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CPG-based circuitry for controlling musculoskeletal model of human locomotor system

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***Abstract*—In this paper, a new neuro-musculoskeletal simulator of human locomotor system is presented. This simulator is dedicated to reproduce healthy or altered walking gaits. It contains three joints per leg (hip, knee, ankle) controlled by twelve human muscle models activated by six specific models of central pattern generator (CPG). The CPG consists of three layers and four types of neurons and controls human leg joints. The CPGs are able to generate variable rhythmic signals by changing their intrinsic neural parameters which are controlled by descending signals from mesencephalic locomotor region (MLR), while output signals of motoneurons of CPGs control muscle models. Simulation results in Matlab show that it is possible to generate different stable walking gaits by changing intrinsic parameters of CPGs. According to these changes, the simulator can exhibit coherent or incoherent coordination between the two legs and consequently, stable or unstable walking gaits starting from the double support phase. Results show that this simulator will allow to reproduce walking gaits altered by basal ganglia decision-making system affected by Parkinson's disease.**

Keywords—central pattern generator; human walking; musculoskeletal model; Parkinson's disease

I. INTRODUCTION

Parkinson's disease (PD) is a progressive neuro-degenerative illness with symptoms like tremor, muscular rigidity, and slow, imprecise movement mainly affecting elderly individuals over 60 years old [1]. It is the second most common neurodegenerative disease of adult onset [2]. PD affects control of movements, especially upper and lower limbs, causes tremor and rigidity [3].

A global model, able to simulate the main parts affected by PD should consist of three major levels:

1) a computational model of basal ganglia (BG) whose output mediates the second level;

2) a model of spinal structures composed of several central pattern generators (CPG) projecting to the muscles through motoneurons;

3) a musculoskeletal simulation of human lower limbs that will execute locomotor movements including physical effects of articular multi-bodies systems interacting with the ground.

There are many works that model and simulate neural structures affected by PD (first level). Most of them are limited to model of BG and relations between its nuclei. These models result in firing rates of neurons or other intrinsic parameters of system as their aim is to identify the source of abnormal oscillations in BG. Therefore, despite interesting ways of modelling BG and PD, these works are not sufficiently connected to consequences of PD on the human movements and especially on the walk.

Indeed, human movements governed by lower structures that receive signals from BG are also perturbed by PD and this impacts the daily life of affected people. Some authors propose computational models of altered walking gait [4] or altered grip gesture [5], which contribute to all three levels on list above. Nevertheless, these works does not explicitly model or simulate lower neural structures that receive signals from BG. These structures are located in spinal cord (second level) and are called central pattern generators. They are involved in rhythmic movements and control of muscle activities [6].

To simulate impact of PD disorders on human walking gait, this paper focuses on the second and third level. The contribution of this work is to propose a CPG-based circuitry, connected to a musculoskeletal model in a closed loop with proprioceptive and exteroceptive feedback. By changing only one intrinsic neural parameter of CPG, this model allows to exhibit different stable gaits and to reproduce altered human walking gait by receiving signals from upper control centers (such as BG) for equilibrium, speed change, or parkinsonian symptoms such as freezing of gait (FoG) [7].

Section II briefly tells about state of the art and methods used in fields of modelling CPGs and musculo-skeleton system. Section III describes the model. Section IV presents obtained simulation results. Finally, Section V concludes this work, with some remarks and future work directions.

II. MATERIALS & METHODS

A. Biological central pattern generator for locomotion

Basal ganglia is connected to central pattern generators through mesencephalic locomotor region (MLR), reticular formation, and extrapyramidal tracts in brainstem [8].

CPG is a set of inter and motoneurons located in the spinal cord. More than a hundred years of investigations has led to the conclusion that rhythmic locomotion activities are largely

controlled by network of spinal neurons [6]. They can generate rhythmic activity by themselves. Descending signals from the higher centers are optional, affect the shape of generated patterns, and contribute to the inter- and intra-CPG synchronization.

Current usage research, modelling, and application of CPG vary from investigation of purpose and regimes of specific groups of neurons in different segments of spinal cord [8, 9] through modelling neural networks resembling CPG [10, 11, 12] to synthesis of control units in robotics that have the same behavior patterns of biological CPGs [13, 14].

B. Model of CPG

The model of CPG that was used in this work was proposed and used as a controller for the walk of a humanoid robot [13]. It is based on work of Rybak et al. [10] for a two-level CPG that separates the timing and activation of the locomotion cycle. This model is rather mathematical; however, it is supported by two neurophysiological studies and combines their propositions in multi-layered multi-pattern CPG model.

