two three-dimensional equilibrium points as point A with $V_m(\infty) = 34.3$, $p(\infty) = 0.2$ and $Ca_{nmda}(\infty) = 32.1$, and point B with $V_m(\infty) = 115.5$, $p(\infty) = 0.99$ and $Ca_{nmda}(\infty) = -38.8$. Clearly, according to Proposition 5.1, $p(\infty)$ should always be positive and $p(\infty) < 1$. The equilibrium points may be stable or unstable. In the unstable case, a sustained oscillation of the membrane potential may be generated. However, in order to obtain an oscillatory membrane potential, the selection of the parameters is important.

Proposition 5.4 Consider the CPG given in (5.1)-(5.5). With $R_m > 0$, $C_m > 0$,

$G_{nmda} > 0$, $G_{kca} > 0$, $\rho_{nmda} > 0$, the membrane potential is locally bounded, i.e., $E_{kca} \leq V_m(t) \leq E_{nmda}$, for $E_{kca} \leq V_m(0) \leq E_{nmda}$ and $Ca_{nmda}(0) > 0$ if $E_{canmda} > 0$ and $E_{canmda} > E_{nmda} > E_{kca}$.

Proof: As $0 < p(t) < 1$ for $-\infty < V_m(t) < \infty$, which is shown in Proposition 5.1, we need verify the boundness of $V_m(t)$ and $Ca_{nmda}(t)$. From (5.1), (5.2) and (5.4), it has

$$\frac{dV_m(t)}{dt} = \{G_{nmda}p(t)[E_{nmda} - V_m(t)] + G_{kca}Ca_{nmda}(t)[E_{kca} - V_m(t)] - V_m(t)/R_m\}/C_m \quad (5.50)$$

with

$$\frac{dCa_{nmda}(t)}{dt} = \rho_{nmda}p(t)[E_{canmda} - V_m(t)] - \delta_{nmda}Ca_{nmda}(t) \quad (5.51)$$

Then,

$$\frac{dV_m(t)}{dt} = \varphi(t)[\zeta(t) - V_m(t)] \quad (5.52)$$

$$\frac{dCa_{nmda}(t)}{dt} = \sigma(t) - \delta_{nmda}Ca_{nmda}(t) \quad (5.53)$$

where $\varphi(t) = \frac{G_{nmda}p(t) + G_{kca}Ca_{nmda}(t) + 1/R_m}{C_m}$, $\zeta(t) = \frac{G_{nmda}p(t)E_{nmda} + G_{kca}Ca_{nmda}(t)E_{kca}}{G_{nmda}p(t) + G_{kca}Ca_{nmda}(t) + 1/R_m}$ and $\sigma(t) = p(t)\rho_{nmda} [E_{canmda} - V_m(t)]$.

Clearly, if $0 < Ca_{nmda}(t) < \infty$ for all $t \geq 0$, we obtain $\varphi(t) > 0$ and $E_{kca} < \zeta(t) < E_{nmda}$. Therefore, according to Lemma 5.1, it can be concluded that for (5.52) $E_{kca} < V_m(t) < E_{nmda}$. Next, we will show by contradiction that $Ca_{nmda}(t) > 0$ for all $t \geq 0$ is satisfied.

Suppose that there exists a time t_1 representing the first time at which it has $Ca_{nmda}(t) = 0$. Since (5.53) is a continuous-time differential equation of Ca_{nmda} , obviously we must have $Ca_{nmda}(t) \geq 0$ during $0 \leq t \leq t_1$ due to $Ca_{nmda}(0) > 0$. In this case, as shown in the above it has $E_{kca} \leq V_m(t) \leq E_{nmda}$ for $E_{kca} \leq V_m(0) \leq E_{nmda}$.

As $0 < p(t) < 1$ is positive and $E_{canmda} > 0$ with $E_{canmda} > E_{nmda}$, it has

$$\sigma(t) = p(t)\rho_{nmda}[E_{canmda} - V_m(t)] > 0 \quad (5.54)$$

in (5.53). This ensures that $Ca_{nmda}(t)$ can not evolve into negative at $t = t_1$ for $Ca_{nmda}(0) > 0$, and it must remain in positive. Also, it must be bounded due to $0 < \sigma(t) < \infty$. Therefore, the assumption of $Ca_{nmda}(t) \leq 0$ at $t = t_1$ will not occur. This has completed the proof.

Proposition 5.5 *There exists a region Ω_u determined by*

$$Ca_{nmda}(t) < -\frac{G_{nmda}p(t)[E_{nmda} - V_m(t)] - \frac{1}{R_m}V_m(t)}{G_{kca}[E_{kca} - V_m(t)]} \quad (5.55)$$

$$Ca_{nmda}(t) > \frac{\rho_{nmda}}{\delta_{nmda}}p[E_{canmda} - V_m(t)] \quad (5.56)$$

so that the membrane potential $V_m(t)$ is unbounded if $\forall V_m(0)$ and $\forall Ca_{nmda}(0) \in \Omega_u$.

Proof: This can be checked with the above conditions (5.55)-(5.56). Let $V_m(t)$ approach positive infinity, then we find $Ca_{nmda}(t)$ goes to negative infinity.

Remark 5.3: This proposition shows that CPG may not provide a sustained oscillatory membrane potential if the initial conditions are inappropriately selected. In another word, it is not globally bounded. Figures 5.3 illustrates the unstable region for $V_m(t)$ and $Ca_{nmda}(t)$ with non-zero initial value of $p(t)$.

Remark 5.4: For the CPG given in (5.1)-(5.5), there exists a region Ω_s such that the CPG is bounded in Ω_s . Then, to generate an oscillatory membrane potential it is necessary that the equilibrium points in the region are unstable. According to Proposition 5.4, there would exist such a region Ω_s such that if $V_m(0)$ and $Ca_{nmda}(0)$ are in the region, then the membrane potential $V_m(t)$ is bounded. For the CPG given

Figure 5.3: Unbounded region is plotted in the plane of $V_m(t)$ and $Ca_{nmda}(t)$.

in (5.1)-(5.5) we have the Jacobian matrix $\Theta = [\theta_{ij}]$, where

$$\begin{aligned}\theta_{11} &= -\{G_{nmda}p(t) - G_{kca}Ca_{nmda}(t) - 1/R_m\}/C_m, & \theta_{12} &= G_{nmda}[E_{nmda} - V_m(t)]/C_m \\ \theta_{13} &= G_{kca}[E_{kca} - V_m(t)]/C_m, & \theta_{21} &= \{A_a e^{[V_m(t) - E_r]/C} [1 - p(t)] + A_b e^{[E_r - V_m(t)]/C} p(t)\}/C \\ \theta_{22} &= -A_a e^{[V_m(t) - E_r]/C} - A_b e^{[E_r - V_m(t)]/C}, & \theta_{23} &= 0 \\ \theta_{31} &= -\rho_{nmda}p(t), & \theta_{32} &= \rho_{nmda}[E_{canmda} - V_m(t)], & \theta_{33} &= -\delta_{nmda}\end{aligned}\quad (5.57)$$

If the Jacobian matrix Θ has unstable eigenvalues at the equilibrium points, which are located in the region Ω_s , then the CPG could not converge to the equilibrium $[V_m(\infty), Ca_{nmda}(\infty), p(\infty)]$ with the initial conditions $[V_m(0), Ca_{nmda}(0), p(0)]$ in the region. As the CPG is bounded in the region, it may generate an oscillatory membrane potential.

Remark 5.5: In the CPG, in order to generate an oscillatory membrane potential, the sigmoid function $p(t)$ is necessary, which actually plays as a switch. Incorporating it into $Ca_{nmda}(t)$, we can achieve two ways of switching. Roughly say, when the current of the N-methyl-D-aspartate current I_{nmda} starts to flow, the current of

calcium-dependent potassium I_{kca} is blocked initially and will be gradually released. As the later flows in opposite direction, when both currents reach the same value in their intensities, the increase of the membrane potential stops. After this state, I_{nmda} is gradually blocked, however, I_{kca} will continue to increase for some further time, which is assured through $E_{canmda} > E_{nmda}$ and appropriate selection of G_{nmda} , G_{kca} and ρ_{nmda} , then it starts decreasing. Therefore, this decrease in $V_m(t)$ can be maintained until it reaches a value near E_{kca} .

Remark 5.6: Selection of parameters is important to obtain a sustained oscillation of the membrane potential. Clearly, the Jacobian matrix Θ in (5.61) should have unstable eigenvalues upon the equilibrium point, which is located in the bounded region. The equilibrium points are determined from Proposition 5.3. Therefore, for the CPG, the following guidelines are established based on the results above.

1. For the equilibrium potentials, we should choose $E_{canmda} > E_{nmda} > E_{kca}$.

The actual equilibrium membrane potential $V_m(t)$ and ion channel potential E_{kca} and E_{nmda} without excitation should be negative. However, in this paper we aim to achieve positive membrane potential, so a bias value 80 has been added to the potentials. In this case, the action potential is always positive and it should be $V_m(t) \in (E_{kca}, E_{nmda})$.

2. For the parameters of $p(t)$, we should choose $A_a > 0$, $A_b > 0$, and $C > 0$.

When $V_m(t)$ is near E_{kca} , $p(t)$ should be very small near zero, therefore,

$$p(V_m = E_{kca}) \approx \frac{A_a e^{(E_{kca} - E_r)/C}}{A_a e^{(E_{kca} - E_r)/C} + A_b e^{(E_r - E_{kca})/C}} = \epsilon \quad (5.58)$$

where ϵ is a value near zero.

When $V_m(t)$ is near E_{nmda} , $p(t)$ should be near 1. That is

$$p(V_m = E_{nmda}) \approx \frac{A_a e^{(E_{nmda} - E_r)/C}}{A_a e^{(E_{nmda} - E_r)/C} + A_b e^{(E_r - E_{nmda})/C}} = \varsigma \quad (5.59)$$

where ς is a value near 1. Using this criterion, we can determine its parameters.

3. G_{nmda} , ρ_{nmda} and G_{kca} are positive constants and it has $G_{nmda} > \rho_{nmda}G_{kca}$. This ensures that the flow of I_{nmda} occurs first and increases rapidly compared with I_{kca} .
4. δ_{nmda} and ρ_{nmda} are two important parameters. They mainly determine the oscillation frequency. In general, increase in δ_{nmda} and ρ_{nmda} will increase the oscillatory frequency. However, when the ratio of δ_{nmda} and ρ_{nmda} reaches a certain large value, the oscillation will cease. In this case, both I_{nmda} and I_{kca} will emerge almost simultaneously, as a result, it will generate a stable output.

A set of the parameters of the CPG selected based on the guideline is given in Table 5.1

Table 5.1: Parameters for one neuron in CPG

Parameters	Value	Parameters	Value
R_m	60 $M\Omega$	C_m	0.045 pF
G_{nmda}	5 μS	G_{kca}	0.04 μS
E_{nmda}	80 mV	E_{canmda}	100 mV
E_{kca}	0 mV	E_r	10 mV
ρ_{nmda}	5.0 $mV^{-1}s^{-1}$	δ_{nmda}	2.0 s^{-1}
A_a	1.0 s^{-1}	A_b	72 s^{-1}
C	17 mV		

5.1.2 Simulation Results

Some simulation study is carried out to verify the properties established in the previous section and the parameters selected in Table 5.1. For the parameters given in Table 1, the Jacobian matrix Θ of (5.57) at the equilibrium points can be achieved. For equilibrium point A with $V_m(\infty) = 34.3$, $p(\infty) = 0.2$ and $Ca_{nmda}(\infty) = 32.1$,

Θ is given by

$$\Theta = \begin{bmatrix} -51.1 & 5077.8 & -30.5 \\ 0.5 & -33.4 & 0 \\ -1.0 & 328.5 & -2.0 \end{bmatrix} \quad (5.60)$$

Then, it has the eigenvalues of $\lambda_1 = -92.8418$ and $\lambda_{2,3} = 3.1751 \pm 3.8722i$. Note that the eigenvalues $\lambda_{2,3}$ are in the right half of the complex plane, so the equilibrium point A is unstable and the system may not converge to the equilibrium points and may generate oscillation if point A is in a bounded area.

For equilibrium point B with $V_m(\infty) = 115.5$, $p(\infty) = 0.99$ and $Ca_{nmda}(\infty) = -38.8$, Jacobian matrix Θ is given by

$$\Theta = \begin{bmatrix} 12.5 & -3944.4 & -102.7 \\ 0.4 & -21.4 & 0 \\ -1.0 & -177.5 & -2.0 \end{bmatrix} \quad (5.61)$$

It has the eigenvalues of $\lambda_1 = 5.2435$ and $\lambda_{2,3} = -8.0927 \pm 35.0748i$. There are also one eigenvalue λ_1 in the right half of the complex plane, so the equilibrium point B is not stable, too.

Next, we should check which area the two points are located in. If it is in a bounded area, then the system may generate oscillation; otherwise the system will diverge. Equilibrium point A is in the bounded area according to Proposition 5.4, and equilibrium point B is in the unbounded area according to Proposition 5.5. Therefore, it is possible to generate oscillation on equilibrium point A according to Proposition 5.6. We can check the two situations in plane of V_m and $Ca_{nmda}(t)$ as shown in Figure 5.4. Different initial conditions are selected. In the bounded area, a limit cycle appears on equilibrium point A , it indicates stable oscillation is generated. In the unbounded area where point B is located, the system diverges.

Figure 5.4: Bounded and unbounded regions in the plane of $Ca_{nmda}(t)$ and $V_m(t)$. Limit cycle indicates stable oscillation. Two equilibrium points A and B are labelled.

Figure 5.5 shows the relationship of $p(t)$ and $V_m(t)$. The oscillatory membrane potential $V_m(t)$ is shown in Figure 5.6 and it can be noted that $V_m(t) \in [0, E_{nmda}]$ and after one cycle $V_m(t) \in [E_{kca}, E_{nmda}]$. The relationship between $V_m(t)$ and $Ca_{nmda}(t)$ is shown in Figure 5.7, where the equilibrium points of $V_m(\infty)$ and $Ca_{nmda}(\infty)$ are also indicated. It can be seen that the equilibrium points are in the bounded region, and as they are unstable, the CPG generates oscillatory membrane potential.

The simulation result in Figure 5.8 shows that the frequency of the oscillatory membrane potential increases when δ_{nmda} and ρ_{nmda} increases. However, this increase has an upper limit and oscillation is ceased when it exceeds this limit. This is due to the equilibrium points in this case become stable, i.e., the Jacobian matrix Θ given in Remark 5.4 has all stable eigenvalues. The following is the calculation for $\delta_{nmda} = 25$ and $\rho_{nmda} = 25$. In this case, the equilibrium point A becomes $V_m(\infty) = 49.7324$, $Ca_{nmda}(\infty) = 30.0598$ and $p(\infty) = 0.5979$. The Jacobian matrix has then the eigenvalues of $\lambda_1 = -109.2$, and $\lambda_{2,3} = -2.09 \pm j6.29$. The eigenvalues

are all in the left half of the complex plane, so that the CPG with the initials in the bounded region will converge to the equilibrium points. Figure 5.9 shows the convergence of $V_m(t)$ and $Ca_{nmda}(t)$ from different initial points.

Figure 5.5: Relationship between $p(t)$ and $V_m(t)$ in stable oscillation.

In the mathematical model, the frequency may be changed by δ_{nmda} and ρ_{nmda} . However, in animal experiments, it is obtained by regulation of Mg^{2+} concentration. Their function in charge of frequency regulation is similar. The simulation result in Figure 5.8 shows good agreement with that shown in Figure 5.10.

5.1.3 Further Results

In the previous sections, we have established the properties of the CPG with the simplified version as it can be effectively used in the practical applications of the musculoskeletal control systems. In reality, the currents I_{na} , I_k and Ca_{ap} exist in the biological neuron and more and less they will affect the rhythmic locomotive

Figure 5.6: Membrane potential $V_m(t)$ in situation of stable oscillation.

Figure 5.7: Relationship between $V_m(t)$ and $Ca_{nmda}(t)$ in situation of stable oscillation.

Figure 5.8: Effect of δ_{nmda} and ρ_{nmda} on the oscillation frequency of CPG.

Figure 5.9: Convergence of $V_m(t)$ and $Ca_{nmda}(t)$ upon a stable equilibrium point.

Figure 5.10: Experimental data from [191]. The oscillatory frequency is varied with Mg^{2+} concentration.

patterns. As an extension, in this section, we will further investigate the properties of the CPG with the currents included. Therefore, we first introduce the currents presented in [14] and [42].

The following equations represent the currents of I_{na} , I_k and Ca_{ap} in one compartment of a neuron.

$$I_{na} = G_{na}m^3h[E_{na} - V_m(t)] \quad (5.62)$$

$$\frac{dm(t)}{dt} = \alpha_m[1 - m(t)] - \beta_m m(t) \quad (5.63)$$

$$\frac{dh(t)}{dt} = \alpha_h[1 - h(t)] - \beta_h h(t) \quad (5.64)$$

$$\alpha_m = \frac{A_{m1}[V_m(t) - B_{m1}]}{1 - e^{[B_{m1} - V_m(t)]/C_{m1}}}, \quad \beta_m = \frac{A_{m2}(B_{m2} - V_m(t))}{1 - e^{[V_m(t) - B_{m2}]/C_{m2}}} \quad (5.65)$$

$$\alpha_h = \frac{A_{h1}[B_{h1} - V_m(t)]}{1 - e^{[V_m(t) - B_{h1}]/C_{h1}}}, \quad \beta_h = \frac{A_{h2}}{1 + e^{[B_{h2} - V_m(t)]/C_{h2}}} \quad (5.66)$$

where $G_{na} > 0$ is the conductance. A_{mi} , B_{mi} , C_{mi} , A_{hi} , B_{hi} , and C_{hi} are constants.

$$I_k = G_k n^4 [E_k - V_m(t)] \quad (5.67)$$

$$\frac{dn(t)}{dt} = \alpha_n [1 - n(t)] - \beta_n n(t) \quad (5.68)$$

$$\alpha_n = \frac{A_{n1}[V_m(t) - B_{n1}]}{1 - e^{[B_{n1} - V_m(t)]/C_{n1}}}, \quad \beta_n = \frac{A_{n2}(B_{n2} - V_m(t))}{1 - e^{[V_m(t) - B_{n2}]/C_{n2}}} \quad (5.69)$$

where $G_k > 0$ is the conductance. A_{ni} , B_{ni} , C_{ni} are constants, and $Ca_{ap}(t)$ given in (4.9),

$$\frac{dq(t)}{dt} = \alpha_q [1 - q(t)] - \alpha_q q(t) \quad (5.70)$$

$$\alpha_q = \frac{A_{q1}[V_m(t) - B_{q1}]}{1 - e^{[B_{q1} - V_m(t)]/C_{q1}}}, \quad \beta_q = \frac{A_{q2}(B_{q2} - V_m(t))}{1 - e^{[V_m(t) - B_{q2}]/C_{q2}}} \quad (5.71)$$

where A_{qi} , B_{qi} , and C_{qi} are constants.

Remark 5.7: The function α_m given in (5.65), where $A_{m1} > 0$ and $C_{m1} > 0$, is monotonically increasing with respect to $V_m(t)$ as

$$\frac{d\alpha_m}{dV_m} = A_{m1} \frac{[(1 - (1 + \frac{V_m - B_{m1}}{C_{m1}})e^{(B_{m1} - V_m)/C_{m1}})]}{[1 - e^{(B_{m1} - V_m)/C_{m1}}]^2} > 0 \quad (5.72)$$

as

$$(1 + \frac{V_m - B_{m1}}{C_{m1}})e^{(B_{m1} - V_m)/C_{m1}} < 1$$

for $V_m \neq B_{m1}$ and $\frac{d\alpha_m}{dV_m} = 0$ when $V_m = B_{m1}$. Similarly, β_m in (5.65), where $A_{m2} > 0$ and $C_{m2} > 0$, is monotonically decreasing with respect to V_m . Figure 5.11 shows the changes of α_m and β_m (with $A_{m1} = 0.2$, $B_{m1} = 0$, $C_{m1} = 1.0$, $A_{m2} = 0.06$, $B_{m2} = -9.0$, $C_{m1} = 20.0$). It can be seen that similar characteristics are also for α_n , β_n , α_q and β_q .

Remark 5.8: In Proposition 5.1, we have verified that the boundness of $p(t)$. Here, there exist A_{mi} , B_{mi} , C_{mi} , A_{hi} , B_{hi} and C_{hi} such that we have $\alpha_m > 0$, $\beta_m > 0$,

Figure 5.11: Changes of α_m and β_m along with $V_m(t)$.

$\alpha_h > 0$ and $\beta_h > 0$ for $E_{kca} \leq V_m(t) \leq E_{na}$ in (5.65) and (5.66), where $E_{na} > E_{nmda}$ and $E_{kca} = E_k$. As a result, we have $0 < m(t) < 1$, and $0 < h(t) < 1$. Also there exist A_{ni} , B_{ni} , C_{ni} , A_{qi} , B_{qi} , and C_{qi} such that $0 < n(t) < 1$ and $0 < q(t) < 1$.

Proposition 5.6 *Consider the membrane potential*

$$C_m \frac{dV_m}{dt} = I_{na} + I_k + I_{nmda} + I_{kca} - \frac{V_m}{R_m} \quad (5.73)$$

where I_{na} , I_k , I_{nmda} and I_{kca} are defined in (5.62), (5.67), (4.5) and (4.7), respectively. Then, there exist a region Ω_b for $E_{kca} < E_{nmda} < E_{na}$ such that the membrane potential $V_m(t)$ is locally bounded, i.e., $E_{kca} \leq V_m(t) \leq E_{na}$ if the CPG starts in the region.

Proof: The proof procedure is similar to in Proposition 5.4. It can be shown that the membrane potential remains in $[E_{kca}, E_{na}]$, while $Ca_{nmda}(t) + Ca_{ap}(t) > 0$.

Remark 5.9: The simulation results presented in [14] show that the rhythmic locomotive patterns from the CPG including the voltage-dependent currents I_{na} and I_k make the oscillations contain many irregular spikes with small amplitude. This is due to the action potential from the two currents. However, for the motor neurons, they receive the rhythmic locomotive pattern which should be smoother as they have low-pass effects and there are many neurons of the same type driving the motoneurons. This means that the average spiking will be much more regular than the spikes of each individual neuron.

Remark 5.10: With the synaptic current I_{syn} given in (4.3) included, the CPG becomes a multi-neuron system, where interactions between the neurons may exist. In this case, in order to generate rhythmic locomotive patterns, it is necessary that at least one of the neurons should be in oscillation according to the theorem presented in [190]. The analysis can also be done by considering all neurons involved, however, it is an extremely difficult task as many coupled nonlinear differential equations are included in the system. So far all results are obtained based on simulation.

5.2 Neural Oscillator

In this section, theoretical analysis about neural oscillator will be given. In view of application, the neural oscillator seems easy to be implemented, but in fact, it is not an easy job because of its nonlinear and chaotic property. Another big problem is how to tune the parameters of the complex CPG network with multiple neural oscillators. At present, there are three methods to resolve these problems. First, the parameters are achieved according to the experience, and it is much like the trial-and-error method [44], [134], [194], [182]. Second, analytic methods are used to determine the parameters such as describing function [8], piecewise linear analysis [56], and contraction theory [170], [190]. Third, the appropriate parameters

are achieved through optimization methods, such as genetic algorithms (GA) [65], [91], [138], [142]. However, so far there are no clear guidelines and adaptive tuning methods for the CPG in realtime control.

In this section, some further exploration will be carried out based on the analytic methods. The analysis will be performed on determination of oscillatory frequency, boundness, stability and characteristics of the network. In the previous work, the effect of parameters on the oscillatory frequency of the neural oscillator was obtained by simulation [194], or roughly estimated by nonlinear analysis [8]. Here, a more precise relationship is developed based on the describing function. Although, the boundness and stability of the neural oscillator was studied in the previous work, this work aims to further explore these properties with piecewise analysis. In addition, the network property of multiple neural oscillators is analyzed.