CPG model is controlled from high-level system (e.g. MLR) that varies the frequency of generated patterns, phase deletion, and clamping of controlling signal. The CPG architecture is composed of three layers (Fig. 1): rhythm-generation neurons (RG); pattern-formation neurons (PF); and motoneurons (MN). Additionally, the model includes feedback sensory neurons (SN) that shape the activity of the CPG neurons.

A neural model of RG neurons was proposed by Rowat and Selverston [12]. It has self-rhythmic generation ability; its oscillation depends on two membrane conductivities for fast and slow current. Depending on these and others cell parameters, RG neurons can generate different patterns: quiescence, almost an oscillator, endogenous oscillator, plateau, depolarization, and hyperpolarization.

Patterns generated by RG layer are shaped by PF neurons. They also chose the domination rhythm for a joint (flexion/extension). They are capable of rhythm deletion of RG layer without resetting its phase. This means, PF neurons can deactivate motoneurons while RG continue to oscillate. The activation function of PF neurons is sigmoid, whose main parameters are amplitude and saturation.

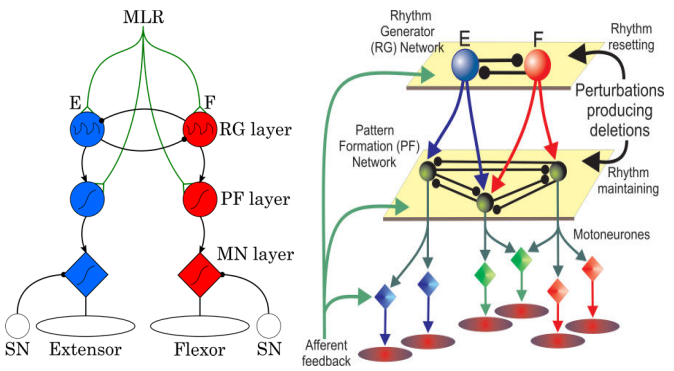

Fig. 1. Left: model of one joint CPG proposed by Rybak et al. [10]. Right: proposed controller with three layers: rhythm generator (RG), pattern-formation (PF), and motoneuron (MN) layers.

Motoneurons directly control the muscles with input from PF layer and proprioceptive sensory neurons. Latter measure angle of joint and excite the corresponding motoneuron thus implementing articular reflex. Exteroceptive SN measure foot/ground contact force to excite ankle joint so it steps on full foot. MN and SN also use sigmoid activation function. For detailed mathematical models of cells, refer to [13].

C. Musculoskeletal model

This work uses a modified version of the musculoskeletal simulator Gait2de proposed by Ton van den Bogert [15]. This realistic dynamic model simulates muscle activities (based on Hill model [16]) and their action on skeleton to produce movements in the sagittal plane taking into account physical phenomena (ground friction, forces and dynamics of limbs, etc.). It has nine kinematic degrees of freedom, seven body segments, eight muscles per leg, and its dynamics and outputs are twice differentiable with respect to all inputs. This model is implemented as Matlab MEX function and it takes ~0.03 ms to compute.

Body segments of model are trunk, thigh, shank and foot in each leg. Each of them has the following parameters: mass, length, center of mass, moment of inertia of a human male with body mass 75 kg and body height 1.8 m.

III. Neuro-Musculoskeletal Model

The musculoskeletal model of the human locomotor system proposed in [15] is modified in order to control the muscles by the signals generated by a circuitry based on our model of CPGs.

A. Modifying the musculoskeletal model

As a preliminary work, the musculoskeletal system's part models six muscles per leg, avoiding the Hamstrings and Rectus muscles that affect two joints at once for easier and more understandable control (Fig 2).

Thus, one CPG control an antagonistic pair of muscles, and CPGs are interconnected together to coordinate the limbs. Forces sensors are implemented on soles to return pressure force on heels and toes to measure the center of pressure. A virtual elastic attached to the top of trunk and able to slide horizontally is added (dotted line on Fig. 2, 4) as lifting support harness like a disabled person. It is implemented as external force F through optional input of the model and is calculated as follows:

$$F = -k \times (d - L_0) - F_p/2,$$

where k is stiffness of elastic, d distance to attaching point, L_0 resting length of elastic, and F_p is force from last time frame for simulation of elastic's energy loss.

B. Connecting the CPGs to the muscles

The full scheme of our neural circuitry consists of MLR projection to CPG (Fig. 1, 3) as i_{inj}, σ_s, α_{MLR}, and θ_{MLR} parameters. A pulse of i_{inj} makes CPG to start oscillating and could alter rate of RG [13] if being a sinusoid for example; σ_s is actually a cell parameter, but it can be affected from upper structures (e.g. with neuromodulator or plasticity mechanism); α_{MLR} and θ_{MLR} control PF neurons, their coupling to MN and balance between flexion and extension.