The recurrent neural oscillator mentioned in Chapter 4, Matsuoka's oscillator, is very popular to simulate the CPG function [125]. It is adopted here for the theoretical analysis. Matsuoka's oscillator consists of two simulated neurons arranged in mutual inhibition, which can be described by the following mathematical equations

$$\tau_1 \dot{x}_1 = -x_1 - \beta v_1 - h y_2 + L s + r \quad (5.74)$$

$$\tau_2 \dot{v}_1 = -v_1 + y_1 \quad (5.75)$$

$$\tau_1 \dot{x}_2 = -x_2 - \beta v_2 - h y_1 - L s + r \quad (5.76)$$

$$\tau_2 \dot{v}_2 = -v_2 + y_2 \quad (5.77)$$

$$y_i = f(x_i) = \max(x_i, 0), i = 1, 2 \quad (5.78)$$

where x_i is the variable similar with the membrane voltage in a neuron, v_i is the membrane current of the slow recovery component, $f(x_i)$ is a nonlinear function, which has a unit gain for x_i when the input is non-negative and zero otherwise, y_i

is the neuronal output, h is the gain for the interaction between the two neurons, β is the adaptive gain for the self-inhibition of the neuron, s is the sensory feedback, r is the tonic input, and L is the feedback gain.

There are some important properties of the neural oscillator that will be explored in the following sections.

5.2.1 Determination of Frequency

Proposition 5.7 *For the neural oscillator given above, the free vibration frequency at which the oscillator oscillates, is determined by*

$$f = \frac{1}{2\pi} \sqrt{\frac{1 + (\frac{\beta}{h} - 1)(\frac{\tau_1}{\tau_2} + 1)}{\tau_1 \tau_2}} \quad (5.79)$$

Proof: The free vibration situation means that there are not any external signals applied to the neural oscillator. Therefore, the sensory feedback and tonic input are both set to zero. Based on the describing function method, by which the system stability and the critical frequency of oscillation can be determined, we can approximate the nonlinear functions $y_1 = f(x_1)$ and $y_2 = f(x_2)$ by

$$\begin{aligned} y_1(j\omega) &= F_1(X(j\omega)) = N_1(j\omega)X_1(j\omega) \\ y_2(j\omega) &= F_2(X(j\omega)) = N_2(j\omega)X_2(j\omega) \end{aligned} \quad (5.80)$$

Then, we have

$$\begin{aligned}
 (j\tau_1\omega + 1)X_1(j\omega) &= -\beta V_1(j\omega) - hN_2(j\omega)X_2(j\omega) \\
 (j\tau_2\omega + 1)V_1(j\omega) &= N_1(j\omega)X_1(j\omega) \\
 (j\tau_1\omega + 1)X_1(j\omega) &= -\beta V_2(j\omega) - hN_1(j\omega)X_1(j\omega) \\
 (j\tau_2\omega + 1)V_1(j\omega) &= N_2(j\omega)X_2(j\omega)
 \end{aligned} \tag{5.81}$$

Solving equation (5.81) for the characteristic equation, it results in

$$[A(j\omega) + \beta N_1(j\omega)][A(j\omega) + \beta N_2(j\omega)] - h^2 N_1(j\omega)(1 + j\omega\tau_2)^2 = 0 \tag{5.82}$$

where

$$A(j\omega) = (1 + j\omega\tau_1)(1 + j\omega\tau_2) \tag{5.83}$$

If $N_1(j\omega) = N_2(j\omega)$, which is true as the two neurons are symmetrical, then we obtain,

$$[A(j\omega) + \beta N_1(j\omega) + hN_1(j\omega)(1 + j\omega\tau_2)][A(j\omega) + \beta N_1(j\omega) - hN_1(j\omega)(1 + j\omega\tau_2)] = 0 \tag{5.84}$$

Clearly, for $\beta > 0$ and $h > 0$,

$$[A(j\omega) + \beta N_1(j\omega) + hN_1(j\omega)(1 + j\omega\tau_2)] = 0 \tag{5.85}$$

must be stable. In order to use Nyquist criterion, we rearrange the equation

$$A(j\omega) + \beta N_1(j\omega) - hN_1(j\omega)(1 + j\omega\tau_2) = 0 \tag{5.86}$$

It has

$$\frac{\beta - h(1 + j\omega\tau_2)}{A(j\omega)} = -\frac{1}{N_1(j\omega)} \tag{5.87}$$

To satisfy equation (5.87), it is necessary that the imaginary part of left side is zero, so it gives

$$(\beta - h)(\tau_1 + \tau_2) + h\tau_2(1 - \tau_1\tau_2\omega^2) = 0 \quad (5.88)$$

Solving equation (5.88) for ω results in

$$\omega = \sqrt{\frac{1 + (\frac{\beta}{h} - 1)(\frac{\tau_1}{\tau_2} + 1)}{\tau_1\tau_2}} \quad (5.89)$$

Remark 5.11: From the achieved result, we can see that the frequency is related with all the parameters in one neuron, τ_1 , τ_2 , β and h . However, if β equals h , the frequency f is the function of τ_1 and τ_2 :

$$f = \frac{1}{2\pi} \sqrt{\frac{1}{\tau_1\tau_2}} \quad (5.90)$$

Their relationship is illustrated in Figure 5.12. This property states that the frequency is mainly determined by τ_1 and τ_2 . For example, the frequency can be increased by decreasing them and can also be decreased through increasing them.

5.2.2 Boundness

Proposition 5.8 *Consider the neural oscillator given by equations (5.74)-(5.78), with the sensory feedback s bounded by $s_{min} < s < s_{max}$. Then the states of the neural oscillator are bounded.*

Proof: It is proved based on the work in [125]. The objective is that we will extend this theory from a single neural oscillator to a network of multiple neural oscillators in Proposition 5.12. The proof proceeds by integrating each of the neural oscillator equations in turn and determining maximum and minimum values for the state given the oscillator parameters and a starting state $(x_1(0), v_1(0), x_2(0), v_2(0))^T$.

Figure 5.12: Output frequency of the neural oscillator with regard to $1/\tau_1$ and $1/\tau_2$ in the situation with $\beta = h$.

Integrating (5.75) gives

$$v_1(t) = v_1(0)e^{-t/\tau_2} + \frac{1}{\tau_2}e^{-t/\tau_2} \int_0^t y_1(u)e^{u/\tau_2} du \quad (5.91)$$

Since $y_1 > 0$, it has

$$v_1(t) \geq -|v_1(0)| \quad (5.92)$$

Equation (5.92) gives the lower bound for $v_1(t)$. Similarly, the lower bound for $v_2(t)$ is given by

$$v_2(t) \geq -|v_2(0)| \quad (5.93)$$

Now integrating (5.74) results in

$$\begin{aligned} x_1(t) = & x_1(0)e^{-t/\tau_1} + r(1 - e^{-t/\tau_1}) - \frac{\beta}{\tau_1}e^{-t/\tau_1} \int_0^t v_1(u)e^{u/\tau_1} du \\ & - \frac{h}{\tau_1}e^{-t/\tau_1} \int_0^t y_2(u)e^{u/\tau_1} du + \frac{L}{\tau_1}e^{-t/\tau_1} \int_0^t se^{u/\tau_1} du \end{aligned} \quad (5.94)$$

Since $y_2 > 0$, it can be seen that

$$x_1(t) \leq |x_1(0)| + r - \frac{\beta}{\tau_1} e^{-t/\tau_1} \int_0^t v_1(u) e^{u/\tau_1} du + Ls_{max} \quad (5.95)$$

The maximum value of $x_1(t)$ can then be calculated by applying (5.92)

$$\begin{aligned} x_1(t) &\leq |x_1(0)| + r + \frac{\beta}{\tau_1} e^{-t/\tau_1} \int_0^t |v_1(0)| e^{u/\tau_1} du + Ls_{max} \\ &= |x_1(0)| + r + \beta e^{-t/\tau_1} |v_1(0)| (e^{t/\tau_1} - 1) + Ls_{max} \\ &\leq |x_1(0)| + r + \beta |v_1(0)| + Ls_{max} \end{aligned} \quad (5.96)$$

In fact, (5.96) represents the upper bound of $x_1(t)$. In a similar way, the maximum value of $x_2(t)$ is thus given by

$$x_2(t) \leq |x_2(0)| + r + \beta |v_2(0)| - Ls_{min} \quad (5.97)$$

The maximum value of $v_1(t)$, representing its upper bound, can be found by applying (5.96) to (5.91)

$$\begin{aligned} v_1(t) &\leq |v_1(0)| + \max \left[\frac{1}{\tau_2} e^{-t/\tau_2} \int_0^t y_1(u) e^{u/\tau_2} du \right] \\ &= |v_1(0)| + \frac{1}{\tau_2} e^{-t/\tau_2} \int_0^t (|x_1(0)| + r + \beta |v_1(0)|) e^{u/\tau_2} du \\ &\leq (1 + \beta) |v_1(0)| + |x_1(0)| + Ls_{max} + r \end{aligned} \quad (5.98)$$

For v_2 , it has

$$v_2(t) \leq (1 + \beta) |v_2(0)| + |x_2(0)| - Ls_{min} + r \quad (5.99)$$

To determine the minimum value of $x_1(t)$, we use equation (5.94)

$$\begin{aligned}
x_1(t) &\geq -|x_1(0)| - \max\left[\frac{\beta}{\tau_1}e^{-t/\tau_1} \int_0^t v_1(u)e^{u/\tau_1} du\right] - \max\left[\frac{h}{\tau_1}e^{-t/\tau_1} \int_0^t y_2(u)e^{u/\tau_1} du\right] \\
&\quad + \min\left[\frac{L}{\tau_1}e^{-t/\tau_1} \int_0^t s(u)e^{u/\tau_1} du\right] \\
&= -|x_1(0)| - \frac{\beta}{\tau_1}e^{-t/\tau_1} \int_0^t ((1+\beta)|v_1(0)| + |x_1(0)| + r)e^{u/\tau_1} du \\
&\quad - \frac{h}{\tau_1}e^{-t/\tau_1} \int_0^t (|x_2(0)| + r + \beta|v_2(0)|)e^{u/\tau_1} du + \frac{L}{\tau_1}e^{-t/\tau_1} \int_0^t s_{min}e^{u/\tau_1} du \\
&\geq -|x_1(0)| - \beta[(1+\beta)|v_1(0)| + |x_1(0)| + r + Ls_{max}] \\
&\quad - h(|x_2(0)| + r + \beta|v_2(0)| - Ls_{min}) + Ls_{min}
\end{aligned} \tag{5.100}$$

Re-arranging (5.100), and we have

$$x_1(t) \geq -(1+\beta)|x_1(0)| - \beta(1+\beta)|v_1(0)| - h|x_2(0)| - h\beta|v_2(0)| - (\beta+h)r + (h+1)Ls_{min} - \beta Ls_{max} \tag{5.101}$$

It is similar with $x_2(t)$, i.e.

$$x_2(t) \geq -(1+\beta)|x_2(0)| - \beta(1+\beta)|v_2(0)| - h|x_1(0)| - h\beta|v_1(0)| - (\beta+h)r - (\beta+1)Ls_{max} + Ls_{min} \tag{5.102}$$

This has completed the proof.

5.2.3 Stability and Oscillation

In order to study the stability of the neural oscillator, we arrange equations (5.74)-(5.78) into the matrix form as

$$\begin{bmatrix} \dot{x}_1 \\ \dot{v}_1 \\ \dot{x}_2 \\ \dot{v}_2 \end{bmatrix} = \begin{bmatrix} -\frac{1}{\tau_1}x_1 - \frac{\beta}{\tau_1}v_1 - \frac{h}{\tau_1}f(x_2) + \frac{1}{\tau_1}(r - Ls) \\ -\frac{1}{\tau_2}v_1 + \frac{f(x_1)}{\tau_2} \\ -\frac{1}{\tau_1}x_2 - \frac{\beta}{\tau_1}v_2 - \frac{h}{\tau_1}f(x_1) + \frac{1}{\tau_1}(r + Ls) \\ -\frac{1}{\tau_2}v_2 + \frac{f(x_2)}{\tau_2} \end{bmatrix} \quad (5.103)$$

$$\begin{bmatrix} y_1 \\ y_2 \end{bmatrix} = \begin{bmatrix} f(x_1) \\ f(x_2) \end{bmatrix} \quad (5.104)$$

As $f(x_i) = \max(x_i, 0)$, $i = 1, 2$, we have $f(x_i) = u(x_i)x_i$, and $u(x_i)$ is defined by

$$u(x_i) = \begin{cases} 1 & x_i \geq 0 \\ 0 & x_i < 0 \end{cases} \quad (5.105)$$

Using the piece-wise linear method, we can transform equation (5.103) into the form of state space such as

$$\begin{aligned} \dot{\mathbf{x}} &= \mathbf{Ax} + \mathbf{b} \\ \mathbf{y} &= \mathbf{Cx} \end{aligned} \quad (5.106)$$

It is well known that CPG can independently generate rhythmic motor patterns in the absence of sensory feedback [124], especially, the stability of the neural oscillator is not influenced by it. Therefore, the sensory feedback is not considered in this work,

i.e. $L = 0$. Then, equation (5.106) can be illustrated in detail as follows

$$\begin{bmatrix} \dot{x}_1 \\ \dot{v}_1 \\ \dot{x}_2 \\ \dot{v}_2 \end{bmatrix} = \begin{bmatrix} -\frac{1}{\tau_1} & -\beta\frac{1}{\tau_1} & -\frac{h}{\tau_1}u(x_2) & 0 \\ \frac{1}{\tau_2}u(x_1) & -\frac{1}{\tau_2} & 0 & 0 \\ -\frac{h}{\tau_1}u(x_1) & 0 & -\frac{1}{\tau_1} & -\frac{\beta}{\tau_1} \\ 0 & 0 & \frac{1}{\tau_2}u(x_2) & -\frac{1}{\tau_2} \end{bmatrix} \begin{bmatrix} x_1 \\ v_1 \\ x_2 \\ v_2 \end{bmatrix} + \begin{bmatrix} \frac{r}{\tau_1} \\ 0 \\ \frac{r}{\tau_1} \\ 0 \end{bmatrix} \quad (5.107)$$

$$\begin{bmatrix} y_1 \\ y_2 \end{bmatrix} = \begin{bmatrix} u(x_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & u(x_2) \end{bmatrix} \begin{bmatrix} x_1 \\ v_1 \\ x_2 \\ v_2 \end{bmatrix} \quad (5.108)$$

After the transformation, now we have the following results.

Lemma 5.2: *For the neural oscillator given in (5.106), it can be transformed into*

$$\dot{\mathbf{z}} = (\mathbf{I}\lambda)\mathbf{z} + \mathbf{P}\mathbf{b} \quad (5.109)$$

with

$$\mathbf{z} = \mathbf{P}\mathbf{x} = \mathbf{P} \begin{bmatrix} x_1 & v_1 & x_2 & v_2 \end{bmatrix}^T \quad (5.110)$$

$$\lambda = \text{diag} \begin{bmatrix} \lambda_1 & \lambda_2 & \lambda_3 & \lambda_4 \end{bmatrix} \quad (5.111)$$

Proof: It is proved as follows. Solving the characteristic equation

$$\det(\lambda\mathbf{I} - \mathbf{A}) = 0 \quad (5.112)$$

then, the eigenvalues are achieved as

$$\begin{aligned}
 \lambda_1 &= -\frac{1}{2}\epsilon_1 + \frac{1}{2}\sqrt{\epsilon_1^2 - 4\frac{1 + \beta u(x_1) - hu(x_1)u(x_2)}{\tau_1\tau_2}} \\
 \lambda_2 &= -\frac{1}{2}\epsilon_1 - \frac{1}{2}\sqrt{\epsilon_1^2 - 4\frac{1 + \beta u(x_1) - hu(x_1)u(x_2)}{\tau_1\tau_2}} \\
 \lambda_3 &= -\frac{1}{2}\epsilon_2 + \frac{1}{2}\sqrt{\epsilon_2^2 - 4\frac{1 + \beta u(x_2) + hu(x_1)u(x_2)}{\tau_1\tau_2}} \\
 \lambda_4 &= -\frac{1}{2}\epsilon_2 - \frac{1}{2}\sqrt{\epsilon_2^2 - 4\frac{1 + \beta u(x_2) + hu(x_1)u(x_2)}{\tau_1\tau_2}}
 \end{aligned} \tag{5.113}$$

where, $\epsilon_1 = \frac{1-hu(x_1)u(x_2)}{\tau_1} + \frac{1}{\tau_2}$ and $\epsilon_2 = \frac{1+hu(x_2)u(x_2)}{\tau_1} + \frac{1}{\tau_2}$. The general form of the transform matrix P relating to λ is too complex. Here, we just show P in the case of $u(x_1) = u(x_2) = 1$ as follows

$$\mathbf{P} = \begin{bmatrix} p_{11} & p_{12} & p_{13} & p_{14} \\ 1 & 1 & -1 & -1 \\ p_{31} & p_{32} & p_{33} & p_{34} \\ 1 & 1 & 1 & 1 \end{bmatrix} \tag{5.114}$$

The eigenvectors in $\mathbf{P}$ are given by

$$\begin{aligned}
 p_{11} &= p_{31} = \frac{\tau_1 - \tau_2(1-h)}{2\tau_1} + \frac{\tau_2}{2}\sqrt{\epsilon_1^2 - 4\frac{1 + \beta - h}{\tau_1\tau_2}} \\
 p_{12} &= p_{32} = \frac{\tau_1 - \tau_2(1-h)}{2\tau_1} - \frac{\tau_2}{2}\sqrt{\epsilon_1^2 - 4\frac{1 + \beta - h}{\tau_1\tau_2}} \\
 p_{13} &= -p_{31} = \frac{\tau_2(1+h) - \tau_1}{2\tau_1} + \frac{\tau_2}{2}\sqrt{\epsilon_2^2 - 4\frac{1 + \beta + h}{\tau_1\tau_2}} \\
 p_{14} &= -p_{41} = \frac{\tau_1 - \tau_2(1+h)}{2\tau_1} - \frac{\tau_2}{2}\sqrt{\epsilon_2^2 - 4\frac{1 + \beta + h}{\tau_1\tau_2}}
 \end{aligned} \tag{5.115}$$

Proposition 5.9: For the neural oscillator given in (5.74)-(5.78), it is stable, i.e.

the states of the oscillator converge as $t \rightarrow \infty$, if

$$\begin{aligned}\beta &> h - 1 \\ h &< 1 + \frac{\tau_1}{\tau_2}\end{aligned}$$

Proof: In order to study stability of the neural oscillator based on Lyapunov method, we define a Lyapunov function,

$$\mathbf{V}(\mathbf{t}) = [\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}]^T[\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}] \quad (5.116)$$

where $\mathbf{x}$ is the state of the neural oscillator (5.106). Then, the derivative of (5.116) is given by

$$\dot{\mathbf{V}}(\mathbf{t}) = [\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}]^T[\mathbf{A}^T + \mathbf{A}][\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}] \quad (5.117)$$

Clearly, if all eigenvalues of A given in (5.113) are stable .i.e. in the left half complex plane, it has

$$\dot{\mathbf{V}}(\mathbf{t}) = [\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}]^T[\mathbf{A}^T + \mathbf{A}][\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}] < 0 \quad (5.118)$$

and the oscillator asymptotically converges. However, due to the existence of the nonlinear part $u(x_i)$, we cannot resolve (5.118) directly. One possible way is to use piecewise linear method. As there are four variable cases of $u(x_i)$, equation (5.118) can be resolved in the following four cases.

For $u(x_1) = u(x_2) = 1$, we get two sets of solutions: $\beta > h - 1$ and $h < 1 + \frac{\tau_1}{\tau_2}$; $\beta > -h - 1$ and $h > -1 - \frac{\tau_1}{\tau_2}$, both of which provide stable eigenvalues for (5.113). In this case, the two neurons in the neural oscillator are inhibitory, i.e. the connection between them is negative, so h must be positive. Therefore, $\beta > h - 1$ and $h < 1 + \frac{\tau_1}{\tau_2}$ are meaningful.

For $u(x_1) = 1, u(x_2) = 0$ and $u(x_1) = 0, u(x_2) = 1$, we get the stable eigenvalues of A , if $\beta > -1$. As β is always positive for self-inhibition, the oscillator in this case is also stable.

For $u(x_1) = 0, u(x_2) = 0$, the oscillator is always stable.

In conclusion, the neural oscillator is stable in the situation of $\beta > h - 1$ and $h < 1 + \frac{\tau_1}{\tau_2}$.

This has completed the proof.

Proposition 5.10: *For the neural oscillator given in (5.74)-(5.78), if*

$$\begin{aligned}\beta &> h - 1 \\ h &> 1 + \frac{\tau_1}{\tau_2} \\ r &\neq 0\end{aligned}\tag{5.119}$$

then the neural oscillator can generate oscillation.

Proof: In a way similar with the proof of Property 5.9, it can be verified that when condition (5.119) is met and if the oscillator has no constraints, i.e. $u(x_i) = 1$, we have

$$\dot{\mathbf{V}}(\mathbf{t}) = [\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}]^T[\mathbf{A}^T + \mathbf{A}][\mathbf{x} + \mathbf{A}^{-1}\mathbf{b}] > 0\tag{5.120}$$

which means the oscillator is unstable. However, with the constraint:

$$f(x_i) = \max(x_i, 0)\tag{5.121}$$

The neural oscillator will not be divergent, and the states are bounded. It is verified by contradiction as follows.

Assume that the neural oscillator is unstable with the constraints $f(x_i) = \max(x_i, 0)$, then after certain time, $|x_i|$ becomes very large and we say $|x_i| \rightarrow \infty$. Arbitrarily,

we select x_1 for consideration. Firstly we assume x_1 is negative infinite, then the constraint is $f(x_1) = 0$. In this case, we get the state equation for both neurons:

$$\begin{bmatrix} \dot{x}_1 \\ \dot{v}_1 \end{bmatrix} = \begin{bmatrix} -\frac{1}{\tau_1} & -\beta\frac{1}{\tau_1} \\ 0 & -\frac{1}{\tau_2} \end{bmatrix} \begin{bmatrix} x_1 \\ v_1 \end{bmatrix} + \begin{bmatrix} -\frac{h}{\tau_1}u(x_2)x_2 + \frac{r}{\tau_1} \\ 0 \end{bmatrix} \quad (5.122)$$

$$\begin{bmatrix} \dot{x}_2 \\ \dot{v}_2 \end{bmatrix} = \begin{bmatrix} -\frac{1}{\tau_1} & -\beta\frac{1}{\tau_1} \\ 0 & -\frac{1}{\tau_2} \end{bmatrix} \begin{bmatrix} x_2 \\ v_2 \end{bmatrix} + \begin{bmatrix} \frac{r}{\tau_1} \\ 0 \end{bmatrix} \quad (5.123)$$

From equation (5.123) we can see that neuron 2 is strictly stable and therefore, the state x_2 converges to the tonic input r . Also, in equation (5.122), x_2 can be considered as an external input converging to r since neuron 1 in this case has no influence to neuron 2. Therefore, x_1 converges to $-hu(x_2)x_2 + r$, which actually contradicts the assumption of negative infinite of x_1 . In the other words, the state x_1 could not reach negative infinite. In another case, assume that x_1 is positive infinite. Since in this case x_2 approaches $-x_1$, it has $f(x_2) = 0$ when $x_2 \leq 0$. This shows neuron 2 will have no influence to neuron 1 when $x_2 \leq 0$ and therefore, x_1 will converge to the tonic input r . This means that the assumption of positive infinite of x_1 is not true.

As a conclusion, this situation $|x_i| \rightarrow \infty$ cannot exist with the constraint $f(x_i)$, and x_i must be finite. Moreover, during $f(x_2) = 0$, x_1 follows r , while during $f(x_1) = 0$, x_2 follows r . As the system is unstable, but the states are bounded and alternate, there can only exist one critical situation: oscillation. Under the initial imbalance conditions of x_1 and x_2 , the neural oscillator can generate oscillation.

This has completed the proof.

5.2.4 Local Stability

For the recurrent neural oscillator given in (5.74)-(5.78), the nonlinear function $y_i = \max(x_i, 0)$ is just as a switch. Therefore, this system can also be looked on as a piecewise linear system and piecewise linear method is adopted to analyze the local stability of the neural oscillator. Assume the switching time is t , and at that time, the switching surface is given by $S_i = \bar{C}_i x + d_i$, where $\bar{C}_i$ is a row vector and $i = 1, 2, \dots, N$.