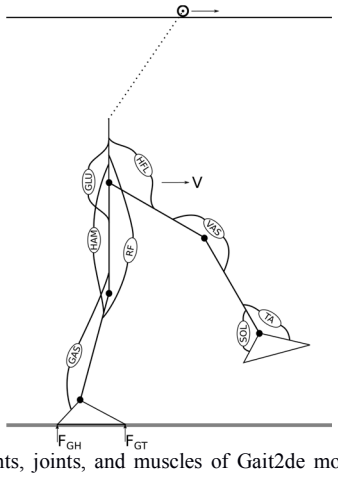

Fig. 2. Body segments, joints, and muscles of Gait2de model. Muscles are drawn for one leg. F_{GH} and F_{GT} are ground reaction forces on heel and toe, V is velocity of pelvis.

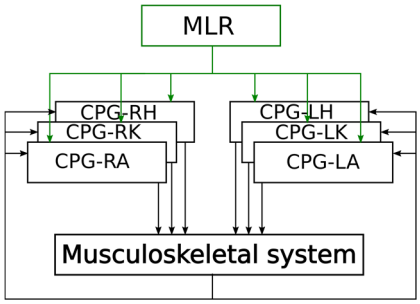

Fig. 3. General scheme of neuro-musculoskeletal simulator. R and L stand for right and left leg; H, K, and A stand for hip, knee, and ankle joint.

Outputs of RG half-centers are connected to PF neurons, which in case of hip simply transform input value range to [0;1], as flexion/extension domination and rhythm deletion through changing α_{MLR} and θ_{MLR} aren't applied. PF outputs are connected to MN which excitate muscles that rotate hip joint.

The input to musculoskeletal system are neural excitations for each muscle, along with initial state of the model and optional external forces and moments applied to body parts and joints. Each MN provide neural excitations for corresponding muscle [15] that control human locomotor model. Iliopsoas, Vasti, and TibialisAnt are flexion muscles that turn joints counterclockwise (positive direction); Glutei, Gastroc, and Soleus are extension muscles that turn joints clockwise (negative direction).

Additionally, output from MN should be limited to [0; 1] as model does not apply such itself. To close the control loop, CPG contains muscle sensory neurons (SN) for articular reflex that transform angle of corresponding joint into inhibitory influence on each motoneuron. In addition, two ground SN that react to force at contact points under heel and toe and excitate MN to help ankle phasing.

IV. Results

A. Walking with constant speed

A distinctive feature of CPG is its ability to produce rhythmic patterns without input like shown on Fig. 4. Figures 5, 6, and 7 show CPG activity, muscle excitations, and joint angles for hip, knee, and ankle respectively.

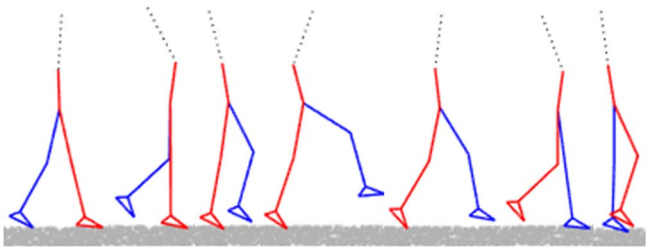

Fig. 4. Several consecutive frames of Gait2de model simulation, time between frames is 0.1 s.

All joints follow the same connection scheme as on Fig. 1, except that knee CPG's PF neurons have additional connection (excitatory for flexor and inhibitory for extensor) from hip's flexor SN that corrects knee phase; and ankle CPG's PF neurons have two ground sensory neurons (GSN), that reacts to toe ground reaction is connected to flexor PF, heel GSN is connected to extensor PF.

Resulting joint angles are qualitatively similar to human walking cycle [17, 18], especially hip and knee joints that are similar to those in [19], where articular angles of the human hip, knee, and ankle are compared to those with a rigid and non-rigid human foot soles.

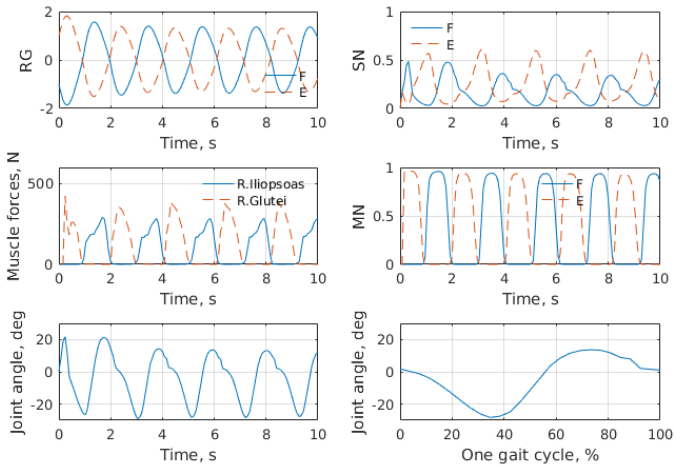

Fig. 5. Right hip CPG with constant frequency. F and E for flexion and extension RG half-center, sensory neuron, and motoneuron, respectively.