Assume the initial switch occurs at time t_1^* on the switching surface S_1 , and the state of neural oscillator is x_1^* at this moment. After a period of t_2^* , the next switch occurs with the switching surface S_2 . There are four switches during one cycle for the neural oscillator here, and the rest may be deduced by analogy (see Figure 5.13). By integrating the state equations in (5.106), the trajectories between the two neighboring switches can be obtained as

$$x_2^* = x(t_2^*) = e^{A_1 t_1^*} (x_1^* + A_1^{-1} b_1) - A_1^{-1} b_1 \quad (5.124)$$

$$x_3^* = x(t_3^*) = e^{A_2 t_2^*} (x_2^* + A_2^{-1} b_2) - A_2^{-1} b_2 \quad (5.125)$$

$$x_4^* = x(t_4^*) = e^{A_3 t_3^*} (x_3^* + A_3^{-1} b_3) - A_3^{-1} b_3 \quad (5.126)$$

Note that $x_1^* = x_5^*$, thus,

$$x_1^* = x_5^* = x(t_4^*) = e^{A_4 t_4^*} (x_4^* + A_4^{-1} b_4) - A_4^{-1} b_4 \quad (5.127)$$

By iterative computation of (5.124)-(5.127) and considering the equations of the switching surface, x_i^* and t_i^* can be obtained. However, this computation method needs to resolve several transcendental equations, it is too complex. An appropriate method to get them is from a numerical computation.

Figure 5.13: Limit cycle of the neural oscillator in x_1 - x_2 plane.

It is well known that the stable nonlinear system exhibits multiple equilibrium points or limit cycles. With regard to the neural oscillator presented above, it has a limit cycle. Though it is hard to prove the existence of the limit cycles, the local stability of the limit cycles can be proved and it is defined by *Poincaré* map (refer to Appendix). Consider the *Poincaré* from some points in a small neighborhood of x_1^* in S_1 , to the point where the trajectory returns to S_1 , the limit cycle is locally stable if the eigenvalues of the Jacobian of P are inside the unit disk.

Proposition 5.11 *Consider the neural oscillator given in (5.74)-(5.78). Assume there exists a limit cycle γ with period t^* . The jacobian of the Poincaré map P is given by $W = W_4 W_3 W_2 W_1$ where*

$$W_i = \left(I - \frac{v_i \bar{C}_{i+1}}{\bar{C}_{i+1} v_i}\right) e^{A_i t_i^*} \quad (5.128)$$

with $v_i = A_i x_{i+1}^* + b_i$, $i = 1, 2, 3, 4$. $\bar{C}_i$ is the coefficient matrix in switching surface. The limit cycle γ is locally stable if W has all its eigenvalues inside the unit disk.

It is unstable if at least one of the eigenvalues of W is outside the unit disk.

The detailed proof is referred to [9], [56].

Remark 5.12: Using this property, we can verify the local stability of the neural oscillator. Arbitrarily, the parameters of the neural oscillator are given by $\tau_1 = 0.127$, $\tau_2 = 0.2$, $h = 2.5$, $\beta = 2.5$, $c = 1$ and $e = 0$. The switch is determined by x_1 and x_2 , and the states equations between each switch are calculated according to (5.103). The values of A and C are shown in Figure 5.14. The value of matrix b_i is the same for the four quadrants, $b = [7.874 \ 0 \ 7.874 \ 0]^T$. The times required

Figure 5.14: Values of matrices A and C in the state equation of neural oscillator. There exist four switches per cycle due to x_1 and x_2 . The states are projected onto $x_1 - x_2$ plane in four quadrants.

for switching from one surface to the next are obtained through the simulation as shown in Figure 5.15. Note that the curves are not regular in the first cycle due to the transient property of the neural oscillator, therefore, the calculation is performed

Figure 5.15: Plots of the states x_1 and x_2 in the time domain. The times between the switching surfaces ($t_1^*-t_4^*$) are labelled.

after the first cycle, and they are arbitrarily selected in the second cycle.

$$t_1^* = 0.147, t_2^* = 0.353, t_3^* = 0.147, t_4^* = 0.353 \quad (5.129)$$

The corresponding states x^* on each switching surface are given by

$$\begin{aligned} x_1^* &= [0.269 \quad 0.303 \quad 0 \quad 0.038]^T \\ x_2^* &= [0 \quad 0.224 \quad 0.334 \quad 0.107]^T \\ x_3^* &= [0 \quad 0.038 \quad 0.269 \quad 0.303]^T \\ x_4^* &= [0.334 \quad 0.107 \quad 0 \quad 0.224]^T \end{aligned} \quad (5.130)$$

and $\bar{C}_i$ of the switching surface is given by

$$\begin{aligned}\bar{C}_1 &= [0 \ 0 \ 1 \ 0] \\ \bar{C}_2 &= [1 \ 0 \ 0 \ 0] \\ \bar{C}_3 &= [1 \ 0 \ 0 \ 0] \\ \bar{C}_4 &= [0 \ 0 \ 1 \ 0]\end{aligned}\tag{5.131}$$

Note that the limit cycle of the neural oscillator only crosses three quadrants of x_1 vs. x_2 plane (see Figure 5.13). The sequence is A_I , A_{IV} , A_I , and A_{II} . Therefore, we get the corresponding relationship: $A1 = A_I$, $A2 = A_{IV}$, $A3 = A_I$, and $A4 = A_{II}$. The values are shown in Figure 5.14. Then, $W_1 - W_4$ can be computed and finally, W is obtained as

$$W = \begin{bmatrix} -0.0463 & -0.1329 & -0.0123 & 0.0954 \\ 0.0121 & -0.0340 & -0.0211 & -0.0007 \\ 0 & 0 & 0 & 0 \\ 0.0006 & -0.0071 & -0.0029 & -0.0021 \end{bmatrix}\tag{5.132}$$

Therefore, the eigenvalues of W are

$$\begin{bmatrix} -0.0391 + 0.0378i \\ -0.0391 - 0.0378i \\ -0.0043 \\ 0 \end{bmatrix}\tag{5.133}$$

Obviously, all the eigenvalues are inside the unit disk. According to Proposition 5.11, the limit cycle is locally stable. As an illustration, we plot the states in $x_1 - x_2$ plane with different initial conditions. It can be seen from Figure 5.16, that all the trajectories converge to the limit cycle asymptotically.

(a)

(b)

Figure 5.16: Convergence of the neural oscillator in x_1 - x_2 plane with different initial values in different quadrants.

5.2.5 Network Property

The propositions above are all related with single oscillator. Next, we will make some exploration on the network property of the multiple oscillators. Proposition 5.8 states that the neural oscillator with two neurons generates bounded states and bounded outputs. Here, we extend this result to multiple neural oscillators, which are fully interconnected. For the n -th order multiple neural oscillators, they can be described by

$$\tau_{i1}\dot{x}_{i1} = -x_{i1} - \beta_i v_{i1} - h_i y_{i2} + r_i - \sum_{j=1, j \neq i}^n (p_{ij}^1 y_{j1} + q_{ij}^1 y_{j2}) + L s_i \quad (5.134)$$

$$\tau_{i2}\dot{v}_{i1} = -v_{i1} + y_{i1} \quad (5.135)$$

$$\tau_{i1}\dot{x}_{i2} = -x_{i2} - \beta_i v_{i2} - h_i y_{i1} + r_i - \sum_{j=1, j \neq i}^n (p_{ij}^2 y_{j1} + q_{ij}^2 y_{j2}) - L s_i \quad (5.136)$$

$$\tau_{i2}\dot{v}_{i2} = -v_{i2} + y_{i2} \quad (5.137)$$

$$y_{i1} = f(x_{i1}) = \max(x_{i1}, 0)$$

$$y_{i2} = f(x_{i2}) = \max(x_{i2}, 0) \quad (5.138)$$

$$i = 1, 2, \dots, n$$

where n indicates the total number of the oscillators, each of which is consisted of 2 neurons. Note that x_{i1} and x_{i2} represent the two states of the i th oscillator. τ_{i1} and τ_{i2} are the time constants. p_{ij}^1 , q_{ij}^1 , p_{ij}^2 and q_{ij}^2 are the weights for the connections amongst the neural oscillators. s_i is the sensory feedback with the gain L . r_i is the tonic input. For the multiple oscillators given by (5.134)-(5.137), we have the following result.

Proposition 5.12 *Consider the multiple neural oscillators given by the equations*

(5.134)-(5.137), the neural oscillator states x_{i1} , v_{i1} , x_{i2} and v_{i2} are bounded if

(1)

$$-h_i y_{i2} + L s_{i1} \leq \sum_{j=1, j \neq i}^n (p_{ij}^1 y_{j1} + q_{ij}^1 y_{j2}) + c_{i1} \quad (5.139)$$

(2)

$$-h_i y_{i1} - L s_{i2} \leq \sum_{j=1, j \neq i}^n (p_{ij}^2 y_{j1} + q_{ij}^2 y_{j2}) + c_{i2} \quad (5.140)$$

(3) The sensory feedback s_i is bounded, i.e. $s_{imin} \leq s_i \leq s_{imax}$.

where c_{i1} and c_{i2} are constants.

Proof: First we verify the boundness of v_{i1} and v_{i2} . Solving (5.135) yields

$$v_{i1}(t) = v_{i1}(0)e^{-t/\tau_{i2}} + \frac{1}{\tau_{i2}}e^{-t/\tau_{i2}} \int_0^t y_{i1}(u)e^{u/\tau_{i2}} du \quad (5.141)$$

Clearly, we have

$$-|v_{i1}(0)| \leq v_{i1}(t) \leq v_{i1}(0) + \max\left[\frac{1}{\tau_{i2}}e^{-t/\tau_{i2}} \int_0^t y_{i1}(u)e^{u/\tau_{i2}} du\right] \quad (5.142)$$

Similarly, for v_{i2}

$$-|v_{i2}(0)| \leq v_{i2}(t) \leq v_{i2}(0) + \max\left[\frac{1}{\tau_{i2}}e^{-t/\tau_{i2}} \int_0^t y_{i2}(u)e^{u/\tau_{i2}} du\right] \quad (5.143)$$

Equation (5.142) shows that v_{i1} is bounded if

$$\max\left[\frac{1}{\tau_{i2}}e^{-t/\tau_{i2}} \int_0^t y_{i1}(u)e^{u/\tau_{i2}} du\right]$$

is bounded. Now, we verify the boundness of x_{i1} and x_{i2} . From (5.134), we obtain

$$x_{i1}(t) = x_{i1}(0)e^{-t/\tau_{i1}} + r_i(1 - e^{-t/\tau_{i1}}) - \frac{\beta_i}{\tau_{i1}}e^{-t/\tau_{i1}} \int_0^t v_{i1}(u)e^{u/\tau_{i1}} du - \frac{h_i}{\tau_{i1}}e^{-t/\tau_{i1}} \int_0^t y_{i2}(u)e^{u/\tau_{i1}} du$$

$$+\frac{1}{\tau_{i1}}e^{-t/\tau_{i1}}\int_0^t\left[\sum_{j=1,j\neq i}^n(p_{ij}^1y_{j1}(u)+q_{ij}^1y_{j2}(u))\right]du+Ls_i \quad (5.144)$$

Applying the condition of (5.139) into (5.144) leads to

$$x_{i1}(t)\leq|x_{i1}(0)|+r_i-\frac{\beta_i}{\tau_{i1}}e^{-t/\tau_{i1}}\int_0^tv_{i1}(u)e^{u/\tau_{i1}}du+Ls_i+c_{i1} \quad (5.145)$$

Using (5.142), we have

$$x_{i1}(t)\leq|x_{i1}(0)|+r_i+\beta_i|v_{i1}(0)|+Ls_{imax}+c_{i1} \quad (5.146)$$

and,

$$x_{i2}(t)\leq|x_{i2}(0)|+r_i+\beta_i|v_{i2}(0)|-Ls_{imin}+c_{i2} \quad (5.147)$$

Applying (5.146) and (5.147) into (5.142) and (5.143), we obtain

$$-|v_{i1}(0)|\leq v_{i1}(t)\leq(1+\beta_i)v_{i1}(0)+x_{i1}(0)+r_i+Ls_{max} \quad (5.148)$$

and

$$-|v_{i2}(0)|\leq v_{i2}(t)\leq(1+\beta_i)v_{i2}(0)+x_{i2}(0)+r_i-Ls_{imin} \quad (5.149)$$

According to (5.144), it has,

$$\begin{aligned} x_{i1}(t) &\geq -|x_{i1}(0)|-\max\left[\frac{\beta_i}{\tau_{i1}}e^{-t/\tau_{i1}}\int_0^tv_{i1}(u)e^{u/\tau_{i1}}du\right]-\max\left[\frac{h_i}{\tau_{i1}}e^{-t/\tau_{i1}}\int_0^ty_{i2}(u)e^{u/\tau_{i1}}du\right] \\ &\quad -\max\left\{\frac{1}{\tau_{i1}}e^{-t/\tau_{i1}}\int_0^t\left[\sum_{j=1,j\neq i}^n(p_{ij}^1y_{j1}(u)+q_{ij}^1y_{j2}(u))\right]du\right\} \\ &\geq -(1+\beta_i)|x_{i1}(0)|-\beta_i(1+\beta_i)|v_{i1}(0)|-h|x_{i2}(0)|-h\beta|v_{i2}(0)| \\ &\quad -\sum_{j=1,j\neq i}^n[(p_{ij}^1|x_{j1}(0)|+q_{ij}^1\beta v_{j2}(0)+q_{ij}^1|x_{j1}(0)|+q_{ij}^1\beta|v_{j2}(0)|] \\ &\quad -[\beta_i+2h_i(n-1)]r_i+(h_i+1)Ls_{imin}-\beta_iLs_{imax} \end{aligned} \quad (5.150)$$

Note that the above process is similar with (5.100).

For $x_{i2}(t)$, it has a similar expression

$$\begin{aligned}
 x_{i2}(t) \geq & -(1 + \beta_i)|x_{i2}(0)| - \beta_i(1 + \beta_i)|v_{i2}(0)| - h_i|x_{i1}(0)| - h_i\beta|v_{i1}(0)| \\
 & - \sum_{j=1, j \neq i}^n [(p_{ij}^2|x_{j1}(0)| + q_{ij}^2\beta v_{j2}(0) + q_{ij}^2|x_{j1}(0)| + q_{ij}^2\beta|v_{j2}(0)|] \\
 & - [\beta_i + 2h_i(n - 1)]r_i - (\beta_i + 1)Ls_{imax} + h_iLs_{imin}
 \end{aligned} \tag{5.151}$$

This has completed the proof.

Remark 5.13: In Proposition 5.12, the conditions of (5.139) and (5.140) state that the influences of external oscillators to the oscillator concerned is relatively weak. This mechanism is realized through the selection of p_{ij}^1 , p_{ij}^2 , q_{ij}^1 and q_{ij}^2 , which are relatively smaller compared with β_i and h_i .

Proposition 5.13 *Consider the multiple neural oscillators given by equations (5.134)-(5.137). Suppose that they have*

- *all states bounded*
- *unstable equilibrium points*
- *unique solutions*

Then, they generate the oscillatory outputs.

Proof: Proposition 5.11 has verified the states of the multiple oscillators are bounded. In sections 5.2.3 and 5.2.4, it has obviously been indicated that the neural oscillator has unstable equilibrium points. For the unique solutions of the multiple oscillator, it can be verified by checking the Lipschitz condition [195]. Since the three conditions mentioned above are all satisfied, it can be concluded that oscillatory outputs must be generated. However, it is difficult to say they are periodic.

5.3 Summary

In this chapter, we have investigated the neural control mechanism from an engineering point of view.

With regard to the CPG, the propositions presented in this chapter reveal the mechanism of neural membrane oscillation and can serve as the guidelines for selection of the parameters of the CPG in practical applications.

With regard to the neural oscillator, the methods adopted here include stability theory, describing function, and linear piecewise analysis. Some important properties of the neural oscillator such as the frequency determination, boundness, stability, and network characteristics have been also investigated. The insightful results will strengthen the foundation of the neural oscillator and enhance the effective application in the FES control system.

Chapter 6

Design of the FES Control System

In this chapter, we will design a FES control system with the neural control mechanism studied in the previous chapters. The ultimate objective of this work is to adopt CPG to design a FES walking control system. As an interim, this FES control system is also applied into the leg swinging and cycling movement. The versatile ability for different control tasks of the proposed FES system will be verified.

6.1 FES Swinging Movement

This work aims to adopt CPG for the rhythmic movement control of lower limb via FES. The mechanism of CPG has been studied thoroughly in Chapter 4 and Chapter 5. In Chapter 6, the proposed FES control system based on CPG will be designed.

A simple rhythmic movement, shank swinging, is firstly considered. The initial FES control system will be proposed and tested, and it will be expanded for the later full FES control system design for cycling and walking. Swinging movement, commonly named as knee joint control, has received a great attention as it is easy to be tested

with real experiments. Another reason is that the dynamic model describing this kind of movement is relatively simple that contains only one segment with one muscle [19], [150], [151], or two muscles [3], [108]. The simple motor control system and its corresponding hierarchical diagram are shown in Figure 6.1. As we have discussed in the previous chapters, the CPG existing in the spinal cord plays an important role in the lower limb movement control. Therefore, the kernel of the proposed FES control system is CPG design.

In fact, except for the CPG there are many other neuronal interactions in the spinal cord. From the perspective of neuroscience, CPG is not directly connected with muscles, there exists a very complex neural circuit between them, which contains different motoneurons (α , β , and γ motoneurons), Renshaw cells, Ia, IaIn, and numerous other interneurons. It not only shapes the signal from the CPG but also processes the sensory feedback signals. Generally, it deals with the negative feedback from Renshaw cells, also named as recurrent inhibition; force feedback from Golgi tendon organs (GTO); and feedback from joint sensors. However, it is still unclear how the complex neural circuit functions and coordinates so many sensory feedback loops and descending control signals from the CPG. Even the effect of each feedback loop seems obvious, the coordination and interaction mechanism amongst them are not clear. In a hierarchical model of motor system, some researchers propose a term “regulator” to represent this networks of neuronal circuit [72], [121]. Similar terms are also proposed by the other researchers, for example, “pattern shaper” in [3], and “pattern formation network” in [165], [166].

Here, from the perspective of control engineering, we will develop a control system to control the shank swinging movement. In this system, two muscles will be stimulated to drive the shank to move. The desired trajectory of the knee joint is defined by a sinusoidal waveform that is almost deemed as the benchmark in the previous literatures. The control system consists of a feedforward controller representing the

Figure 6.1: Simple physiological diagram of the rhythmic motor control of lower limbs and its equivalent hierarchical diagram.

CPG in the spinal cord, and a feedback controller (regulator), dealing with the sensory feedback of the motor control. The neural oscillator replacing the CPG is developed in the previous chapter, which has two coupled neurons. One is used to stimulate the flexor and the other is to stimulate the extensor. The regulator is realized by an artificial neural network (ANN) that contains two sub-networks, and the two outputs from the CPG are adjusted by the two sub-networks, respectively. Though it may not ideally simulate the functions of the real biological neuronal circuit, it is believed that this design should be accurate enough for the human locomotion control from the macroscopic perspective.

In the previous chapters, we have developed the neural oscillator and its features are also studied in detail. It is shown that the output of the neural oscillator can be regulated by the tonic input r , and the output frequency of the neural oscillator is determined by the time constants τ_1 and τ_2 . We have stated that the output frequency of the oscillator varies inversely as τ_1 and τ_2 , and the gain is defined as K_f . The error sensory feedback e entrains the neural oscillator with the gain L . When

L is zero, the error information can be ignored and in this case, the neural oscillator corresponds to a pure feedforward controller. However, when L is very large, the neural oscillator is immediately entrained to the error signals. On the other hand, if the sensory feedback e is eliminated, the neural oscillator will generate the invariant rhythmic pattern regardless of the changes in L . Because the neural oscillator has feedback pathways, it should be more appropriate to consider it in a feedback rather than feedforward context. Cohen [22] proposed a term “heterarchical control” for this type of interaction.

6.1.1 Simulation Study

The musculoskeletal model relating the shank swinging is shown in Figure 6.2, and its detailed mathematical model is discussed in Chapter 3. The simulation study of the swinging movement is completed in Matlab/Simulink environment with Runge-Kutta solver and the integration time-step of 1 ms. Here, the flexor is hamstrings and

Figure 6.2: (a) Knee joint control in experiments. (b) Musculoskeletal model of the shank swinging movement.

the extensor is vastus group. They are stimulated by two electrical pulse channels, z_1 and z_2 , respectively. As discussed previously, the electrical pulse width is controlled to stimulate the muscles generally, which is represented by z_1 and z_2 here. In order to achieve the swinging movement at the frequency of 1 Hz, the neural oscillator's

parameters are tuned as follows: $\tau_1 = 0.121$, $\tau_2 = 0.2$, $\beta = 2.5$, $h = 2.5$, $c_1 = c_2 = 100.0$, the tonic input $r_1 = r_2 = 10.0$ and the sensory feedback gain $L = 1$. Figure 6.3 shows the structure of the regulator. Note that the regulator has two

Figure 6.3: Structure of regulator network. It contains two sub-networks, and each sub-network with the radial basis function (RBF) neural network.

sub-networks. An adaptive radial basis function (RBF) neural network is applied in the design of the sub-network, which has three layers, i.e. input layer, hidden layer and output layer. In this network, the inputs represent the signals from the CPG units, while the output signals are considered as the electrical pulse z_i for stimulation of the muscles so that the swinging movement can be realized. The output of the regulator is given by:

$$z_i = \sum_{k=1}^m W_{ik} \Phi_{ik} \quad (6.1)$$

The membership of each hidden neuron in the network is the Gaussian function

defined by:

$$\Phi_{ik} = \exp\left(-\frac{\|y_i - u_{ik}\|^2}{2\sigma_{ik}^2}\right) \quad (6.2)$$

where $i = 1, 2$, k is the number of the hidden neurons in each sub-network. The main steps of the RBF algorithm at each sampling time include: (1) computation of the center u_{ik} and width of the Gaussian functions σ with the input data, (2) computation of the output of the hidden layer Φ_{ik} , (3) updating the weights W_{ik} ; and (4) repeat the process above. The weights W_{ik} are associated with the sensory feedback and are updated based on a learning rule as follows:

$$\Delta W_{ik}(t) = \eta e(t) \sum_{j=1}^n \frac{1}{n} \Phi_{ik}(t - jT) \quad (6.3)$$

where η is the learning rate, z is the output of the regulator, Φ_{ik} is the output of the hidden neuron, n is the filter order, T is the sampling period, and e is the feedback information from the joint angles of the lower limb, which is in fact defined as the error between the actual angle θ and the desired angle θ_{des} , i.e. $e(t) = \theta(t) - \theta_{des}(t)$. Here, θ_{des} is defined as $\theta_{des} = 120 + \sin(2\pi t)$.

The impedance controller is described by:

$$z_c = k_1(\theta_{des} - \theta) + k_2(\dot{\theta}_{des} - \dot{\theta}) \quad (6.4)$$

where k_1 and k_2 are two parameters that need be tuned. θ_{des} and $\dot{\theta}_{des}$ are the desired joint angle and angle velocity, respectively. It can be noted that the structure of the impedance controller is similar with a proportional-derivative (PD) controller.

In the design of the regulator, we set the sampling period $T = 50ms$ and the frequency $f=1$ Hz. The number of the neurons in each sub-network is selected as $k = \frac{1}{Tf} = 20$; the learning rate η is chosen as 0.002; and the filter order n is 10. For the impedance controller, its parameters are tuned as $k_1 = 20$, $k_2 = 0.1$.

Under this configuration of the control system, we get the tracking response of the knee joint as shown in Figure 6.4. It can be seen that this control system yields good tracking performance, especially the transient response is improved significantly compared to the results presented in [3], [19], and [45].

Figure 6.4: (Top) Angular tracking response of the knee joint. (Middle and bottom) Actual pulse width of the two channels with z_1 for vastus muscle and z_2 for hamstring muscle.

6.1.2 Effect of Sensory Feedback

It is shown in the work [105] that combination of a feedforward control and a feedback control in the design of the control system exhibits many advantages. However, the CPG is often deemed a pure feedforward controller in the previous work, and very few work has studied the effect of the sensory feedback on the CPG in a control system. Here, we will incorporate the sensory feedback information into the control system, such that the regulation of the CPG becomes adaptive, and therefore, it becomes a more interesting heterarchical control system.

If the rhythm of the desired trajectory has a sudden change, the control system without the adaptive mechanism for the CPG would not provide satisfactory control performance, because in this case the output of the CPG is with a fixed frequency. Since the adaptive mechanism incorporated in the control system proposed in this work, we can obtain the output of the CPG with varying frequencies and the satisfactory control performance can be ensured. In fact, it is realized by adjusting τ_1 and τ_2 , and by adding a frequency gain K_f to obtain the desirable sensitivity. The effectiveness of this adaptive mechanism can be evaluated from the results shown in Figure 6.5.

In Figure 6.5(a), note that at the 5th second, the rhythm of the desired trajectory is varied from 2Hz to 1Hz. At the same time the frequency gain K_f is changed from 8.0 to 1.0, correspondingly. In this case, we can see that the tracking performance is not affected by the adaptive mechanism incorporated in the control system. Figure 6.5(a) shows that when the desired rhythm is varied from 1Hz to 0.5Hz, K_f will be automatically changed from 1.0 to 0.5. From the simulation, it can also be found that it needs less tracking time at the movement with lower frequency.

The above simulation study is performed on an ideal FES control system without any noise and disturbance, so the control performance is very good. However, ex-

(a)

(b)

Figure 6.5: (a) Knee joint tracking response with the rhythms of 2Hz and 1Hz. (b) Knee joint tracking response with the rhythms of 1Hz and 0.5Hz. K_f is the frequency gain of CPG .

ternal disturbance, measurement noise and biological noise always exist in an actual motor control system. The effect of sensory feedback will play an important role to resist the noise. It is well known that a feedforward controller cannot compensate for unknown external disturbances, while a feedback controller is sensitive to the measurement noise. In Chapter 4, we accomplished the simulation study on robustness of the CPG, and it is shown that a large tonic input to the CPG can effectively resist against a strong noise. Using this result, we can set an adaptively adjusted gain K_t , so that the tonic input of the CPG can be changed along with the variation of the measurement noise. Figure 6.6 shows the simulation results. In this simulation, a white noise of power 0.5 indicating the measurement noise is applied to the sensory feedback. The tracking performance is affected severely without the adaptive mechanism (see Figure 6.6(a)), because the CPG can not produce an ideal rhythmic pattern. However, if the tonic adaptive gain K_t is applied based on the level of the noise, the performance can be improved significantly as shown in Figure 6.6(b). Figure 6.6(c) shows the tracking errors with two different tonic gains. Clearly, the tracking error with a large tonic gain is much smaller than that with a small tonic gain. Therefore, the adaptive tonic input gain introduced can effectively improve the robustness to resist the measurement noise.

6.2 FES Cycling Movement

The ultimate objective of the research in the human movement control is to design a neural control system for human walking with FES. Cycling movement is an important research topic and can provide the foundation for locomotion. It is known that the coordination of the muscles in cycling is similar with that in walking. That means the stimulation patterns generated from the spinal cord for stimulating the muscles are similar in the cycling and walking movements. However, since the subject sits on the saddle of bicycle and the upper body has no effect on the

(a)

(b)

(c)

Figure 6.6: (a) The tracking response without tonic gain (b) The tracking response with tonic gain. (c) The comparison of errors with the different tonic gains: $K_t=10$ and $K_t=1.0$.

lower limb during a cycling movement, the dynamics of the cycling movement is less complex than that of locomotion. Moreover, in this case there is no need to consider the body balance problem. In a word, the proposed FES control system for cycling extends the one initiated in the swinging movement, and it is also a good interim for the realization of locomotion.

The neural control system for the FES cycling movement is developed, and its detailed block diagram is shown in Figure 6.7, which is an extension of the model shown in Figure 6.1. This control system is also suitable for the FES walking control. There are several components in the proposed system that need to be designed, i.e. the CPG, regulator, musculoskeletal model, and sensory feedback. Note that the posture control is not considered during cycling movement. With regard to the sensory feedback, noise sensor has been studied for the swinging movement in the previous section, so this section is focused on the foot touch sensor and the overall speed sensor.

6.2.1 Musculoskeletal Model of Cycling

The musculoskeletal model of cycling movement is shown in Figure 6.8. In this model, each leg has three segments and nine muscles. In fact, it is a hard job to regulate the stimulation patterns for the nine muscles. Moreover, the harmony between the two legs is another formidable task to handle.

The muscle model used in this study has been presented in Chapter 3, and the dynamics of the three segments is given in Appendix. Here, only the action between the foot and pedal will be highlighted. The force applied on the pedal by foot can be divided in two directions as shown in Figure 6.8. One is tangential force F_t and the other is radial force F_r . The external torque applied to the foot by the pedal ($E(\theta)_3$ in Appendix) has the same value of crank torque T_c , as they are a pair of

Figure 6.7: Proposed neural control system for the human lower limb movements with FES. Motor command from supraspinal system is nonspecific, which performs simple actions such as “start” and “stop”. Central pattern generator (CPG) generates the rough rhythmic walking patterns which will be finely regulated by regulator network. Postural control is in charge of body balance control during walking, and it is triggered by foot touch sensor. Impedance control is to provide rough regulation and resist disturbance. The final control signal z_m is the result of combined actions of these controllers. Several types of sensors are fed back to regulate the controllers. Musculoskeletal model is the plant to be controlled.

acting and reacting torques. The data of these three variables are adopted from [96], and they are plotted in Figure 6.9.

Figure 6.8: Musculoskeletal model of the cycling movement. Each leg contains nine muscles and three skeletons. The names of muscles are abbreviated in the contents. Gluteus maximus: GMAX, iliopsoas: ILI, vastus group: VAS, biceps femoris short-head: BFsh, tibialis anterior: TA, soleus: SOL, rectus femoris: RF, hamstrings: HAM, gastrocnemius: GAS.

6.2.2 Design of Neural Control System

CPG

The FES control system for cycling in 6.7 is designed according to the hierarchical diagram of Figure 6.1, and it is an extension of the neural control system for the swinging movement.

In the design of the control system, the key part is also the CPG design. The CPG

Figure 6.9: Forces (tangential F_t , radial F_r) and torque acted on foot by pedal during cycling. Experimental data from [96].

mainly generates stimulation signals of rhythmic motor patterns with required frequencies, and phasing (timing). As the CPG needs generate nine rhythmic patterns for the nine muscles related to the hip, knee and ankle of each leg, we have designed 3 pairs of oscillators for each leg. Therefore, the CPG actually consists of 6 pairs of oscillators for both legs (see Figure 6.11). Each oscillator contains two coupled neurons with self and mutual inhibition, and unilaterally it can be described by the following ordinary differential equations.

$$\frac{1}{K_f} \tau_{i1} \dot{x}_{i1} = -x_{i1} - \beta_i v_{i1} - h_i y_{i2} + K_t r_i - \sum_{j=1, j \neq i}^6 (p_{ij}^1 y_{j1} + q_{ij}^1 y_{j2}) + H(F_{foot}) + Le \quad (6.5)$$

$$\tau_{i2} \dot{v}_{i1} = -v_{i1} + y_{i1} \quad (6.6)$$

$$\frac{1}{K_f} \tau_{i1} \dot{x}_{i2} = -x_{i2} - \beta_i v_{i2} - h_i y_{i1} + K_t r_i - \sum_{j=1, j \neq i}^6 (p_{ij}^2 y_{j1} + q_{ij}^2 y_{j2}) + H(F_{foot}) - Le \quad (6.7)$$

$$\tau_{i2} \dot{v}_{i2} = -v_{i2} + y_{i2} \quad (6.8)$$

$$y_{i1} = f(x_{i1}) = \max(x_{i1}, 0)$$

$$y_{i2} = f(x_{i2}) = \max(x_{i2}, 0) \quad (6.9)$$

$$i = 1, 2, 3$$

where i is the index for hip oscillator, knee oscillator, and ankle oscillator, each of which consists of two neurons. x_{i1} and x_{i2} represent the two states of the i th oscillator. τ_{i1} and τ_{i2} are the time constants. p_{ij}^1 , q_{ij}^1 , p_{ij}^2 and q_{ij}^2 are the weights for the connections amongst the neural oscillators, and j indicates the index of the weights. r_i is the tonic input. K_f is the adaptive speed feedback gain. K_t is the adaptive tonic input gain. $H(F_{foot})$ is the foot touch sensory feedback. e represents the proprioceptor in joint sensing the joint position and velocity, the gain is L . In order to describe the clear relationship between the CPG neurons and muscles, we

introduce y_i to represent the outputs of CPG. That is $y_1 = y_{11}$, $y_2 = y_{12}$, $y_3 = y_{21}$, $y_4 = y_{22}$, $y_5 = y_{31}$, $y_6 = y_{32}$.

The block diagram of the CPG system is shown in Figure 6.11. We can see that it is a fully connected system. That means not only the two coupled neurons interact with each other, all the three neural oscillators in one leg are also interacted by the synapse mechanism. Moreover, bilateral coupling mechanism between the two legs is also considered in this system. The most promising feature is that four types of sensory feedback are incorporated in the CPG system, which will provide adaptive ability for CPG. The corresponding relationship between the muscles and neurons are illustrated clearly in Figure 6.11. For example, the hip extensor and flexor are stimulated by the signals from the two neurons in the hip oscillator. Similarly, the muscles of knee joint are stimulated by the knee oscillator, and the muscles of ankle joint are stimulated by the ankle oscillator.

However, the relationship discussed above is only suitable for uniarticular muscles. Some revisions should be made for biarticular muscles, because they cross two joints, and the information from two joints will have influence on them. Note that there are three biarticular muscles: muscle 7 (RF), muscle 8 (HAM) and muscle 9 (GAS). RF crosses hip and knee joints, which serves as the flexor of hip joint partly and the extensor of knee joint partly during a full cycle. Therefore, the hip flexor neuron (neuron 2) and knee extensor neuron (neuron 3) are combined together to provide the patterns for RF. The relationship is defined as $y_7 = c_2 y_2 + c_3 y_3$, where c_2 and c_3 are the coefficients. The mechanism is similar for HAM and GAS. The difference is that GAS mainly gets signal from the dominant neuron 6, so its activation pattern is similar with muscle 6 (SOL). After the parameters are properly selected, CPG can generate the rough muscle activation patterns as shown in Figure 6.10. The work also appears in my publication [208].

Here, we propose three sets of the CPGs (neural oscillator) for each leg, i.e. a hip

Figure 6.10: Outputs (y_1 - y_9) of CPG with 1 Hz rhythmic motor pattern. These rough motor pattern will be regulated finely by the following component, regulator.

oscillator, a knee oscillator and an ankle oscillator. Each oscillator contains two coupled neurons with a self-inhibition. The outputs of the neuron will be sent to the different muscles in the end. However, the regulator will refine them from CPG meanwhile, so that the performance during the process can be enhanced, and then the desired stimulation pattern for the muscles can be achieved.

Figure 6.11: CPG structure for the cycling (walking) movement control. Two sets of full connected CPGs are constructed for two legs. Four types of sensory feedback are sent back to CPG. Dashed line means the weak connection. Note that CPG is not connected with the muscles directly, and there exists a regulator between them. The arrows between them only indicate the corresponding relationships.

6.2.3 Regulator

As our work aims to accomplish an engineering task, macroscopic level structure is applicable in future. Here, we will develop an artificial neural network to mimic the function of regulator from control engineering perspective. Research on human movement has shown that, there is a separate network in the nervous system to control each limb, and the network of this limb can be subdivided into sub-network

units involved in the control of single joints and groups of muscles [58]. Coincidentally, a structured functional neural network with seven sub-networks method has been used in the robot control, and it achieves good performance [132]. So this type of network structure may have promising potential to design the regulator network for the proposed FES control system here.

Radial basis function (RBF) neural network is adopted in the regulator design, because it is suitable to solve the problems with a small number of inputs and it costs less training time. It contains nine sub-networks, and the nine outputs from the CPG are adjusted by each sub-network respectively. Though it cannot replace the function of the real complex neuron circuit completely, it's enough for the FES cycling (locomotion) control from a macro perspective. As the CPG can only provide rough rhythmic motor patterns which contains basic information such as frequency, phasing, and rough amplitude, the fine tuning for accurate reshaping is realized by the regulator, i.e RBF neural network. The structure of regulator network is shown in Figure 6.12, and there are nine sub-networks to regulate nine muscle stimulation patterns for cycling (walking). This structure of the neural network control has also been tested for the FES standing control [207].

The input of the RBF is y_i from the CPG, and the output of the regulator is given by:

$$z_i = \sum_{k=1}^m W_{ik} \Phi_{ik} \quad (6.10)$$

The membership of each hidden neuron in the network is the Gaussian function defined by:

$$\Phi_{ik} = \exp\left(-\frac{\|y_i - u_{ik}\|^2}{2\sigma_{ik}^2}\right) \quad (6.11)$$

where $i = 1, 2, \dots, 9$, k is the number of the hidden neurons in each sub-network. The general steps of the RBF algorithm at each sampling time include: (1) computation of the center u_{ik} and width of the Gaussian functions σ with the input data, (2)

Figure 6.12: Structure of regulator network. It contains nine sub-networks. Inputs are y_i from CPG network, and outputs are sent to stimulate muscles. RBF is adopted for each sub-network.

computation of the output of the hidden layer Φ_{ik} , (3) updating the weights W_{ik} ; and (4) repeat the process above.

However, if we choose the centers arbitrarily, it will not satisfy the requirements that centers should suitably sample the input domain and require an unnecessary large RBF network. As the performance of the RBF neural network critically depends upon the chosen centers. The center is selected using a similar method presented in [174], which will map the input to a series of Gaussian function curves.

In another aspect, the structured neural network containing 9 sub-networks is too complex. In order to reduce the size of the network, a sequential learning algorithm for RBF neural network named as minimal resource allocation network (M-RAN) is used [16], [122]. M-RAN performs the growth criterion and the pruning strategy as well as the adjustment of the network parameters with the network input data. It can achieve good performance with relatively smaller network size. The M-RAN learning algorithm can be simplified as the following steps:

(1) Estimation criteria for current error, the error must be greater than the threshold E_1 :

$$e(k) = \theta(k) - \theta_{des}(k) \geq E_1 \quad (6.12)$$

(2) Novelty criteria, the nearest center distance must be greater than the threshold E_2 :

$$\inf_{j=1}^m \|y_i(k) - u_{ij}(k)\| \geq E_2 \quad (6.13)$$

(3) Windowed mean error criteria, the windowed mean error must be greater than the threshold E_3 :

$$\frac{1}{T} \sum_{i=0}^T [\theta(k-i+T) - \theta_{des}(k-i+T)] \geq E_3 \quad (6.14)$$

At each sampling step, when the three criteria above are met, one neuron unit is

added. The new neuron is initialized with the following center, variance and weight respectively:

$$u_{m+1}(k) = y_i(k) \quad (6.15)$$

$$\sigma_{m+1} = \lambda \ln f_{j=1}^m \|y_i(k) - u_{ij}(k)\| \quad (6.16)$$

$$W_{m+1}(k) = e(k) = \theta(k) - \theta_{des}(k) \quad (6.17)$$

where λ is a constant called “overlapping factor”. When any of the above criteria is not satisfied, the tuning parameters in the basis function of the network will be updated as:

$$W(k+1) = W(k) - \eta e(k) \frac{dz(k)}{dW(k)} \quad (6.18)$$

where e is defined as the error between the actual and desired joint angles, i.e. $e = \theta - \theta_{des}$.

To avoid an excessive increase of the network size, a pruning strategy is applied. The pruning strategy is used to remove those hidden neurons that are minimal contributors to the RBF network outputs. Consider the output of the i th hidden neuron, $\phi_{ik} = W_{ik} \exp(-\frac{\|y_i - u_{ik}\|^2}{2\sigma_{ik}^2})$, $i = 1, \dots, 9, k = 1, \dots, m$. If ϕ_{ik} is small, it is due to one or more of the following reasons: W_{ik} or σ_{ik} is small, or $\|y_i - u_{ik}\|$ is large. The output is first normalized to the value of the highest output:

$$r_{ik} = \frac{\phi_{ik}}{\max(\phi_{i1}, \phi_{i2}, \dots, \phi_{im})} \quad (6.19)$$

If r_{ik} falls below a threshold for several consecutive outputs, the neuron is pruned. The dimensionality of the appropriate network size will be adjusted. It was reported that this method yielded good performance for flight control and system identification in the previous literatures.

6.2.4 Impedance Controller

Impedance control was proposed by Hogan [81] as a fundamental strategy for controlling multi-joint robot system in a constrained environment. It regulates both the force and the trajectory to endow the end-effector with dynamic properties that may be inherently different from its mechanical properties. This method is widely used for robot [158], [189] and human movement control [179], [182], [187]. In general, impedance of lower limb is the result of passive dynamics of the lower limb, intrinsic impedance of activated muscles, and reflexive contributions. The basic impedance control can be represented by [189]

$$F_{im} = K_1(x_0 - x_d) + K_2(\dot{x}_0 - \dot{x}_d) - K_3\ddot{x}_d \quad (6.20)$$

Where x , $\dot{x}$ and $\ddot{x}$ are the position, velocity and acceleration vector of the segment in the task space, and the indices $*0$ and $*d$ refer to the actual and desired values, respectively. F_{im} is the force produced by impedance control. The matrices K_1, K_2, K_3 indicates the stiffness, damping, and inertia coefficients, respectively.

However, for FES control, the force generated by specific muscles with regard to a joint is an intermediate variable that is controlled by electrical pulse applied. That means the electrical pulse indeed is a control variable. Therefore, some minor revisions are made. Here, a gain for the electrical pulse width z_{ci} is used to replace F in equation (6.20). The inertia coefficient is not considered as [182]. This format is very similar with the proportional-derivative (PD) controller. The function of the impedance controller is to give a rough tuning of the control performance and produce enough torque to support the human body during walking.

Finally, in my work, an impedance controller is designed based on (6.20),

$$z_{ci} = k_{1i}(\theta_{desi} - \theta_i) + k_{2i}(\dot{\theta}_{desi} - \dot{\theta}_i) \quad (6.21)$$

where k_{1i} and k_{2i} are the parameters that needs to be tuned. θ_{des} , $\dot{\theta}_{des}$ are the desired joint angles and angle velocities, respectively. $i = 1, 2, 3$ is the index representing the corresponding joints, i.e. ankle, knee and hip.

As the control variable is electrical pulse not force (torque), some further regulation should be added. A switch for z_{ci} is designed. If $z_{ci} > 0$, then z_{ci} will be summed to the stimulation for extensor (plantar flexor); otherwise, it will be for flexors (dorsiflexor). The extensor and flexor are classified with regard to the three joints of lower limb. Hip extensor is muscle 1 (GMAX), and hip flexor is muscle 2 (ILI); knee extensor is muscle 3 (VAS), and knee flexor is muscle 4 (BF); ankle dorsiflex is muscle 5 (TA), and ankle plantar flexor is muscle 6 (SOL). Biarticular muscles are not considered here.

Contralateral Effect

It is found that during the rhythmic movement of the two legs, the movement of one leg will be affected by another leg due to the bilateral coupling mechanism [178], [206]. The coupling mechanism is generally deemed as the connection between the bilateral CPG units, or the mechanical coupling from the sensory feedback. However, the complete mechanism of the leg rhythmic movement is not fully understood.

From appearance, the phenomenon can be observed as the movement of the two legs are phase-locked in an anti-phase relationship. It indicates that the activation pattern of the ipsilateral limb may have some phase relationship with that of the contralateral limb. There are some assumptions about the causation of this phenomenon. (1) contralateral sensorimotor signals mediated through neural inter limb coupling mechanisms and (2) ipsilateral afferent signals triggered by the forces transmitted to the ipsilateral leg due to acceleration, or movement of the contralateral leg. That means the mechanical sensory feedback and neural coupling both affect the contralateral activation of the muscles, and it's hard to isolate the two factors from experiments [185].

In the previous research, different methods were used to model this effect. Some used the mechanical sensory feedback with global sensing to realize this effect [142], [194], while the others used the weak connections with global sensing amongst the CPG units [182]. However, it still cannot be determined whether the bilateral coordination through mechanical coupling is equivalent, inferior, or superior to having explicit neural connections in bilateral CPG. But, one thing is sure that the mechanical sensory feedback with global sensing is easy to be performed in simulation study, as the parameters that need tuning for the CPG are much fewer.

Here, the mechanical sensory feedback from the tactile receptor of the foot is used to coordinate the patterns of muscles for the two limbs. It can be formulated as the function $H(F_{foot})$, where $H(x)$ is the unit function defined as $H(x) = 1$ for $x > x_0$ and $H(x) = 0$ for $x \leq x_0$, and x_0 is a threshold value. Generally, F_{foot} is the reaction force (torque) sensed by the foot, which means the force (torque) reacted by the pedal. However, for cycling, the force information is not ideal to distinguish the phase difference of the contralateral legs (see upper two plots in Figure 6.9). Crank torque (T_c) could be used as an effective and accurate feedback information to indicate foot touch here. For more tolerance, x_0 is selected as 5 Nm. Note that H_{foot} indicates the vertical ground reaction force (GRF) for walking.

From this formulation, the mechanical sensory feedback from foot is normalized to 1 and it is quantified to a series of pulses. When it is fed back to the CPG, it can entrain the CPG and change the phase of the CPG's output. This mechanism is illustrated clearly in Chapter 4. Here, we use it to make the two legs perform the cycling movement with the anti-phase relationship.

6.2.5 Simulation Study

The main part of the simulation study is to tune the parameters of the CPG. The CPG should provide appropriate patterns for the nine muscles. The experimental EMG patterns of the muscle during cycling can be found in [155], [201] and we expect that the output of the CPG should match them satisfactorily.

The parameters of the CPG are tuned according to the analysis introduced in Chapter 5. There are also some criteria published in [44], [90], [182], [194]. For example, the time constants τ_1 and τ_2 should be tuned to produce the desired frequency. They can be adjusted to the values of a resonance frequency of a simple pendulum corresponding to the lower limb. In order to produce the locomotion of 1Hz for the biped, the parameters are set as $\tau_2=0.2$, $\beta=2.5$, $h=2.5$, $r_1=r_2=10.0$, and $L=1.5$. These parameters are identical for all CPG units. However, τ_1 has a little difference for all CPG units, for example, for the ankle oscillator $\tau_1=0.12$, for the knee oscillator $\tau_1=0.11$ and for the hip oscillator $\tau_1=0.121$. Also, the values of connections among all CPG units are different. Actually, they are $m_{13} = m_{31} = m_{24} = m_{42} = m_{36} = m_{63} = m_{45} = m_{54} = 0.2$; $m_{14} = m_{41} = m_{23} = m_{32} = m_{35} = m_{53} = m_{46} = m_{64} = -0.2$; $m_{15} = m_{51} = m_{26} = m_{62} = -0.1$; $m_{16} = m_{61} = m_{25} = m_{52} = 0.1$. As the two legs are symmetric, only the parameters for one leg are listed.

The regulator contains nine sub-networks and its structure is described in the swinging movement control in the previous section. Each sub-network serves for one muscle correspondingly. The proposed structure of the regulator can be linked to the structure developed from neuroscience, in which there exists a separate network in the nervous system to control each limb, and the network of this limb can be subdivided into sub-network units involved in the control of single joints and groups of muscles [58].

The errors between the actual angles and desired angles are sent back to adjust the

network. The desired trajectories are collected from a real experiment as shown in Figure 6.13, which is conducted in Sports and Recreation Center at Nanyang Technological University. In this experiment, a healthy subject sits on the saddle performing a cycling movement, then the ankle, knee and hip joint angles are detected using the goniometers from Biometrics Ltd., UK. As the subject cannot maintain the same constant speed of cycling when repeating the experiment, the trajectories collected from the experiment are different at each time. After a careful comparison based on the maximum, minimum, mean values and the mean frequencies, we select one set of data regarding to the three joint angles. However, there is much noise affecting the hip trajectory as the subject wears the short pants during cycling. In the later stage, a Bessel filter is adopted to process the data of hip joint angle. Then the final three desired trajectories are achieved and shown in Figure 6.14.

In the previous work, the control objective is nearly all about the speed control of the cycling [20], [86]. The angular tracking performance during cycling is seldom considered. This is because the hip position is fixed on the saddle, and the foot position is fixed on the pedal in their work. We can imagine that the angles are nearly pre-defined by the constrained routine. Their control task is relatively simple. Here, we consider the control performance for not only the cycling speed but also the joint angles. The overall speed sensor will sense the desired crank speed and regulate the time constants of CPG with K_f in order to produce the desired rhythmic patterns.

In fact, during a real experiment, even the same person performs the same cycling movement for several times, the curves of each time are somewhat different. So the motivation about the impedance controller is not to achieve the perfect tracking performance, it aims to provide a “direction”; another important function is to enhance the stability of the system. Accordingly, the burden of the regulator will

Figure 6.13: Measurement of the desired joint angles of lower limb during cycling movement via goniometers. Eight sets of data are collected for ankle, knee and hip joint angles.

Figure 6.14: Desired trajectories of three joints (ankle, knee and hip) during cycling. The data are collected on a healthy subject as shown in Figure 6.13.

be relieved much.

It can be said that the purpose of the impedance controller is for a rough tuning of the tracking, which in fact provides a direction of the actual trajectories in order to prevent a large excursion, while the regulator is for a fine tuning of the tracking through the feedback control. The parameters of the impedance controller are given in Table 6.1.

Table 6.1: The parameters of the impedance controller. ($i=1,2,3$)

$k_{1i}[1/deg/s]$	2.0	2.0	1.0
$k_{2i}[1/deg]$	0.15	0.15	0.20

The tracking performance for the cycling speed is discussed as follows. Note that the crank speed represents the overall speed of the cycling movement. In order to achieve the desired speed as shown in Figure 6.16(b), the sensor K_f is changed adaptively. In consequence, different rhythms of the leg movement can be achieved.

The trajectories of the three joint angles and the corresponding values of K_f are shown in Figure 6.16(a).

Figure 6.15: Simulation results. Three joint angles of lower limbs during cycling and the foot touch sensory feedback to the CPG. (a) is for left leg, (b) is for right leg.

The tracking performance for the cycling speed is discussed here. Note that the

crank speed represents the overall speed of the cycling movement. In order to achieve the desired speed as shown in Figure 6.16(b), the sensor K_f is changed adaptively. In consequence, different rhythms of the leg movement can be achieved. The trajectories of the three joint angles and the corresponding values of K_f are shown in Figure 6.16(a). Please note that the regulator is not considered at this stage, therefore, the curves of the three joint angles are different with the desired angles shown in Figure 6.14.

The muscle torques acting at each joint are demonstrated in Figure 6.17. It can be noted that the ankle and hip torques are significant because the hip extensor (GMAX) and ankle planar flexors (SOL and GAS) contribute much. Another interesting feature we can observe from the result is that a high crank speed requires a large muscle torque. In fact, the large muscle force can be achieved in two ways: (1) increasing the tonic intensity and (2) auto-tuning from the impedance controller and regulator with errors. Some previous research has proved that the intensity of the tonic input affects the rhythmic speed [57], [182]. However, changing the tonic intensity only is not enough because it cannot change the rhythmic patterns of the muscles. Biological experiments show that increasing the intensity of the electrical stimulation applied in the supraspinal locomotor region (mesencephalic) may produce a high speed movement of the limbs. However, their electrical stimulation should not be deemed as the tonic input here. In fact, their stimulation intensity has affected some parameters of the ionic channels in the CPG. Thereafter, the rhythmic pattern of the output is changed.

Finally, the conclusion is that the tonic intensity combined with the frequency gain K_f together contributes to the speed tracking performance.

The regulator's function is to perform a fine regulation, and improve the angular tracking performance. After the regulator begins to work, the tracking performance of the joint angles is shown in Figure 6.18. We can see that the three joint angles can

(a)

(b)

Figure 6.16: Speed control. (a) Speed variation of angles along with changes of the frequency gain K_f . (b) The desired crank speed.

Figure 6.17: Simulation result. Muscle torques acting at the three joints (ankle, knee and hip) during cycling movement under the proposed FES control system shown in Figure 6.7. The result is the combined action of the components: CPG, impedance controller and sensor feedback. Regulator is not considered.

track the desired trajectories in the time less than 0.4 sec, and the performance is satisfactory. Along with Figure 6.18, the forces produced by the muscles are shown in Figure 6.19. It is the result under the co-work of CPG, impedance controller and regulator in the full FES control system.

Figure 6.18: Angular tracking performance of the three joints (ankle, knee and hip) for one leg during cycling.

Figure 6.19: Simulation results of the forces generated by the nine muscles during cycling movement. The horizontal thick bar in the first cycle indicates the desired EMG pattern [155], [201].

6.3 FES Walking Movement

A full gait cycle of the human walking is illustrated in Figure 6.20. It is commonly divided into two phases, swing and stance. Roughly, the standing phase occupies 2/3 of a cycle of walking, and swing phase occupies 1/3.

Figure 6.20: The full gait cycle of the human walking. It has two phases: swing and stance. TO indicates “toe off”, and HC indicates “heel contact”.

The structure of the biological motor control system for human walking is shown in Figure 6.21. With regard to a complete hierarchical human motor control system (see Figures 2.1 and 2.2), it belongs to the lowest level. Since the brain has not much effect on the rhythmic movements, the neural control mechanism mainly derives from the spinal cord circuit. Some key components of the motor control system can be generalized from Figure 6.21, e.g. spinal cord circuit, afferent and efferent signals, musculoskeletal system, sensory feedback, and reactions from the external environment. Here, a FES control system is developed based on the biological motor control system shown in Figure 6.21. Its block diagram is shown in Figure 6.7. Next, we will describe each component of the FES control system, respectively.

The motor control system for the walking movement is similar to that of the cycling

Figure 6.21: A simplified physiological model for human walking.

movement as shown in Figure 6.7. It is also a hierarchical structure containing the CPG, regulator and sensory feedback. However, some modification is made on the CPG design, and new algorithms are proposed for the regulator network in order to improve the control performance and reduce the tracking time. Different from the cycling movement, the body balance control in the walking movement is a key problem. For this reason, a fuzzy logic controller is designed.

6.3.1 Musculoskeletal Model of Walking

The musculoskeletal model of human walking is shown in Figure 6.22. It contains 7 segments and 18 muscles altogether. The two legs are symmetric, and without loss of generality, only nine muscles of one leg are illustrated. The body segments include HAT (head, arms and trunk), thighs, shanks and feet. The muscle model is developed from the popular Hill-type muscle model, which is used widely in the engineering practice. The details of the muscle model have been elaborated in

Chapter 3. The skeletal dynamics of the human body can roughly be computed from a robotic method (See Appendix).

Figure 6.22: Musculoskeletal model of human walking. The names of muscles are abbreviated in the contents. Gluteus maximus: GMAX, iliopsoas: ILI, vastus group: VAS, biceps femoris: BF, tibialis anterior: TA, soleus: SOL, rectus femoris: RF, hamstrings: HAM, gastrocnemius, GAS.

Each leg experiences stance and swing phases during a gait cycle. For the ipsilateral side, the human body can be looked as an inverted pendulum during stance, and the dynamics of four segments is needed. However, only three segments are considered during the swinging state. Particularly, when the foot touches the ground during the stance state, the ground will produce a counterforce to it, and therefore the mechanical model between the foot and ground should be modeled. Here, it is modeled by visco-elastic elements, i.e. dampers and springs. The mechanical structure is shown in Figure 6.23. Note that for horizontal, the viscosity and elasticity coefficients are set as $200Ns/m$ and $5000N/m$, respectively; for vertical, the coefficients are set as

500Ns/m and 25000N/m, respectively. In addition, some anthropometric data of the foot is labelled, which serves for the calculation in the posture control. In fact, this is a revised model based on [136], where the ground reaction force (GRF) is calculated due to the deformation of the ground or the shoes. However, it is too complex to be practical here.

Figure 6.23: Biomechanical model of foot with action to the ground

The foot angle (θ_{foot}) with regard to the horizontal solid ground is obtained from [193]. We can calculate the foot horizontal displacement and vertical displacement during stance state, which is multiplied by the visco-elastic element coefficient, and then the GRF is achieved. The value of the vertical displacement is much larger than the real vertical deformation of the floor, but their shapes are identical. Therefore, a proportional gain is added to match the real data [137]. The GRF is shown in Figure 6.24. The foot touch sensory feedback can be calculated from the GRF with the function $H(GRF)$, which is used to entrain the CPG in order to make the movement of the two legs have an out-of-phase relationship as shown in Figure 6.28(b).

6.3.2 Control System Design

The proposed control system for FES walking is similar with that for FES cycling as shown in Figure 6.7. The same components are CPG, regulator, impedance

Figure 6.24: Ground reaction force (GRF) to the foot. Left: The horizontal force acted to the foot by ground. Right: The vertical force acted to the foot by ground.

controller and three types of sensory feedback (foot touch, speed sensor and noise sensor). For these components, the basic mechanism is the same, only slight regulation is made for FES walking. The difference is that two new components are added, posture controller and COG (center of gravity) sensor that serves for posture controller. Another feature, i.e. the phase-switch, is added to trigger the posture controller. In a word, this section will emphasize on posture controller design.

Research has shown that the phenomena of posture control and rhythmic movement control are not separate. Actually, they interact with each other at different levels of the central nervous system, from the motor cortex to the spinal cord circuit. That means the posture balance control is not only regulated by the spinal cord, but also by the supraspinal system. Therefore, we should not only generate the desired rhythmic movement, but also control the body balance at the same time. This is possible because the recent research through experiments has shown these two motor functions share some common principles of spatial organization [62].

During the stance phase, the human body is like an inverted pendulum. In order to keep the body from collapsing, the stability at this stage is especially important. For the multi-segment inverted pendulum control, the objective is to control the central of gravity (COG) or central of mass (COM) within the stable region. The

idea is the same with that in the human body balance control. In order to formulate this mechanism, global variables are needed. We look on the inertial angles from each segment as the global variables, which are defined with respect to the normal to the ground as shown in Figure 6.25. The outputs of the skeletal dynamics in equation (2.1) are the joint angles, and there is no feedback information about the inertial angles, so we should transform the joint angles into the inertial angles for the segments.

Figure 6.25: Definition of the angles of lower limb.

In the real human motor control system, the vestibular and visual system can sense the orientation of gravity, but this extreme complex mechanism is not fully understood at present. A simple way is to use the inertial angle to indicate the COG of the segments from some aspects. In real FES application, accelerometers or tilt sensors are often used to measure the inertial angles. Here, they can be easily obtained from the computation of the joint angles. Then only goniometers are needed in the future design. Their relationship is achieved with geometric calculation according

to Figure 6.25.

$$\theta_{\Delta} - \theta_a + \theta_{foot} = \theta_{shank} \quad (6.22)$$

$$\theta_k + \theta_{shank} = \theta_{thigh} \quad (6.23)$$

$$\theta_h + \theta_{thigh} = \theta_{HAT} \quad (6.24)$$

A fuzzy logic controller is designed to realize the posture control. The inputs of the fuzzy controller are the global variables, i.e. inertial angles and angular velocities of the three segments. There are three sets altogether: θ_{shank} and $\dot{\theta}_{shank}$; θ_{thigh} and $\dot{\theta}_{thigh}$; θ_{HAT} and $\dot{\theta}_{HAT}$. The outputs are the gains for the electrical stimulation to the muscles G_z , which are multiplied to the output of the regulator. A temporary variable, torque change (ΔT), is introduced in order to make the fuzzy logic controller be easily understood. When (ΔT) is negative, the gain of the electrical stimulation ($1 + \Delta G_z$) is applied to the extensor muscles.; when it is positive. Their relationship is illustrated in Figure 6.26 (a). Generally, the fuzzy logical control contains four principal procedures: fuzzification interface, logical reasoning, defuzzification interface and rule table. The details are discussed subsequently.

There are three segments, shank, thigh and HAT, that should be controlled. The universe membership function for the inertial angle of each segment is shown in Figure 6.26 (b), and the membership functions for the angular velocity are shown in Figure 6.26 (c). Triangular membership function is selected as it has several advantages: (1) the calculation is simple; (2) the overlap between neighboring fuzzy sets is appropriate; (3) it yields less steady-state error than the other membership functions. The values of the membership function for each segment are shown in Table 6.2.

For each segment, the inertial angle (θ) and angular velocity ($\dot{\theta}$) are defined as the inputs. Not that there are 5 fuzzy sets for each inertial angle and 3 fuzzy sets for

Figure 6.26: (a) Membership functions for the stimulation gain change ΔG_z . (b) Membership functions for the inertial angles. (c) Membership functions for the inertial angular velocities of the segments.

Table 6.2: Values of the membership functions for the fuzzy rules.

	C_{NL}	C_{NS}	C_{ZE}	C_{PS}	C_{PL}
θ_{shank} [deg]	-40	-20	0	10	20
θ_{thigh} [deg]	-15	-7	0	10	20
θ_{HAT} [deg]	-10	-5	0	5	9

Table 6.3: Fuzzy rule sets for the posture control of the three segments.

output ΔT		inertial $\dot{\theta}$		
		N	ZE	P
inertial θ	NL	PL	PL	PL
	NS	PL	PS	NS
	ZE	PS	ZE	NS
	PS	PS	NS	NL
	PL	NL	NL	NL

the angular velocity, so there are altogether 15 ($=3*5$) fuzzy rules for the control of each segment (see Table 6.3). The rules are constructed based on biomechanical reasoning and some common sense insight. The quantified values are calculated from data of [193], [126], and the criteria are listed as follows.

One key feature to keep the body balance is that the COG of the body should be limited to the length of the foot in the sagittal plane during the stance phase. Once the COG extends beyond the dimension of the foot, the body cannot exert appropriate torques to counteract gravity, then the body cannot maintain the stable posture. Therefore,

$$-0.05 < l_{c1}\sin\theta_{HAT} + l_{c2}\sin\theta_{thigh} + l_{c3}\sin\theta_{shank} < 0.15 \quad (6.25)$$

where l_{c1} , l_{c2} and l_{c3} are the distance from the proximal joints of the three segments to their centers of gravity. The values are listed in Table 3.1.

According to the data of the actual human walking, the stable region for the inertial angles of the full cycle is constrained as: $-12^\circ < \theta_{HAT} < 9^\circ$, $-17^\circ < \theta_{thigh} < 25^\circ$, and $-54^\circ < \theta_{shank} < 22^\circ$. However, the inertial angles during swing phase should not affect the fuzzy rules. For this, the data of the swing phase are truncated. In this case, some large positive data are removed, that means the fuzzy controller will act significantly if the inertial angles are positive and large. It is meaningful for the body balance control because the muscle can produce large torque to prevent the

body from tumbling over backward.

Every rule is simply stated in “IF-THEN” linguistic language. For example, “IF θ is NL AND $\dot{\theta}$ is N, THEN ΔT is PL”. The “AND” operator in the premise is defined as a minimum operator, that is

$$u_{\Delta G_z} = AND(u_\theta, u_{\dot{\theta}}) = \min(u_\theta, u_{\dot{\theta}}) \quad (6.26)$$

where $u_{\Delta G_z}$, u_θ and $u_{\dot{\theta}}$ are the membership degrees for the gain of electrical stimulation, inertial angle and angular velocity respectively.

The defuzzication action uses the center of area (COA) method, which provides good performance for reducing steady-state error and has a simple algorithm in implementation.

$$\Delta G_z = \frac{\sum_{i=1}^N u_{\Delta G_{zi}} * \Delta G_{zi}}{\sum_{i=1}^N u_{\Delta G_{zi}}} \quad (6.27)$$

In fuzzy logic toolbox of Matlab, the function “centroid” can perform it easily.

6.3.3 Sensory Feedback

The sensory feedback plays a very important role during the human walking movement. Different types of sensory feedback are integrated to assist the walking movement. For example, the proprioceptor in the joint senses the positions and velocities of the joint; the cutaneous mechanosensor of the feet senses the touch of the ground; the vestibular system senses the body orientation during locomotion; the visual system senses the overall speed and direction of the locomotion. However, so far, it is impossible to incorporate all the sensory feedback information into FES design. There are several reasons for it. First, the function and mechanism of some sensory feedback still needs further exploration. Second, harmonization of so much different feedback information is also a big problem. Third, for the real application of

FES control system, there are not so advanced artificial sensors that can have the function as the human natural sensors. Therefore, we select several types of sensory feedback that are suitable for the FES locomotion, and some simplifications are made to match the practical application.

Noise Sensor: Increasing the CPG tonic input can resist the measurement noise significantly, and enhance the robustness of the FES control system. This function is realized by the noise sensor that detects the measurement noise, and then the feedback gain K_t is multiplied to the tonic input of CPG (see equations (6.5)-(6.8)).

Overall Speed Sensor: If the overall speed of the locomotion is changed, the patterns provided by CPG should also be changed correspondingly. In the CPG, the time constants τ_1 and τ_2 have the most significant effect on the rhythmic frequency. Here, an adaptive gain K_f is used to regulate the value of the time constant. In fact, we can also deem it as a signal from supraspinal system to initiate the locomotion at a certain speed. However, in this work, the locomotion is defined as a tracking problem. Therefore, we look on K_f as a feedback to sense the desired overall speed (see equations (6.5)-(6.8)).

Foot Touch Sensor: Foot touch sensor is a kind of cutaneous sensor, which aims to deal with the contralateral effect between the two legs during walking (see Figure 6.21).

The previous research has found that during the human walking movement, the rhythm of one leg will be affected by the other due to the bilateral coupling mechanism [206]. The coupling mechanism is generally deemed as the connection between the bilateral CPG units or the mechanical coupling from sensory feedback. The phenomenon can be observed as the movements of the two legs are phase-locked in an anti-phase relationship. That indicates the activation pattern of ipsilateral limb may have some phase relationship with that of the contralateral limb. However, the extremely complex mechanism is faraway to be fully understood. There are

only some assumptions about the causation of this phenomenon. (1) contralateral sensorimotor signals mediated through neural inter limb coupling mechanisms and (2) ipsilateral afferent signals triggered by the forces transmitted to the ipsilateral leg due to the acceleration or movement of the contralateral leg. That means the mechanical sensory feedback and neural coupling can both have effects on the contralateral activation of the muscles, and in fact it's hard to isolate these two factors during experiments [185].

In simulation study, different methods were used to model this effect. Some used the mechanical sensory feedback with global sensing [138], [194], [142]; while the others used the weak connections with global sensing between the CPG units [182]. It is still not determined whether bilateral coordination through mechanical coupling is equivalent, inferior, or superior to having explicit neural connections in bilateral CPG. But one thing is sure: mechanical sensory feedback with global sensing is easy to be performed in simulation study, as the parameters that need tuning for the CPG are much fewer.

Here, the mechanical sensory feedback through the foot touch sensors is used to coordinate the patterns of muscles for the two limbs. It can be formulated by the function $H(F_{foot})$, where $H(x)$ is the unit function that is defined as $H(x) = 1$ for $x > 0$ and $H(x) = 0$ for $x \leq 0$; F_{foot} is the reaction force sensed by foot. With regard to walking, it indicates the ground reaction force (GRF). Using this formulation, the mechanical sensory feedback from foot is normalized to 1 and it is quantified to a series of pulses. When it is fed back to the CPG, it can entrain the CPG and change the phase of the CPG's output. Thus, it is employed to make the two legs perform the walking movement with an anti-phase relationship.

Phase Switch: Sometimes the stance-swing phase switching control is proposed for FES walking [28]. With a neurophysiological point of view, position and load (force, [153]) are the most important variables concerned for the phase switching

in walking. It is believed that they are combined together to regulate the phase transition [23], [25], [140]. Although we know the two variables are correlated, it is still not clear how they are correlated at present. In real application, some researchers deem the position variable is indicated by inertial angle of body segment that is defined in Figure 6.25, tilt sensors or inclinometers are used to measure the inertial angles [28]. Recently, accelerometer and gyroscope are used to detect the gait phase based on the position variables (velocity and acceleration) [21], [101]. On another hand, load (force) variable can be represented by GRF, which can be measured by foot touch sensor [49], [197]. However, it is still not determined which variable is better in real application, and the combination of force and position is proposed by Pappas et. al. [141].

In fact, my work is not focused on the phase-switching control, and the stance-swing detection is not significant here because stimulation patterns for the muscles concerned are achieved via CPG during the full gait cycle. The stance-swing information is not useful to determine the stimulation patterns. Here, the stance-swing switch has different function with the traditional applications. Instead it just serves for the posture controller, because it is important for the body to keep balance during the stance phase. Therefore, sensor is needed to switch the posture controller during stance phase. Here, we use GRF to accomplish this task that will reduce the sensors needed such as accelerometer etc.

Vestibular System: The vestibular system is important for the posture control, as it can sense the orientation of the body and provide feedback information for the motor system. Here, it is simply represented by the term “COG”. The details are elaborated in the posture controller design of the previous section.

6.4 Simulation Study

6.4.1 General Study

Generally, there are two methods to generate walking movement in simulation study. One method is to define a task objective first such as minimization of the metabolic energy expenditure [6] and optimization of the travelling distance [138], then adjust the muscle excitations. The other method is to solve the tracking problem by adjusting the muscle excitations (stimulation) to replicate the experimental walking data as well as possible regardless whether the energy is minimized or the travelling distance gets away from the local minimum [137]. We prefer the latter approach as it is more promising to study the pathological gait and subject-specific gait. The desired joint angles during walking are adopted from [193], which are the control objectives for the tracking problem. The simulation study is performed in Matlab/Simulink environment. At first, the chief problem is to produce a stable rhythmic movement no matter whether it is close to the real human walking. Since the regulator's function is to perform the fine tuning with the errors between desired and actual trajectories in the later stage, it is temporally blocked.

The CPG, impedance controller and posture controller are used to control the FES locomotion. The CPG structure has been shown in Figure 6.11. Adjustment of the stimulation patterns for the muscles is mainly accomplished by the CPG. The parameters are achieved to guarantee the stimulation pattern conform to the desired EMG patterns of the muscles from real experimental measurement. The impedance controller uses the error feedback to regulate the intension of the electrical stimulation, which not only produces the large torques (i.e. provides the muscle with strong stimulation) for the segments, but also gives a coarse regulation for the joint angles (i.e. reduces the tracking errors). The fuzzy logic controller plays an important role here, which can maintain the body balance during walking. The sensory feedback of

Figure 6.27: Desired trajectories (ankle, hip and knee) of the lower limb during the real human walking.

foot touch is defined by the function $H(GRF)$, which can make the two legs move in the locked anti-phase. The GRF is shown in Figure 6.24 and the simulation result of $H(GRF)$ is shown in Figure 6.28(b).

Under this configuration, the system can control the human musculoskeletal model to generate a stable walking movement at 1 Hz as shown in Figure 6.28(a).

6.4.2 Speed Control

The tracking performance for desired overall speed is addressed here. In order to achieve the walking movement with varying speed, the speed sensory gain K_f is changed adaptively. Accordingly, different rhythms of the leg movement are achieved. The trajectories of the three joint angles during locomotion and the corresponding values of K_f are shown in Figure 6.29. We can see that the joint angles of high speed are larger than that of low speed. It is accordant with the actual human locomotion. When a person increases walking speed, his stride length is also increased, so the joint angles of limbs are changed largely. This phenomenon is very common in experiments and it also occurred in the simulation of the previous work.

6.4.3 Improvement

Although the musculoskeletal model can generate stable locomotion with the above FES control system and the output rhythm is accordant with the desired speed, the shape of the joint curves is far away from the natural human walk. The results are only acceptable in simulation study. In fact, a real human is different from the biped model and the natural and smooth trajectories are important. First, with the desired trajectories, the feedback information can regulate the muscles to coordinate well and produce appropriate stimulation to each muscle. Second, the natural human

Figure 6.28: Simulation results of (a) Three joint angles of the two lower limbs during walking. (b) Foot touch sensory feedback to the CPG .

Figure 6.29: Variation of the locomotion speed corresponding to the adaptive feedback frequency gain K_f . Walk rhythm is related with K_f : $K_f = 2 \rightarrow 1.2Hz$, $K_f = 1 \rightarrow 1.0Hz$, $K_f = 1.5 \rightarrow 1.2Hz$, $K_f = 0.6 \rightarrow 0.8Hz$.

joint angles of walking are trained through human nervous system since childhood, and they are the optimal curves that fit the human biomechanics perfectly. Human will feel comfortable and walk efficiently with the desired trajectories. Third, from the control engineering perspective, stability and tracking performance are two important factors to evaluate a control system. If the control system cannot control the output of the plant to track a desired trajectory, then it is not useful at all. So the significant meaning of this neural control system is to make the musculoskeletal model clone the real human walking perfectly. Moreover, if it can control the model to walk well, that means it can also control the model to track any other possible desired curves, and thus different types of movements can also be achieved. This versatile ability is meaningful.

The regulator is the key part to improve the tracking performance, which can regulate the stimulation intensity to the muscles in order to achieve the best result with the feedback errors. After the regulator is incorporated with the other components, the FES control system is finally fully constructed. The tracking performances

of the three joint angles are shown in Figures. 6.30-6.32, respectively.

We can see that the joint angles can track the desired trajectories in a time less than a cycle. Therefore, the tracking time is very short in fact. The steady tracking errors for the hip and knee joint are quite ideal, as they are less than 5° . For the ankle angle, the error seems a little larger, which is perhaps due to the irregular shape of the ankle curves. Another possible reason is that the posture control affects the torque of the ankle joint much, that will affect the tracking performance. Even so, the steady error is still acceptable.

The transient response for the three joints can be observed clearly from the plots of errors in Figures 6.30-6.32. The overshoot is mainly due to the time delay of the neural network in the regulator, however it does not affect the whole performance of the FES control system, because the posture controller has constrained it not to exceed the stable region. Moreover, the gait cycle begins at the swing phase, and large variations of the joint angles will not deteriorate the stability.

Figure 6.30: Tracking performance of hip joint. Tracking error is shown below.

Figure 6.31: Tracking performance of knee joint. Tracking error is shown below.

After the regulator is incorporated, the FES control system is fully developed as shown in Figure 6.7. Under the co-work of different controllers (e.g. CPG, regulator, posture controller, and impedance controller) and feedback sensors, the musculoskeletal model can successfully generate desired walking movement as shown in Figures 6.30-6.32. Along with the results of joint angles, the forces produced by each muscle can also be achieved in simulation, which are illustrated separately in Figure 6.35.

An important task is to check whether the force pattern generated in simulation is reasonable. An available method is to use EMG to compare with the force pattern in Figure 6.35. The experiment to measure EMG pattern of lower limb muscles during walking is conducted on a healthy subject in Biomedical Instrumentation Lab. Wireless EMG sensing system (Naroxon, USA) is used as it is convenient to measure EMG signal during human movement. The disadvantage is that the

Figure 6.32: Tracking performance of ankle joint. Tracking error is shown below.

signal is not as good as that of the wire EMG system such as Biopac system. The experiment on EMG measurement is shown in Figure 6.33. Altogether 9 channels are used to measure the surface EMG on nine muscles as shown in Figure 6.22. However, only four muscles can get ideal measurement of EMG and the results are shown in Figure 6.34. The possible reasons are given as follows. (1) Limitation of the wireless system. Its signal is not good in comparison with the wire system such as Biopac. (2) Surface EMG in itself contains much noise. (3) It needs much clinical experience to put the EMG electrodes on the accurate location of the specific muscle. In fact, some muscles of lower limb (e.g. ilipsoas) are located deeply under skin. Their EMG signals are very weak, and easily contaminated by noise. However, it doesn't matter, because some reputable literature can provides more accurate EMG patterns of the muscles concerned in walking [202].

The desired EMG patterns are shown during the first cycle in Figure 6.35. Note

Figure 6.33: Experiment on EMG measurement of a healthy subject during walking.

that the force pattern shows a good agreement with the EMG patterns. That means the CPG can provide appropriate patterns for the muscles. From the observation of the muscle's force, the role that each muscle plays during a cycle can be understood well. It is obvious that VAS and SOL make more contribution during the stance phase compared to the other muscles. With respect to VAS and RF, they were often combined together as “quadriceps” to be stimulated for the knee joint control in the previous work [19], [45]. Here, the simulation results show that their functions are very different. RF is active at two periods during one gait cycle (Note that its activity is sometimes absent in the beginning of the stance), and its force is not so significant as VAS. In fact, ankle planar flexors (SOL, GAS) and hip extensors (GMAX, HAM) are the most active muscles. Their active periods occur at the late stance and the beginning stance, respectively. Therefore, they can restore energy to the body near double support. It is an effective mechanism to utilize the passive

Figure 6.34: Raw EMG signals of four muscle groups (TA, BF, RF, and VAS) on a healthy subject during walking.

locomotion properties of the body. Particularly, GAS contributes much to the swing initiation. For the biarticular muscles (GAS, HAM), their lengths are nearly at the optimal length during the stance, and thus, it's possible and efficient for them to produce the maximum force.

Figure 6.35: Simulation results of the force produced by the nine muscles. The red bar indicates the desired EMG patterns. TO indicates “toe off” and HC indicates “heel contact”.

The torques acting at the three joints are shown in Figure 6.36. It can be seen that the ankle torque is nearly zero during the swing phase. This is reasonable, because the passive torque produced by the leg gravity is predominant at that phase and there is no need for the ankle to produce torque. Similarly, the hip and knee torques are not significant at the swing phase, too. The torque is irregular in the first cycle because of the transient overshoot of the regulator. This is accordant with the angle tracking performance.

Figure 6.36: Simulation results of the muscle torque acting at the three joints: ankle, knee and hip.

6.4.4 Further Evaluation

The posture control performance can be observed through the inertial angle's variation in Figure 6.37 and the phase plot of the inertial angles in Figure 6.38. It is shown that the inertial angles for the segments (HAT, thigh, and shank) are ideal in Figure 6.37, especially during the stance phase, because none exceeds the stable constraints. This is mainly due to the function of the fuzzy logic controller.

The relationship between the inertial angles and angular velocities are shown in Figure 6.38. The results are acceptable as every segment's trajectory finally stays in the stable cycles. The transient response is large (refer to the tracking performance) at the beginning in the phase plot, and it looks like going to be divergent, however within less than a cycle the trajectory converges to the steady state.

To test the ability of this FES control system to resist the mechanical disturbance, we

Figure 6.37: Simulation result of the inertial angles during walking movement. It is emphasized at the stance phase (black bar).

Figure 6.38: Phase plots of inertial angles for the four segments: (a) HAT, (b) thigh, (c) shank and (d) foot.

assume one leg is tumbled by an unexpected obstacle, and arbitrarily it happens at the time of 3rd second. In the simulation study, an impulse of torque with two times of the normal torque is applied. The simulation results in Figure 6.39 demonstrate good robustness to external disturbance in this case. With a slight vibration for less than 0.3 seconds, the curves of the joint angles return to the satisfactory level. This is mainly attributed to the function of the feedback controllers, i.e. impedance controller.

Figure 6.39: Ability of the FES control system to resist an unexpected disturbance. Unexpected torque is applied to foot at the 3rd second.

6.5 Discussion

The novelty of this FES control system is the combination of artificial control techniques and biological neural control mechanisms. This could be the future direction for the traditional control system.

The neural mechanism of the biological motor control system, CPG, is adopted

in the FES walking design. Most previous work deemed CPG a pure feedforward controller. Here, some feedback information are incorporated to regulate it. Therefore, it is appropriately named as “heterarchical” controller [22]. In fact, the CPG mechanism for the movement control is hardly to be defined because the neural and mechanical systems are dynamically coupled to each other and to the environment, and it is not always clear what exactly is being controlled and where the control originates.

Tuning the parameters of the CPG is the most important work. So far, there are three methods. First, the parameters are achieved according to the experience, and it is much like the trial-and-error method [194], [182]. Second, analytic methods are used to determine the parameters such as describing function [8] and contraction analysis [190]. Third, the appropriate parameters are achieved through optimization methods, such as genetic algorithms (GA) [65], [91], [138], [142]. This work mainly used the first two methods. However, due to the nonlinearity and complexity of the CPG, there is still not a mature adaptive mechanism for the precise tuning of the CPG’s parameters, especially during the realtime control. Therefore, this work is not only a rudimental try, it can also afford the possibility of further exploration.

The idea of the regulator is similar with the work of [3]. It models the complex neuronal circuit and pathways in the spinal cord except for the CPG and it is designed at a macroscopic level here, which is different from the other’s work based on the microscopic level [72], [138], [197]. It is quite understandable as our objectives are different. They aim to model the real neural circuit and pathways truly. However, this work aims to accomplish an engineering task, so it is important to make a good trade-off between complexity and tractability.

The integration of the sensory feedback is an active research area, too [32]. This work attempts to adopt as much as possible feedback information that is useful for the locomotion control. The integration of the feedback can provide the adaptive

mechanism for the CPG, the errors for the regulator and impedance controller, and inputs for the posture controller. However, visual information is not considered although it occupies about 80% of all the feedback information during a real human locomotion. In fact, this should be a potential direction for the further exploration.

With regard to the realization of the proposed FES control system, it is feasible, as the CPG has made great progress recently along with the robotic control, and some hardware has already been developed for the CPG design [113], [133]. Also, the traditional control techniques adopted here, such as fuzzy control and NN control are very mature and easy to be implemented. On the other hand, the sensors needed are also very common such as goniometer for the joint angles, press transducer for the foot touch and so on.

However, for practical clinical application, some problems such as muscle fatigue, atrophy, spasticity, contracture, unwanted reflex responses, discomfort, voluntary intervention, will definitely undermine the success of the proposed system. In my thesis, muscle fatigue is considered in the muscle model. However, for the paralyzed patients, generally the fatigue must be more severe than that of healthy subjects. In simulation, the value of fatigue time constant τ_{fat} in equation (3.19) can be reduced to indicate the case of severe fatigue. Due to muscle atrophy of the patients, the maximum force F_{max} in equation (3.2) is set as 70% of that for the normal person in simulation study. Spasticity is a big problem in FES application. So far, there is not an effective method that can cure the spasticity. It is reported that FES as a therapeutic method can reduce spasticity [30], [68], [77], [47], [176], [177], [167]. But with a point of view on FES control, spasticity has changed the normal muscle properties; therefore, the control performance under FES will be deteriorated. This problem is a large issue that deserves much exploration. For unwanted reflex response, we have a new project on this issue. For example, the involuntary tremor along with some stroke and Parkinson patients is expected to

be suppressed via FES [210]. All the problems mentioned above cannot be resolved once for all in my thesis, and they are the tasks for all the researchers in FES area. Perhaps the real application of the proposed FES system can be only suitable for a specific group of paralyzed patients, but the most highlighted point is the soft idea proposed in the thesis. It may have the potential for bio-inspired robot control in future.

Furthermore, the proposed model not only can be used for the future FES application, it is also a good tool to study the neural control mechanism and reveal the mechanical functions and coordinations of muscles during locomotion.

6.6 Summary

In this chapter, the neural mechanism of the biological motor system, CPG, was modeled for the FES control system design. In fact, this is a hybrid control system that combines biology and engineering together. Several control techniques were combined together to accomplish three types of the lower limb movements with FES: swinging, cycling and walking. Integration of different sensory feedback was incorporated to enhance the control performance. This FES control system has demonstrated satisfactory tracking performance, good stability and robustness. Moreover, these results are matched well with the experimental data presented in the previous work.

With regard to the FES swinging movement, this neural network control system designed in this chapter provides the perfect tracking performance with less tracking time. Especially, in this control system the sensory feedback effect is added into the CPG, which can endow the system with robustness and strong adaptive ability, so that a strong resistance against the measurement noise can be ensured and it is able to track the movement with varying speed. This is an important contribution.

A full FES control system is designed in this chapter for the cycling movement. It is worthy to note that the proposed FES system should be the most complete one for the leg cycling control. In terms of control performance, the tasks on tracking desired trajectories and maintaining crank speed are considered. In the design of the CPG, a fully connected CPG is developed, and mechanical coupling of the human body is built for the coordinated movement of the bilateral limbs.

For the FES walking movement, several important components are combined together to accomplish this delicate and fascinate walking task. For example, the CPG is in charge of generating the approximate rhythmic pattern, the regulator and impedance controller aim to reduce the tracking errors, and the posture controller is to maintain the body balance during walking. In fact, this complete control system can also be deemed a complex hybrid FES control system reflecting the natural human motor system creditably. New algorithms are developed in the RBF neural network design for the regulator. A fuzzy logic controller is designed to serve for the posture control. Also several types of sensory feedback are integrated for tuning this neural control system adaptively. Simulation study demonstrates the musculoskeletal system can perform smooth and accurate locomotion and maintain the posture well with this newly developed FES control system.

Chapter 7

Conclusions

7.1 Summary

This thesis has studied the central pattern generator (CPG) and modeled its key mechanisms to design a control system for human lower limb movements via functional electrical stimulation (FES). The main work is about the FES control. Chapter 3 provided the musculoskeletal model. Chapter 4 and Chapter 5 analyzed the neural control mechanism. The FES control system design was elaborated in Chapter 6.

With regard to the musculoskeletal model, the revised Hill-type muscle model is formulated and identified using experimental data from the reputable literatures. It refines the physiological properties of the muscle such as isometric recruitment curve, force-length-velocity relationship, viscous (elastic) passive element, and muscle fatigue etc. The skeletal dynamics is achieved with the method commonly used in robotics. This musculoskeletal model is useful to test the performance of the FES control system. This complex musculoskeletal model with 18 muscles and 7 segments is deemed the control plant for FES cycling and walking in my work.

With regard to the neural control mechanism, the kernel is the central pattern generator (CPG). We have performed the thorough exploration on CPG. The intrinsic and network mechanisms of CPG have been studied. The effects of the sensory feedback and tonic input are emphasized and some new results are achieved. Moreover, theoretical analysis is carried out on the neural control mechanism. For example, mathematical methods for nonlinear system such as describing function, piecewise linear analysis, contraction and stability theory are adopted. The following important properties are achieved from biology to engineering: stability, local stability, role of key ionic channels, conditions of oscillation, determination of frequency, network property etc. These theoretical analyses have laid a firm foundation and provided the effective tools for the design of FES controller.

The most important work is the FES control system design, which contains the CPG, regulator network, posture controller, impedance controller, and an integration of several types of sensory feedback. An artificial neural network is applied into the regulator design, and a fuzzy logic controller is in charge of the posture control. In fact, this is a hierarchical and hybrid control system modeling the natural human motor system. Three types of lower limb movements, swinging, cycling and walking, have been tested with this FES control system and it has demonstrated satisfactory performance.

7.2 Conclusion

The musculoskeletal model is formulated on the base of various reputable literatures. It has served as the control plant instead of a real patient in my work.

The CPG is the kernel of this FES control system. It aims to produce the rhythmic patterns for the individual muscles in order to achieve the desired movement. Its stable endogenous oscillatory curve is due to (1) the intrinsic ion channels of the

neuron and (2) the interaction amongst the coupled neurons. The neural oscillator is a simplified and generalized version of the complex CPG. Using the neural oscillator to perform the control task is not new, however, the adaptivity of the CPG with the sensory feedback was seldom considered in the previous work. This work has emphasized this part. In addition, a thorough theoretical analysis of the neural control mechanism is performed, and the properties of the CPG and neural oscillator are established by the mathematical methods such as describing function, piecewise linear analysis, and other modern control theories. The analysis has constructed a firm foundation for the FES control system design.

With regard to the FES control system design, it borrows the mechanism from biological motor system, at the same time, traditional techniques such as neural network control and fuzzy logic control are also adopted. Moreover, the integration of different sensory feedbacks has endowed the system with adaptive ability and enhanced its robustness.

Three control tasks have been accomplished, starting from the simple to the difficult ones, which are swinging, cycling and walking. For swinging (knee joint movement), the proposed neural control system can be transformed into a typical feedforward plus a feedback control system, which is quite competent for the simple control task. Moreover, in the premise of perfect tracking performance with less tracking time, its important feature is to track a trajectory with varying frequencies. For cycling movement, the comprehensive musculoskeletal model has been controlled via FES, and two objectives, angular control and cadence control, have been accomplished. For walking movement, posture control and rhythmic control are two important tasks. The simulation study has demonstrated good performance with this neural control system.

The work accomplished in the thesis is significant because it uses insights from neurobiological systems in the design of engineering control system. It can be seen

that the biological system can efficiently solve the problems that are similar with those faced in many engineering applications. The current work is mainly on the simulation stage, but it has the potential for the clinical application in future.

7.3 Future Work

So far this work has just explored a very small part of FES and human motor control, which actually is involved with a vast area. Therefore, it is believed that there are many topics that deserve further study.

- **Muscle Model**

The muscle model used in my work is based on the Hill-type muscle model. The identification of the muscle model except the muscle fatigue is mainly dependant on the experimental data from other literatures. In future, we expect to identify the muscle model using our own experiments. Moreover, some other important nonlinear effects, such as the influence of loading, length and activation history on length-force characteristics, should be studied. A more general muscle model is desired in future and more improvements are expected to make better compromise between tractability and credibility.

- **Body Dynamics**

Here, the planar model is constructed in two dimensions, which can only accounts for the basic movements in the sagittal plane. In fact, some movements such as pelvic lift occurs in frontal plane and hip rotation occurs in the transverse plane. In order to mimic the human natural movement, three-dimension (3D) model is desired. Recent research has provided a direction for the 3D movement [140]. However, the extreme large timing expenditure for computation is another problem along with it.

- **Central Pattern Generator**

In this dissertation, most analysis and simulation study is focused on single CPG and single neural oscillator. In future, the network property of multiple neural oscillators should be further explored. Some algorithms such as genetic algorithm (GA) should be developed to optimize the large number of parameters in CPG network. On the other hand, more investigation will be performed on the adaptive mechanism of CPG.

- **Sensory Feedback**

The role of sensory feedback is very important for the human movement control. Only several types of simple sensory feedback are considered in this dissertation. However, some important natural sensors such as visual sensor are not included into this FES control system. In the real human movement, vision occupies nearly 80% of all the sensory feedback information. In future, the vision information will be incorporated. In fact, some latest research has touched this area. Another problem is how to deal with the integration and synergy of the different types of sensory feedback in order to make efficient use of them.

- **Clinical Application**

The clinical application is the ultimate objective, so this control system should be evaluated on the human subjects. Now, the cooperation with National Neuroscience Institute and Tan Tock Seng Hospital has already begun. The data of experiments will verify the credibility of the musculoskeletal model and test the performance of the FES control system. In fact, the experimental project is underway in BioRobotics Lab at Nanyang Technological University.

- **Broadening Application**

This neural control mechanism can also be exploited for the bio-inspired robot

or biped control. Its characteristics derived from biological motor control is especially constructive for the artificial control system design.

Author's Publications

Journal Papers

- D. G. Zhang and K. Y. Zhu, "Model and control of the locomotion of a bio-mimic musculoskeletal biped", Journal of Artificial Life and Robotics, vol.10, pp.91-95, 2006.
- D. G. Zhang and K. Y. Zhu, "Computer simulation study on central pattern generator: From biology to engineering," International Journal of Neural Systems, vol.16, pp.405-422, 2006.
- D. G. Zhang and K. Y. Zhu, "Modeling biological motor control for human locomotion with functional electrical stimulation," Biological Cybernetics, vol.96, pp.79-97, 2007.
- D. G. Zhang and K. Y. Zhu, "Model and neural control of leg cycling movement," International Journal of Modelling, Identification and Control, accepted, 2006.
- D. G. Zhang and K. Y. Zhu, "Theoretical analysis on neural oscillator toward bio-mimic robot control," International Journal of Humanoid Robotics, accepted, 2007.
- D. G. Zhang and K. Y. Zhu, "On neural control mechanism of biological motor system," under review, IEEE Trans. System, Man and Cybernetics.

Conference Papers

- D. G. Zhang, H. G. Tan, W. Ferdinan, and W. T. Ang, "Functional electrical

stimulation in rehabilitation engineering: A survey,” iCREATe Conference, Singapore, April 2007.

- D. G. Zhang, K. Y. Zhu, and L. Lan, “Analysis of neural oscillator for bio-inspired robot control,” IEEE International Conference on Robotics, Automation and Mechatronics (RAM), Bangkok, June 2006. {Finalist of best student paper award.}
- D. G. Zhang and W. T. Ang, “Tremor suppression of elbow joint via functional electrical stimulation: A simulation study,” IEEE Conference on Automation Science and Engineering (CASE), Shanghai, October 2006.
- D. G. Zhang, K. Y. Zhu and H. Zheng, “Model the leg cycling movements with neural oscillator,” IEEE International Conference on Systems, Man and Cybernetics, Holland, October 2004.
- D. G. Zhang and K. Y. Zhu, “A structured neural network control system for FES cycling,” The 9th Annual Conference of International FES Society, Bournemouth, UK, September 2004.
- D. G. Zhang and K. Y. Zhu, “Simulation study of FES-Assisted standing up with neural network control,” The 26th Annual International Conference of the IEEE Engineering in Medicine and Biology Society, San Francisco, September 2004.
- D. G. Zhang and K. Y. Zhu, “Neural network control for leg rhythmic movements via functional electrical stimulation,” IEEE International Joint Conference on Neural Networks, Budapest, July 2004.
- D. G. Zhang, K. Y. Zhu, and S. M. Krishnan, “Design of a control system for leg planar cyclic movement,” World Congress on Medical Physics and Biomedical Engineering, Sydney, August 2003.
- D. G. Zhang and K. Y. Zhu, “A control system for knee joint control with functional electrical stimulation,” IEEE Intelligent Automation Conference, Hong Kong, December 2003.
- K. H. Ew, C. L. Wee, D. G. Zhang, K. Y. Zhu, and H. Zheng, “Effects of functional electrical stimulation relating to leg movement,” The 27th Annual International Conference of the IEEE Engineering in Medicine and Biology Society, Shanghai, September 2005.

- H. Zheng, K. Y. Zhu and D. G. Zhang, “Design of an adaptive drug delivery control system,” International Conference on Control and Automation (ICCA2005), Budapest, June 2005.
- K .Y. Zhu, D. G. Zhang, and L. Lan, “On central pattern generator of biological motor system,” International Conference on Control, Automation, Robotics and Vision (ICARCV), Singapore, 2006.
- L. Lan, K .Y. Zhu, and D. G. Zhang, “Modeling and control of human motor system with generalized predictive control,” IEEE International Conference on Robotics, Automation and Mechatronics (RAM), Bangkok, June 2006.

Bibliography

- [1] J. J. Abbas and R. J. Triolo, "Experimental evaluation of an adaptive feed-forward controller for use in functional neuromuscular stimulation systems," *IEEE Trans. Rehab. Eng.*, vol.5, no.1, Mar. 1997.
- [2] J. J. Abbas, *Neural Network Control of Functional Neuromuscular Stimulation System*, Ph.D Thesis, 1992.
- [3] J. J. Ababas and H. J. Chizeck "Neural networks for control of functional neuromuscular stimulation systems: computer simulation study," *IEEE Trans. Biomed. Eng.*, vol.4, no.11, pp.1117-1127, 1995.
- [4] M. M. Adamczyk and P. E. Crago, "Simulated feedforward neural network coordination of hand grasp and wrist angle in a neuroprosthesis," *IEEE Trans. Rehab. Eng.*, vol.8, no.3, pp.297-304, Sep. 2000.
- [5] J. H. J. Allum, B. R. Bloem, M. G. Carpenter, M. Hulliger and M. Hadders-Algra, "Proprioceptive control of posture: A review of new concepts," *Gait and Posture*, vol.8, pp.214-242, 1998.
- [6] F. C. Anderson and M. G. Pandy, "Dynamic optimization of human walking," *J. Biomech. Eng.*, vol.123, pp.381-390, 2001.
- [7] F. C. Anderson, *A dynamic optimization solution for a complete cycle of normal gait*, PhD thesis. Univ. Tex., Austin. 1999.
- [8] A. M. Arsenio, "On stability and error bounds of describing functions for oscillatory control of movements," *IEEE Int. Conf. Intelligent Robots and Systems*, 2000.
- [9] K. J. Aström, "Oscillations in systems with relay feedback," in *Adaptive Control, Filtering, and Signal Processing*, vol.74, pp.1-25, 1995.

- [10] J. S. Bay and H. Hemami, "Modeling of a neural pattern generator with coupled nonlinear oscillators," *IEEE Biomed. Eng.*, vol.34, pp.297-306, 1987.
- [11] A. Billard, and A. J. Ijspeert, "Biologically inspired neural controllers for motor control in a quadruped robot," *IEEE Intern. Joint Conference on Neural Networks (IJCNN)*, pp.637-641, 2000.
- [12] J. Bobet and R. B. Stein, "A simple model of force generation by skeletal muscle during dynamic isometric contractions," *IEEE Trans. Biomed. Eng.*, vol.45, no.8, pp.1010-1016, 1998.
- [13] R. Brissot, P. Gallien, M. P. L. Bot, A. Beaubras, D. Laisne, J. Beillot, and J. Dassonville, "Clinical experience with functional electrical stimulation-assisted gait with parastep in spinal cord-injured patients," *Spine*, vol.25, pp.501-508, 2000.
- [14] L. Brodin, H. Traven, A. Lansner, P. Wallen, S. Grillner, "Computer simulations of N-Methyl-D-Aspartate receptor-induced membrane properties in a neuron model," *J. Neurophy.*, vol.66, pp.473-484, 1991.
- [15] S. J. Butt, R. M. Harris-Warrick, and O. Kiehn, "Firing properties of identified interneuron populations in the mammalian hindlimb central pattern generator," *J. Neurosci.*, vol.22, pp.9961-71, 2002.
- [16] G. Campa, F. L. Fravolini, and M. Napolitano, "A library of adaptive neural network for control purpose," *Proc. IEEE Int. Symp. on Computer Aided Control System Design*, Glasgow, pp.115-120, Sep. 2002.
- [17] P. Cesari, T. Shiratori, P. Olivato, and M. Duarte, "Analysis of kinematically redundant reaching movement using the equilibrium-point hypothesis," *Biol. Cybern.* vol.84, pp.217-226, 2001.
- [18] H. J. Chizeck, "Adaptive and nonlinear control methods for neural prostheses," in *Neural Prostheses: Replacing Motor Function After Disease or Disability*, Oxford University Press, pp.298-328, 1992.
- [19] G. C. Chang, J. J. Luh, G. D. Liao, J. S. Lai, C. K. Cheng, B. I. Kuo and T. S. Kuo, "A neuro-control system for the knee joint position control with quadriceps stimulation," *IEEE Trans. Rehab. Eng.*, vol.5, no.1, pp.2-11, Mar. 1997.

- [20] J. J. Chen, N. Y. Yu, D. G. Huang, B. T. Ann, and G. C. Chang, "Applying fuzzy logic to control cycling movement induced by functional electrical stimulation," *IEEE Trans. Rehab. Eng.*, vol.5, no.2, pp.158-168, Jun. 1997.
- [21] I. Cikajlo and T. Bajd, "Swing phase estimation in paralyzed persons walking," *Technology and Health Care*, vol.5, pp.425-433, 2002.
- [22] A. H. Cohen, "The role of heterarchical control in the revolution of central pattern generators," *Brain Behav. Evol.*, vol.40, pp.112-124, 1992.
- [23] H. Cruse, "Which parameters control the leg movement of a walking insect?" *J. exp. Biol.* vol.116, pp.357-362, 1985.
- [24] H. Cruse "What mechanisms coordinate leg movement in walking arthropods?" *Trends in Neurosciences*, vol.12, pp.15-21, 1990.
- [25] P. E. Crago, M. A. Lemay, and L. Liu, "External control of limb movements involving environmental interactions," in *Multiple Muscle Systems*, New York: Springer-Verlag, 1990.
- [26] P. E. Crago, R. J. Nakai, and H. J. Chizeck, "Feedback regulation of hand grasp opening and contact force during stimulation of paralyzed muscle," *IEEE Trans. Biomed. Eng.*, vol.38, no.1, pp.17-28, Jan. 1991.
- [27] P. R. Davidson, R. D. Jones, J. H. Andreae, and H. S. Sirisena, "Simulating closed- and open-loop voluntary movement: a nonlinear control-systems approach," *IEEE Trans. Rehab. Eng.*, vol.49, no.11, pp.2-11, Nov. 2002.
- [28] R. Dai, R. B. Stein, B. J. Andrews, K. B. James, and M. Wieler, "Application of tilt sensors in functional electrical stimulation," *IEEE Trans. Rehab. Eng.*, vol.4, pp.63-72, 1996.
- [29] R. Davoodi and B. J. Andrews, "Computer simulation of FES standing up in paraplegia: A self-adaptive fuzzy controller with reinforcement learning," *IEEE Trans. Rehab. Eng.*, vol.6, no.2, pp.151-161, Jun. 1998.
- [30] J. P. Dewald, J. D. Given, and W. Z. Rymer, "Long-lasting reductions of spasticity induced by skin electrical stimulation," *IEEE Trans. Rehab. Eng.*, vol.4, pp.231-242, 1996.
- [31] S. L. Delp, "Parameters for a model of the lower limb," Online data of ISB website, <http://isbweb.org/data/delp/index.html>

- [32] M. H. Dickinson, C. T. Farley, R. J. Full, M. A. R. Koehl, R. Kram, and S. Lehman, "How animals move: An integrative view," *Science*, vol.288, pp.100-106, Apr. 2000.
- [33] J. Ding, A. S. Wexler, and S. A. Binder-Macleod, "A predictive fatigue model I: Predicting the effect of stimulation frequency and pattern on fatigue," *IEEE Trans. Neural Systems and Rehab. Eng.*, vol.10, pp:48-58, 2002.
- [34] J. Ding, A. S. Wexler, and S. A. Binder-Macleod, "A predictive fatigue model II: Predicting the effect of resting times on fatigue," *IEEE Trans. Neural Systems and Rehab. Eng.*, vol.10, pp:59-67, 2002.
- [35] K. Doya, H. Kimura, and M. Kawato, "Neural mechanisms of learning and control," *IEEE Control Systems Magazine*, vol.21, no.4, pp.42-54, 2001.
- [36] S. J. Dorgan and M. J. O'Malley, "A nonlinear mathematical model of electrically stimulated skeletal muscle," *IEEE Trans. Rehab. Eng.*, vol.5, No.2, pp.179-194, June 1997.
- [37] H. Dou, K. K. Tan, T. H. Lee, and Z. Zhou, "Iterative learning feedback control of human limbs via functional electrical stimulation," *Control Engineering Practice*, vol.7, pp.315-325, 1999.
- [38] W. K. Durfee and K. E. Maclean, "Methods for estimating isometric recruitment curves of electrically stimulated muscle," *IEEE Trans. Biomed. Eng.*, vol.36, pp.654-666, Jul. 1989.
- [39] W. K. Durfee and K. I. Palmer, "Estimation of force-activation, force-length, and force-velocity properties in isolated electrically stimulated muscle," *IEEE Trans. Biomed. Eng.*, vol.41, no.3, pp.205-216, 1994.
- [40] T. Edrich, R. Riener, and J. Quintern, "Analysis of passive elastic joint moments in paraplegics," *IEEE Trans. Biomed. Eng.*, vol.47, pp.1058-1065, 2000.
- [41] Ö. Ekeberg, "Simulation of the spinal circuits controlling swimming movement," in *Biomechanics and Neural Control of Posture and Movement*, pp.221-230, 2002.
- [42] Ö. Ekeberg, P. Wallen, A. Lansner, H. Traven, L. Brodin, and S. Grillner, "A computer based model for realistic simulations of neural networks," *Biol. Cybern.*, vol.65, pp.81-90, 1991.

- [43] Ö. Ekeberg, "A combined neuronal and mechanical model of fish swimming." *Biol. Cybern.*, vol.69, pp.363-374, 1993.
- [44] G. Endo, J. Morimoto, J. Nakanishi, and G. Cheng, "An empirical exploration of a neural oscillator for biped locomotion control," *IEEE Intern. Conf. on Robotics and Automation (ICRA)*, LA, April, 2004.
- [45] M. Ferrarin, F. Palazzo, R. Riener, and J. Quintern, "Model-based control of FES-induced single joint movements," *IEEE Trans. Neural System and Rehab. Eng.*, vol.9, pp.245-257, 2001.
- [46] M. Frasca, P. Arena, and L. Fortuna, *Bio-inspired Emergent Control of Locomotion Systems*, World Science, 2004.
- [47] A. Franek, B. Turczynski, and J. Opara "Treatment of spinal spasticity by electrical stimulation," *J. Biomed. Eng.*, vol.10, pp.266-270, 1988.
- [48] H. M. Franken, P. H. Veltink, G. Baardman, R. A. Redmeyer, and H. B. K. Boom, "Cycle-to-cycle control of swing phase of paraplegic gait induced by surface electrical stimulation," *Med. Biol. Eng. Comput.*, vol.33, no.3, pp.440-451, 1995.
- [49] Y. Fukuoka, A. H. Cohen, "Adaptive dynamic walking of a quadruped robot on irregular terrain based on biological concepts," *Intern. J. Robotics Research*, vol.22, pp.187-202, 2003.
- [50] M. Fujita, "Adaptive filter model of the cerebellum," *Biol. Cybern.*, vol.45, pp.195-206, 1982.
- [51] A. P. Georgeopoulos and A. B. Schwartz, "Neuronal population coding of movement direction," *Science*, vol.233, pp.1416-1419, 1986.
- [52] B. K. Ghosh and J. He, "Some new challenge," *IEEE Control Systems Magazine*, vol.21, no.4, pp.27, Aug. 2001.
- [53] R. M. Ghigliazza and P. Holmes, "Minimal models of bursting neurons: how multiple currents, conductances, and timescales affect bifurcation diagrams," *SIAM J. Applied Dynamical Systems*, vol.3, no.4, pp.636-670, 2004.
- [54] R. M. Ghigliazza and P. Holmes, "A minimal model of a central pattern generator and motoneurons for insect locomotion," *SIAM J. Applied Dynamical Systems*, vol.3, no.4, pp.671-700, 2004.

- [55] H. Gollee, D. J. Murray-Smith, and J. C. Jarvis, "A nonlinear approach to modelling of electrically stimulated skeletal muscle," *IEEE Trans. Biomed. Eng.*, vol.48, pp.406-415, 2001.
- [56] J. M. Goncalves, "Regions of stability for limit cycles of piecewise linear system," *IEEE Conf. Decision and Control*, Maui, Hawaii, Dec. 2003.
- [57] J. Gordon, "Spinal Mechanisms of Motor Coordination." in *Principles of Neural Science*, 3rd Ed. (Norwalk, Connecticut: Appleton Lange), pp.581-95, 1991.
- [58] S. Grillner, L. Cangiano, G. Y. Hu, R. Thompson, R. Hill, and P. Wallen, "The intrinsic function of a motor system - from ion channels to networks and behavior," *Brain Research*, vol.886, pp.224-236, 2000.
- [59] S. Grillner and P. Wallen, "Cellular bases of a vertebrate locomotor system - steering, intersegmental and segmental co-ordination and sensory control," *Brain Research Reviews*, pp.92-106, Oct. 2002.
- [60] D. Graupe and H. Kordylewski, "Artificial neural network control of FES in paraplegics for patient responsive ambulation," *IEEE Trans. Biomed. Eng.*, vol.42, no.7, pp.699-707, Jul. 1995.
- [61] S. Grillner, "Neural networks for vertebrate locomotion," *Sci. Am.*, pp.48-53, 1996.
- [62] R. Grasso, M. Zago, and F. Lacquaniti, "Interactions between posture and locomotion: Motor patterns in humans walking with bent posture versus erect posture," *J. Neurophysiol.*, vol.83, pp.288-300, 2000.
- [63] S. Grillner, "Neurobiological bases of rhythmic motor acts in vertebrates," *Science*, vol.228, pp.143, 1985.
- [64] H. Hatze, "The inverse dynamics problem of neuromuscular control," *Biol. Cybern.*, vol.82, pp.133-141, 2000.
- [65] K. Hase, K. Miyashita, S. Ok and Y. Arakawa, "Human gait simulation with a neuromusculoskeletal model and evolutionary computation," *J. Visual. Comput. Animat.*, vol.14, pp.73-92, 2003.
- [66] H. Hatze, "Neuromusculoskeletal control systems modeling - A critical survey of recent developments," *IEEE Trans. Automat. Control*, vol.AC-25, pp.375-385, 1980.

- [67] M. S. Hatwell, B.J. Oderkerk, C. A. Sacher, and G. F. Inbar, "The development of a model reference adaptive controller to control the knee joint of paraplegics," *IEEE Trans. Automat. Contr. Eng.*, vol.36, no.6, pp.683-691, Jun. 1991.
- [68] J. S. Han, X. H. Chen, Y. Yuan, and S. C. Yan, "Transcutaneous electrical nerve stimulation for treatment of spinal spasticity," *Chin Med. J. (Engl.)*, vol.107, pp.6-11, 1994.
- [69] R. Happee, "Inverse dynamic optimization including muscular dynamics, a new simulation method applied to goal directed movements," *J. Biomech.*, vol.27, pp.953-960, 1994.
- [70] R. L. Hart, K. L. Kilgore and P. H. Peckham, "A comparison between control methods for implanted FES Hand-Grasp systems," *IEEE Trans. Rehab. Eng.*, vol. 6, no. 2, pp. 201-208, Jun. 1998.
- [71] F. T. Hambrecht, "A brief history of neural prostheses for motor control of paralyzed extremities," in *Neural Prostheses: Replacing Motor Function after Disease or Disability*, Oxford University Press, pp.1-14, 1992.
- [72] J. He, M. G. Maltenfort, Q. Wang and T. M. Hamm, "Learning from biological system: Modeling neural control," *IEEE Control Systems Magazine*, vol.21, no.4, pp.55-69, Aug. 2001.
- [73] D. O. Hebb, *The Organization of Behaviour*, Wiley, New York, 1949.
- [74] J. He, W. S. Levine, and G. E. Loeb, "Feedback gains for correcting small perturbations to standing posture," *IEEE Trans. Automatic Control*, pp.322-332, vol.36, no.3, 1991.
- [75] A. V. Hill, "The heat of shortening and dynamic constants of muscle," *Proc. R. Soc., Lond. [Biol.]*, vol.159, pp.136-195, 1938.
- [76] T. L. Hill, *Free Energy Transduction in Biology*, New York: Academic Press, 1977.
- [77] A. E. Hines, P. E. Crago, and C. Billian, "Functional electrical stimulation for the reduction of spasticity in the hemiplegic hand. *Biomed. Sci. Instrum.*, vol.29, pp.259-266, 1993.
- [78] S. L. Hooper and R. A. Dicaprio, "Crustacean motor pattern generator networks," *Neurosignals*, vol.13, pp.50-69, 2004.

- [79] A. L. Hodgkin and A. F. Huxley, "A quantitative description of membrane current and its application to conduction and excitation in nerve," *J. Physiol. Lond.*, vol.117, pp.546-562, 1952.
- [80] J. J. Hopfield, "Neural network and physical systems with emergent collective computational abilities," *Proc. of the National Academy of Sciences*, vol.79, pp.2554-2558, 1982.
- [81] N. Hogan, 1985, "Impedance control: An approach to manipulation," *J. Dyn. Syst. Meas. Control*, vol.107, pp.1-24, 1985.
- [82] M. G. Hoy, F. E. Zajac and M. E. Gordon, "A musculoskeletal model of the human lower extremity: the effect of muscle, tendon, and moment arm on the moment-angle relationship of musculotendon actuators at the hip, knee, and ankle," *J. Biomech.*, vol.23, pp.157-69, 1990.
- [83] F. C. Hoppensteadt and E. M. Izhikevich, *Weakly Connected Neural Networks*, Springer, 1997.
- [84] J. C. Houk, T. Buckingham, and A. G. Barto, "Models of the cerebellum and motor learning," *Behavioral and Brain Science*, vol.19, in press, 1996.
- [85] K. J. Hunt, R. Jaime, and H. Gollee, "Robust control of electrically-stimulated muscle using polynomial H-infinity design," *Control Engineering Practice*, vol.9, no.3, pp.313-328, 2001.
- [86] K. J. Hunt, B. Stone, N. Negard, T. Schauer, M. H. Fraser, A. J. Cathcart, C. Ferrario, S. A. Ward, and S. Grant, "Control strategies for integration of electric motor assist and functional electrical stimulation in paraplegic cycling: Utility for exercise testing and mobile cycling," *IEEE Trans. Neural Systems and Rehab. Eng.*, vol.12, no.1, 2004.
- [87] K. J. Hunt, M. Munih, and N. Donaldson, "Feedback control of unsupported standing in paraplegia-part I: optimal control approach," *IEEE Trans. Rehab. Eng.*, vol.5, no.4, pp.331-340, Dec. 1997.
- [88] K. J. Hunt, M. Munih, N. Donaldson, and F. M. D. Barr, "Optimal control of ankle joint moment: toward unsupported standing in paraplegia," *IEEE Trans. Automat. Control*, vol.43, pp.819-832, 1998.
- [89] A. F. Huxley, "Muscle structure and theories of contraction," *Prog. Biophys. Biophys. Chem.*, vol.7, pp.257-318, 1957.

- [90] J. J. Hu, M. M. William, and G. A. Pratt, "Bipedal locomotion control with rhythmic oscillators," *Proc. Int. Conf. Intelligent Robots and Systems*, pp.1475-1481, 1999.
- [91] A. J. Ijspeert, J. Hallam, and D. Willshaw, "Evolving swimming controllers for a simulated lamprey with inspiration from neurobiology," *Adaptive Behavior*, vol.7, pp.151-172, 1999.
- [92] J. C. Jarvis and N. J. M. Rijkhoff, "Functional electrical stimulation for control of internal organ function," *Neuromodulation*, vol.4, no.4, pp.155-164, 2001.
- [93] S. Jezernik, R. G. V. Wassink, and T. Keller, "Sliding mode closed-loop control of FES: Controlling the shank movement," *IEEE Trans. Biomed. Eng.*, vol.51, no.2, pp.263-272, 2004.
- [94] R. Jacobs and A. B. Jacobs, "Neuromuscular control strategies in postural coordination," in *Biomechanics and Neural Control of Posture and Movement*, pp.300-309, 2002.
- [95] S. Jonic, T. Jankovic, V. Gajic, and D. Popovic, "Three machine learning techniques for automatic determination of rules to control locomotion," *IEEE Trans. Biomed. Eng.*, vol.46, pp.300-310, Mar. 1999.
- [96] S. A. Kautz, E. F. Coyle, and A. M. Baylor, "The pedaling technique of elite endurance cyclists: changes with increasing workload at constant cadence," *Int. J. Sport Biomech.*, vol.7, pp.29-53, 1991.
- [97] M. Kawato, "Internal models for motor control and trajectory planning," *Current Opinion in Neurobiology*, vol.9, pp.718-727, 1999.
- [98] T. Keller, M. R. Popovic, and I. P. Muller, "Transcutaneous functional electrical stimulator 'Compex Motion'," *Artif. Organs.*, vol.37, pp.219-223, 2002.
- [99] G. Khang and F. E. Zajac, "Paraplegic standing controlled by functional neuromuscular stimulation: I, II," *IEEE Trans. Biomed. Eng.*, vol.36, pp.873-884, 1989.
- [100] K. L. Kilgore and R. F. Kirsch, "Upper and lower extremity motor neuroprostheses," in *Neuroprosthetics: Theory and Practice*, pp.845-877, 2004.
- [101] D. Kotiadis, H. J. Hermens, P. H. Veltink, P. Slycke, "Inertial gait phase detection system: design," 9th Annual Conference of the International FES Society, Bournemouth, UK, September, 2004.

- [102] A. Kostove, B. J. Andrews, D. B. Popovic, R. B. Stein, and W. W. Armstrong, "Machine learning in control of functional electrical stimulation systems for locomotion," *IEEE Trans. Biomed. Eng.*, vol.42, no.6, pp.541-551, Jun. 1995.
- [103] A. Kralj and T. Bajd, *Functional Electrical Stimulation: Standing and Walking after Spinal Cord Injury*, CRC Press, Boca Raton, FL, USA, 1989.
- [104] A. Kralj, T. Bajad, M. Munih, and R. Turk, "FES gait restoration and balance control in spinal cord injured patients," *Progress Brain Res.*, vol.97, pp.387-396, 1993.
- [105] A. D. Kuo, "The relative roles of feedforward and feedback in the control of rhythmic movements," *Motor Control*, vol.6, pp.129-145, Jun. 2002.
- [106] A. D. Kuo, "An optimal control model for analyzing human posture balance," *IEEE Trans. Biomed. Eng.*, vol.42, no.1, pp.87-101, Jan. 1995.
- [107] N. Lan, "Stability analysis for posture control in a two-joint limb system," *IEEE Trans. Rehab. Eng.*, vol.10, pp.249-259, 2002.
- [108] N. Lan, P. E. Crago, and H. J. Chizeck, "Control of end-point forces of a multi-joint limb by functional neuromuscular stimulation," *IEEE Trans. Biomed. Eng.*, vol.38, no.10, pp.953-965, Oct. 1991
- [109] N. Lan, H. Q. Feng, and P. E. Crago, "Neural network generation of muscle stimulation patterns for control of arm movements," *IEEE Trans. Rehab. Eng.*, vol.2, no.4, pp.213-224, Dec. 1994.
- [110] N. Lan and P. E. Crago, "Optimal control of antagonistic muscle stiffness during voluntary movements," *Biol. Cybern.*, vol.71, pp.123-135, 1994.
- [111] N. Lan, "Analysis of an optimal control model of multi-joint arm movements," *Biol. Cybern.*, vol.76, pp.107-117, 1997.
- [112] M. A. Lewis, M. J. Hartmann, R. E. Cummings, A. H. Cohen, "Control of a robot leg with an adaptive VLSI CPG chip," *Neurocomputing*, pp.38-40, pp.1409-1421, 2001.
- [113] M. A. Lewis, E. C. Ralph, M. J. Hartmann, Z. R. Xu, and A. H. Cohen, "An in silico central pattern generator: silicon oscillator, coupling, entrainment, and physical computation," *Biol. Cybern.*, vol.88, pp.137-151, 2003.

- [114] M. A. Lewis, F. Tenore, and R. E. Cummings, "CPG design using inhibitory networks," *Intern. Conf. on Robotics and Automation (ICRA)*, Barcelona, 2004.
- [115] W. S. Levine and G. E. Loeb, "The neural control of limb movement," *IEEE Control Systems Magazine*, vol.12, no.6, pp.38-47, Dec. 1992.
- [116] L. Liu, A. B. Wright, and G. T. Anderson, "Trajectory planning and control for a human-like robot leg with coupled neural-oscillators," *Proc. of Mechatronics*, 2000.
- [117] W. T. Liberson, H. J. Holmquest, D. Scott, and M. Dow, "Functional electrotherapy in stimulation of the peroneal nerve synchronized with the swing phase of gait in hemiparetic patients," *Arch. Phys. Med. Rehabil.*, vol.42, pp.101-105, 1961.
- [118] D. C. Lin and W. Z. Rymer, "A quantitative analysis of pendular motion of the lower leg in spastic human subjects," *IEEE Trans. Biomed. Eng.*, vol.38, pp.906-918, 1991.
- [119] X. F. Li and S. J. Xiao, "Model predictive control of human elbow joint movement," *Proc. Int. Conf. IEEE EMBS*, vol.20, no.5, pp.2366-2368, 1998.
- [120] W. Lohmiller and J. E. Slotine, "On contraction analysis for nonlinear systems," *Automatica*, vol.34, no.6, pp.683-696, 1998.
- [121] G. E. Loeb, I. E. Brown, E. J. Cheng, "A hierarchical foundation for models of sensorimotor control," *Exp. Brain Res.*, vol.126, no.1, pp.1-18, 1999.
- [122] Y. Lu, N. Sundararajan, and P. Saratchandran, "Performance evaluation of a sequential minimal radial basis function (RBF) neural network learning algorithm," *IEEE Trans. Neural Network*, vol.9, pp.308-318, 1998.
- [123] G. M. Lyons, T. Sinkj, J. H. Burridge, and D. J. Wilcox, "A review of portable FES-based neural orthoses for the correction of drop foot," *IEEE Trans. Neural Syst. Rehab. Eng.*, vol.10, no.4, pp.260-279, 2002.
- [124] E. Marder and D. Bucher, "Central pattern generators and the control of rhythmic movements," *Current Biology*, vol.11, no.23, pp.986-996, 2001.
- [125] K. Matsuoka, "Sustained oscillations generated by mutually inhibiting neurons with adaption," *Biol. Cybern.*, vol.52, pp.367-376, 1985.

- [126] Z. Matjacic and T. Bajd, "Arm-free paraplegic standing—Part I: control model synthesis and simulation," *IEEE Trans. Rehab. Eng.*, vol.6, no.2, pp.125-138, Jun. 1998.
- [127] M. Matsugu, J. Duffin, and C. S. Poon, "Entrainment, instability, quasi-periodicity, and chaos in a compound neural oscillator," *J. Computational Neuroscience*, vol.5, pp.35-51, 1998.
- [128] W. S. McCulloch and W. Pitts, "A logical calculus of the ideas immanent in nervous activity," *Bulletin of Mathematical Biophysics*, vol.9, pp.127-147, 1943.
- [129] T. A. McMahon, *Muscles, Reflexes, and Locomotion*, NJ: Princeton University Press, 1984.
- [130] S. Mori, "Integration of posture and locomotion in acute decerebrate cats in awake, freely moving cats," *Prog. Neurobiol.*, vol.28, pp.161-195, 1987.
- [131] J. H. Moe and H. W. Post, "Functional electrical stimulation for ambulation in hemiplegia," *Lancet*, vol.82, pp.285-288, 1962.
- [132] D. P. T. Nanayakkara, K. Watanabe, K. Kiguchi, and K. Izumi, "Fuzzy self-adaptive RBF neural network based control of a seven-link redundant industrial manipulator," *Advanced Robotics*, vol.15, no.1, pp.17-43, 2001.
- [133] K. Nakada, T. Asai, and Y. Amemiya, "An analog CMOS central pattern generator for interlimb coordination in quadruped locomotion," *IEEE Trans. Neural Networks*, vol.14, pp.1356-1365, 2003.
- [134] J. Nakanishi, J. Morimoto, G. Endo, G. Cheng, S. Schaal, and M. Kawato, "Learning from demonstration and adaptation of biped locomotion," *Robotics and Autonomous Systems*, vol.47, pp.79-91, 2004.
- [135] R. H. Nathan, "Control strategies in FNS systems for the upper extremities," *Cri. Rev. Biomed. Eng.*, vol.21, pp.485-568, 1993.
- [136] P. R. Neptune, I. C. Wright, A. J. van den Bogert, "A method for numerical simulation of single limb ground contact events: application to heel-toe running," *Comparative Methods of Biomechanical and Biomedical Engineering*, vol.3, no.2, pp.321-334, 2000.

- [137] P. R. Neptune, S. A. Kautz, and F. E. Zajac, "Contribution of the individual ankle planar flexors to support, forward progression and swing initiation during walking," *J. Biomech.*, vol.34, pp.1387-1398, 2001.
- [138] N. Ogihara and N. Yamazaki, "Generation of human bipedal locomotion by a bio-mimetic neuro-musculo-skeletal model," *Biol. Cybern.*, vol. 84, pp.1-11, 2001.
- [139] M. G. Pandy, "Computer modelling and simulation of human movement," *Annu. Rev. Biomed. Eng.*, vol.3, pp.245-273, 2000.
- [140] M. Y. C. Pang and J. F. Yang, "The initiation of the swing phase in human infant stepping: importance of hip position and leg loading" *J. Physiology*, vol.528, pp.389-404, 2000.
- [141] I. P. I. Pappas, M. R. Popovic, T. Keller, V. Dietz, and M. Morari, "A reliable gait phase detection system," *IEEE Trans. Neural. Syst. Rehab. Eng.*, vol.9, no.2, 2001.
- [142] C. Paul, "Bilateral decoupling in the neural control of biped locomotion," *Proc. 2nd Intern. Symp. on Adaptive Motion of Animals and Machines*, Kyoto, Japan, 2003.
- [143] M. G. Pandy, F. E. Zajac, E. Sim and W. S. Levine, "An optimal control model for maximum-height human jumping," *J. Biomech.*, vol.23, pp.1185-98, 1990.
- [144] J. S. Petrofsky, "New algorithm to control a cycle ergometer using electrical stimulation," *Med. Biol. Eng. Comput.*, vol.41, pp.18-27, 2003.
- [145] D. Popovic, and T. Sinkjer, *Control of Movement for the Physically Disabled*, in press, Springer, 2000.
- [146] M. R. Popovic, T. Keller, Ion P. I. Pappas, V. Dietz, and M. Morari, "Surface-stimulation technology for grasping and walking neuroprostheses," *IEEE Eng. Med. Biol.*, vol.20, pp.82-93, 2001.
- [147] D. Popovic, M. Radulovic, L. Schwirtlich, and N. Jaukovic, "Automatic vs hand-controlled walking of paraplegics," *Med. Eng. Phys.*, vol.25, pp.63-73, 2003.
- [148] D. Popovic, R. B. Stein, M. N. Oguztoreli, M. Lebedowska, and S. Jonic, "Optimal control of walking with functional electrical stimulation: A computer simulation study," *IEEE Trans. Rehab. Eng.*, vol.7, pp.69-79, Mar. 1999.

- [149] M. B. Popovic and D. Popovic, "Cloning biological synergies improves control of elbow neuroprostheses," *IEEE Eng. Med. Biol.*, vol.20, pp.74-81, 2001.
- [150] F. Previdi and E. Carpanzano, "Design of a gain scheduling controller for knee-joint angle control by using functional electrical stimulation," *IEEE Trans. Control Systems Technology*, vol.11, no .3, pp.310-323, 2003.
- [151] F. Previdi, T. Schauer, S. M. Savaresi, and K. J. Hunt, "Data-driven control design for neuroprostheses: A virtual reference feedback tuning (VRFT) approach," *IEEE Trans. Control Systems Technology*, vol.12, pp.176-182, 2004.
- [152] A. Prochazka and S. Yakovenko, "Locomotor control: from spring-like reactions of muscles to neural prediction," in *The Somatosensory System: Deciphering The Brain's Own Body Image*, Boca Raton, CRC Press, pp.141-181, 2001.
- [153] A. Prochazka, "Comparision of natural and artificial control of movement," *IEEE Trans. Biomed. Eng.*, vol.1, no.1, pp.7-17, 1993.
- [154] A. Prochazka, M. Gauthier, M. Wieler, and Z. Kanwell, "The bionic gloves: An electrical stimulator garment that provides controlled grasp and hand opening in quadriplegia," *Arch. Physical Med. and Rehab.*, vol.78, pp.1-7, 1997.
- [155] C. C. Raasch, F. E. Zajac, B. Ma, and W. S. Levine, "Muscle coordination of maximum-speed pedaling," *J. Biomech.*, vol.6, pp.595-602, 1997.
- [156] J. Riess and J. J. Abbas, "Adaptive neural network control of cyclic movements using functional neuromuscular stimulation," *IEEE Trans. Rehab. Eng.*, vol.8 no.1, pp.42-52, Mar. 2000.
- [157] J. Riess and J. J. Abbas, "Adaptive control of cyclic movement as muscle fatigue using functional neuromuscular stimulation," *IEEE Trans. Neural Syst. and Rehab. Eng.*, vol.9, no.3, pp.326-330, Sep. 2001.
- [158] R. Richardsona, M. Brownb, B. Bhaktac, and M. Levesleyd, "Impedance control for a pneumatic robot-based around pole-placement, joint space controllers," *Control Engineering Practice*, vol.13, pp.291-303, 2005.
- [159] L. Righetti and A. Ijspeert, "Programmable central pattern generators: an application to biped locomotion control," *IEEE Intern. Conf. on Robotics and Automation (ICRA06)*, Orlando, pp.1585-1590, May, 2006.

- [160] D. A. Rosenbaum, *Human Motor Control*, San Diego: Academic Press, 1991.
- [161] R. Riener, J. Quintern, and G. Schmid, "Biomechanical model of the human knee evaluated by neuromuscular stimulation," *J. Biomech.*, vol.29, no.9, pp.1157-1167, 1996.
- [162] R. Riener and T. Fuhr "Patient-driven control of FES-supported standing up: A simulation study," *IEEE Trans. Rehab. Eng.*, vol.6, no.2, pp.113-124, 1998.
- [163] R. Riener, M. Ferrarin, E. E. Pavan, and C. A. Frigo, "Patient-driven control of FES-supported standing up and sitting down: Experimental results," *IEEE Trans. Rehab. Eng.*, vol.8, pp.523-529, Dec. 2000.
- [164] W. L. C. Rutten, "Selective electrical interfaces with the nervous system," *Annu. Rev. Biomed. Eng.*, vol.4, pp.407-452, 2002.
- [165] I. A. Rybak, K. Stecina, N. A. Shevtsova, and D. A. McCrea, "Modelling spinal circuitry involved in locomotor pattern generation: insights from the effects of afferent stimulation," *J. Physiol. Lond.*, vol.577, no.2, pp.617-639, 2006.
- [166] I. A. Rybak, N. A. Shevtsova, M. Lafreniere-Roula, and D. A. McCrea, "Modelling spinal circuitry involved in locomotor pattern generation: insights from deletions during fictive locomotion," *J. Physiol. Lond.*, vol.577, no.2, pp.641-658, 2006.
- [167] L. R. Scheker, S. P. Chesher, and S. Ramirez, "Neuromuscular electrical stimulation and dynamic bracing as a treatment for upper-extremity spasticity in children with cerebral palsy," *J. Hand Surg. [Br.]* vol.24, pp.226-232, 1999.
- [168] G. Shue, P. E. Crago, H. J. Chizeck, "Muscle-joint models incorporating activation dynamics, moment-angle, and moment-velocity properties," *IEEE Trans. Biomed. Eng.*, vol.42, pp.212-223, 1995.
- [169] M. Simon, M. Sorensen, G. Cymbalyuk, R. Calabrese, and S. Deweerth, "Control of bursting properties in a silicon neuron CPG," *Neurocomputing*, pp.44-46, 2002.
- [170] J. E. Slotine, "Modular stability tools for distributed computation and control," *J. Adaptive Control and Signal Processing*, vo.17, no.6, pp.397-416, 2002.
- [171] A. M. Smith, "Does the cerebellum learn strategies for the optimal time-varying control of joint stiffness?" *Behavioral and Brain Science*, vol.19, in press, 1996.

- [172] M. Solomonow, E. Anguilar, E. Reisin, R. V. Baratta, R. Best, T. Coetzee, and R. D'Ambroisa, "Reciprocating gait orthosis powered with electrical muscle stimulation, I: Performance evaluation of 70 paraplegic patients," *Orthopedics*, vol.20, pp.315-324, 1997.
- [173] S. Srinivasan, R. E. Gander, and H. C. Wood, "A movement pattern generator model using artificial neural networks," *IEEE Trans. Biomed. Eng.*, vol.39, no.7, pp.716-722, Jul. 1992.
- [174] E. C. Sites and J. J. Abbas, "Sensitivity and versatility of an adaptive system for controlling cyclic movements using functional neuromuscular stimulation," *IEEE Trans. Biomed. Eng.*, vol.47, no.9, pp.1287-1292, Sep. 2000.
- [175] V. A. Straub, K. Staras, G. Kemenes, and P. R. Benjamin, "Endogenous and network properties of lymnaea feeding central pattern generator interneurons," *J. Neurophysiol.*, vol.88, pp.1569-1583, 2002.
- [176] A. Stefanovska, L. Vodovnik, N. Gros, S. Rebersek, and R. A. Janezic, "FES and spasticity," *IEEE Trans. Biomed. Eng.*, vol.36, no.7, 1989.
- [177] A. Stefanovska, S. Rebersek, T. Bajd, and L. Vodovnik, "Effects of electrical stimulation on spasticity. *Crit. Rev. Phys. Med. Rehab.* vol.3, pp.59-99, 1991.
- [178] P. S. G. Stein, J. C. Victor, E. C. Field, S. N. Currie, "Bilateral control of hindlimb scratching in the spinal turtle: Contralateral spinal circuitry contributes to the normal ipsilateral motor pattern of fictive rostral scratching," *J. Neurosci.* vol.15, pp.4343-4355, 1995.
- [179] S. Stroeve, "Impedance characteristics of a neuromusculoskeletal model of the human arm I: Posture control," *Biol. Cybern.*, vol.81, pp.475-494, 1999.
- [180] J. F. Stein, "Role of the cerebellum in the visual guidance of movement," *Nature*, vol.323, pp.217-221, 1986.
- [181] M. Sugisaka, K. Imamura, and K. Tokuda, "A new artificial life body: biological inspired dynamic bipedal humanoid robots," *Artif. Life and Robot*, vol.8, pp.1-4, 2004.
- [182] G. Taga, "A model of the neuro-musculo-skeletal system for human locomotion I, II," *Biol. Cybern.*, vol.73, pp.97-121, 1995.

- [183] K. Takahashi, N. Hoshimiya, H. Mastuki, and Y. Handa, "Externally powered implantable FES system," *Jap. J. Med. Electro. and Bio. Eng.*, vol.37, pp.43-51, 1999.
- [184] J. Tegner, J. H. Kotalleski, A. Lansner, and S. Grillner, "Low voltage activated calcium channels in the lamprey locomotor network: simulation and experiment," *J. Neurophysiol.*, vol.77, pp.1795-1812, 1997.
- [185] L. H. Ting, S. A. Kautz, D. A. Brown, and F. E. Zajac, "Contralateral movement and extensor force generation alter flexion phase muscle coordination in pedaling" *J. Neurophysiol.*, vol.83, pp.3351-3365, 2000.
- [186] R. Tomovic, D. Popovic, and R. B. Stein, "Nonanalytical methods for motor control," in press, World Science, Singapore, 1995.
- [187] F. Towhidkhah, R. E. Gander, and H. C. Wood, "Model predictive impedance control: A model for joint movement," *J. Mot. Behav.*, vol.29, no.3, pp.209-222, 1997.
- [188] K. Y. Tong and M. H. Granat, "Gait control system for functional electrical stimulation using neural networks," *Med. Biol. Eng. Comput.*, vol.37, no.1, pp.35-41, 1999.
- [189] T. Valency and M. Zacksenhouse, "Accuracy/robustness dilemma in impedance control," *J. Dyn. Syst. Meas. Control*, vol.125, pp.310-19, 2003.
- [190] W. Wang and J. E. Slotine, "On partial contraction analysis for coupled non-linear oscillators," *Biol. Cybern.*, vol.92, no.1, pp.38-53, 2004.
- [191] P. Wallen and S. Grillner, "N-methyl-D-aspartate receptor induced, TTX-resistant membrane potential oscillations in lamprey neurons active during locomotion," *Neurosci. Lett.*, vol.56, pp.87-93, 1985.
- [192] P. Wallen and S. Grillner, "N-methyl-D-aspartate receptor-induced inherent oscillatory activity in neurons active during locomotion in the lamprey," *J. Neurosci.*, vol.7, pp.2745-2755, 1987.
- [193] D. A. Winter, *Biomechanics and Motor Control of Human Movement*, 3rd edn., in press, Wisley, Singapore, 2005.
- [194] M. M. Williamson, "Neural control of rhythmic arm movements," *Neural Networks*, vol.11, pp.1379-1394, Apr. 1998.

- [195] M. M. Williamson, *Robot Arm Control Exploiting Natural Dynamics*, Ph.D Thesis, MIT, USA, 1999.
- [196] A. B. Wright "Derivation of equations of motion for a four-link robotic leg for walking vehicle," *J. Arkansas Academy of Science*, vol.53, pp.137-142, 1999.
- [197] S. Yakovenko, V. Gritsenko, A. Prochazka, "Contribution of stretch reflexes to locomotor control: a modeling study," *Biol. Cybern.*, vol.90, pp.146-155, 2004.
- [198] G. T. Yamaguchi and F. E. Zajac, "Restoring unassisted natural gait to paraplegics via functional neuromuscular stimulation: A computer simulation study," *IEEE Trans. Biomed. Eng.*, vol.37, no.9, pp.886-902, Sep. 1990.
- [199] K. Yoshida and R. Riso, "Peripheral nerve recording electrodes and techniques," in *Neuroprosthetics: Theory and Practice*, in press, World Science, pp.683-744, 2004.
- [200] K. Yoshida and K. Horch, "Closed-loop control of ankle position using muscle afferent feedback with functional neuromuscular stimulation," *IEEE Trans. Biomed. Eng.*, vol.43, no.2, pp.167-176, 1996.
- [201] F. E. Zajac, R. R. Neptune, and S. A. Kautz, "Biomechanics and muscle coordination of human walking Part I: Introduction to concepts, power transfer, dynamics and simulations," *Gait and Posture*, vol.16, pp.215-232, 2002.
- [202] F. E. Zajac, R. R. Neptune, and S. A. Kautz, "Biomechanics and muscle coordination of human walking Part II: Lessons from dynamical simulations and clinical implications," *Gait and Posture*, vol.17, pp.1-17, 2003.
- [203] G. I. Zahalak and S. P. Ma, "Muscle activation and contraction: Constitutive relations based directly on cross-bridge kinetics," *J. Biomech. Eng.*, vol.112, pp.52-62, 1990.
- [204] G. I. Zahalak, "An overview of muscle modeling," in *Neural Prostheses: Replacing Motor Function after Disease or Disability*, Oxford University Press, pp.17-57, 1992.
- [205] F. E. Zajac, "Muscle and tendon: properties, model, scaling, and application to biomechanics and motor control," *CRC Crit. Rev. Biomed. Eng.*, vol.17, pp.359-411, J. R. Bourne, Ed. Boca Raton, FL: CRC Press, 1989.

- [206] E. P. Zehr and J. Duysens, "Regulation of arm and leg movement during human locomotion," *The Neuroscientist*, vol.10, no.4, pp.347-361, 2004.
- [207] D. G. Zhang and K. Y. Zhu, "Simulation study of FES-assisted standing up with neural network control," *Proc. 26th Int. Conf. IEEE EMBC*, San Francisco, vol.7, pp.4877-4880, Sep. 2004.
- [208] D. G. Zhang and K. Y. Zhu, "Neural network control for leg rhythmic movements via functional electrical stimulation," *Proc. IEEE Int. Joint Conf. Neural Networks (IJCNN)*, Budapest, vol.2, pp.1245-1248, Jul. 2004.
- [209] D. G. Zhang, K. Y. Zhu, and H. Zheng, "Model the leg cycling movements with neural oscillator," *Proc. IEEE Int. Conf. Systems, Man and Cybernetics (SMC)*, Netherlands, vol.1, pp.740-744, Oct. 2004.
- [210] D. G. Zhang and W. T. Ang, "Tremor suppression of elbow joint via functional electrical stimulation: A simulation study," *IEEE Conference on Automation Science and Engineering (CASE)*, Shanghai, October, 2006.
- [211] D. G. Zhang, and K. Y. Zhu, "Modeling biological motor control for human locomotion with functional electrical stimulation," *Biol. Cybern.*, vol.96, pp.79-97, 2007.
- [212] D. G. Zhang, K. Y. Zhu, and L. Lan, "Analysis of neural oscillator for bio-inspired robot control," *IEEE Int. Conf. Robotics, Automation and Mechatronics (RAM)*, Bangkok, June, 2006.

Appendix

I. Three-segment Dynamics

Using Newton-Euler method, the inverse dynamics of the three-segment is represented as [17]:

$$\begin{bmatrix} T_1 \\ T_2 \\ T_3 \end{bmatrix} = \begin{bmatrix} M(\theta)_{11} & M(\theta)_{12} & M(\theta)_{13} \\ M(\theta)_{21} & M(\theta)_{22} & M(\theta)_{23} \\ M(\theta)_{31} & M(\theta)_{32} & M(\theta)_{33} \end{bmatrix} \begin{bmatrix} \ddot{\theta}_1 \\ \ddot{\theta}_2 \\ \ddot{\theta}_3 \end{bmatrix} + \begin{bmatrix} C(\theta, \dot{\theta})_1 \\ C(\theta, \dot{\theta})_2 \\ C(\theta, \dot{\theta})_3 \end{bmatrix} + \begin{bmatrix} G(\theta)_1 \\ G(\theta)_2 \\ G(\theta)_3 \end{bmatrix} + \begin{bmatrix} E(\theta)_1 \\ E(\theta)_2 \\ E(\theta)_3 \end{bmatrix} \quad (.1)$$

where $M(\theta)_{11} = m_1 d_1^2 + I_1 + m_2(l_1^2 + d_2^2 + 2l_1 d_2 \cos(\theta_2) + I_2 + m_3[l_1^2 + l_2^2 + d_3^2 + 2l_1 l_2 \cos(\theta_2) + 2l_1 d_3 \cos(\theta_2 + \theta_3) + 2l_2 d_3 \cos \theta_3] + I_3$

$M(\theta)_{12} = m_2 d_2^2 + l_1 d_2 \cos \theta_2 + I_2 + m_3[l_2^2 + d_3^2 + l_1 l_2 \cos \theta_2 + l_1 d_3 \cos(\theta_2 + \theta_3) + 2l_2 d_3 \cos \theta_3] + I_3$

$M(\theta)_{13} = m_3[d_3^2 + l_1 d_3 \cos(\theta_2 + \theta_3) + l_2 d_3 \cos \theta_3] + I_3$

$M(\theta)_{21} = m_2(d_2^2 + l_1 d_2 \cos \theta_2) + I_2 + m_3[l_2^2 + d_3^2 + l_1 l_2 \cos \theta_2 + l_1 d_3 \cos(\theta_2 + \theta_3)] + I_3$

$M(\theta)_{22} = m_2 d_2^2 + I_2 + m_3(l_2^2 + d_3^2 + l_2 d_3 \cos \theta_3) + I_3$

$M(\theta)_{23} = m_3(d_3^2 + l_2 d_3 \cos \theta_3) + I_3$

$M(\theta)_{31} = m_3[d_3^2 + l_1 d_3 \cos(\theta_2 + \theta_3) + l_2 d_3 \cos \theta_3] + I_3$

$M(\theta)_{32} = m_3(d_3^2 + l_2 d_3 \cos \theta_3) + I_3$

$M(\theta)_{33} = m_3 d_3^2 + I_3$

$C(\theta, \dot{\theta})_1 = -[(m_2 l_1 d_2 + m_3 l_1 l_2) \sin \theta_2 + m_3 l_1 d_3 \sin(\theta_2 + \theta_3)](\dot{\theta}_2^2 - 2\dot{\theta}_1 \dot{\theta}_2) - [m_3 l_1 d_3 \sin(\theta_2 + \theta_3) + m_3 l_2 d_3 \sin \theta_3](\dot{\theta}_3^2 - 2\dot{\theta}_1 \dot{\theta}_3 + 2\dot{\theta}_2 \dot{\theta}_3)$

$C(\theta, \dot{\theta})_2 = [(m_3 l_1 l_2 + m_2 d_2 l_1) \sin \theta_2 + m_3 d_3 l_1 \sin(\theta_2 + \theta_3)]\dot{\theta}_1^2 - m_3 d_3 l_2 \sin \theta_3(\dot{\theta}_3^2 - 2\dot{\theta}_1 \dot{\theta}_3 + 2\dot{\theta}_2 \dot{\theta}_3)$

$$\begin{aligned}
C(\theta, \dot{\theta})_3 &= [m_3 l_1 d_3 \sin(\theta_2 + \theta_3) + m_3 l_2 d_3 \sin \theta_3] \dot{\theta}_1^2 + m_3 l_2 d_3 \sin \theta_3 (\dot{\theta}_2^2 - 2\dot{\theta}_1 \dot{\theta}_2) \\
G(\theta)_1 &= m_1 g d_1 \cos \theta_1 + m_2 g [l_1 \sin \theta_1 + d_2 \cos(\theta_1 + \theta_2)] + m_3 g [l_1 \sin \theta_1 + l_2 \sin(\theta_1 - \theta_2) + \\
&\quad d_3 \sin(\theta_1 - \theta_2 - \theta_3)] \\
G(\theta)_2 &= m_2 g d_2 \sin(\theta_1 - \theta_2) + m_3 g [l_2 \sin(a_1 - a_2) + d_3 \sin(\theta_1 - \theta_2 - \theta_3)] \\
G(\theta)_3 &= m_3 g d_3 \sin(\theta_1 - \theta_2 - \theta_3)
\end{aligned}$$

Some parameters should be noted as follows. Subscript $i=1, 2, 3$ means hip, knee and ankle joint of the lower limb during walking (or cycling movement). θ_i is the angle of joint i . l_i is the length of segment i . d_i is the distance from the proximal joint of the segment i to its center of gravity (COG). m_i is the mass of segment i . I_i is the moment of inertia of segment i . T_i is moment of joint i . g is the gravitational constant. $E(\theta)_i$ is the external torque acted on the joint i , which includes the disturbance torque and ground (pedal) torque applied to the foot in this work.

II. Poincaré Map:

Consider a dynamical system of the form as

$$\dot{X} = F(X), X \in \mathbb{R}^m \quad (.2)$$

having a periodic solution $X(t) \subset \mathbb{R}^m$ with period $T > 0$. Such a periodic solution is called a limit cycle. It is denoted by $\gamma \in \mathbb{R}^m$. Let $P \subset \mathbb{R}^m$ be an $m - 1$ dimensional local cross section, namely a hyperplane or a manifold intersecting γ transversally as well as orbits close to γ (see Figure 1). Without loss of generality, we may assume that γ intersects P in a unique point p . Such a set P is called the *Poincaré* section for the dynamical system. Let $U \subset P$ be a neighborhood of p , any orbit $\Phi_t(X)$ starting from a point $\Phi_0(X) = X \in U$ returns to P in a finite time $T(X) \approx T$. Then the *Poincaré* mappings $P : U \rightarrow P$ is defined for all points X from U as $P(X) = \Phi_T(X)$. The map can transform the m dimensional dynamics into the $m - 1$ discrete system by the hyperplane P .

Figure 1: The *Poincaré* mapping $U \rightarrow P$.