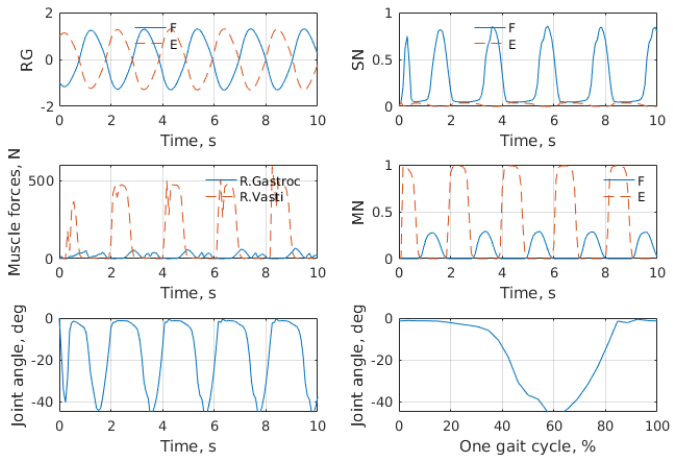

Fig. 6. Right knee CPG. F and E for flexion and extension RG half-center, sensory neuron, and motoneuron, respectively.

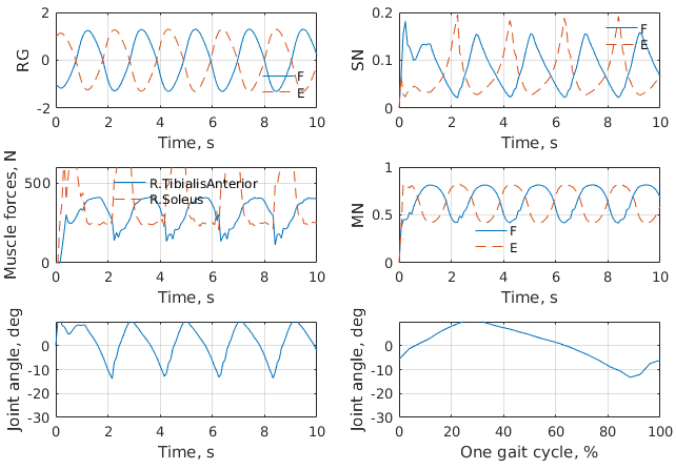

Fig. 7. Right ankle CPG. F and E for flexion and extension RG half-center, sensory neuron, and motoneuron, respectively.

Walking simulation starts from double support phase. Transient phase can be observed at the beginning of the walk on the muscle activities. Supporting elastic slightly contributes to oscillations of trunk like a real harness.

B. *Walking with variable speed*

Frequency of RG is controlled by neural parameter σ_s which acts like a frequency gain in a rhythmic activity. Fig. 8 shows variable sagittal velocity of pelvis of biped (Fig. 2) and its moving average (sliding window width is 8 seconds) reacting to manual change of σ_s as square signal. Dynamical changes of σ_s is equivalent of neural plasticity effects. Indeed, in future work, σ_s can be modified by a learning or adaptive law that models a homeostatic effect.

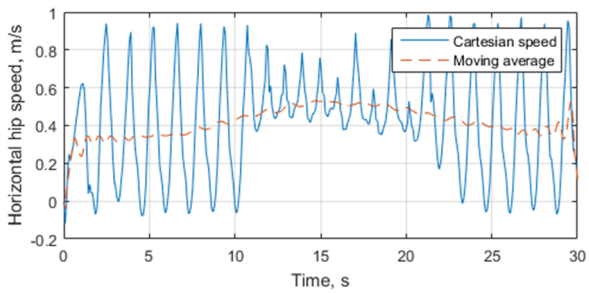

Fig. 8. Horizontal hip speed and its moving average. σ_s starts at 30, changes to 50 at 10 seconds, and back to 30 at 20 s.

After change of σ_s parameter, model needs about 3 seconds to stabilize its speed showing the global stability of the gait.

V. Conclusion

This paper presents a new neuro-musculoskeletal model based on a circuitry of six central pattern generators controlling twelve muscles of the two legs of human locomotor system. Each CPG consists of three layers and four types of neurons. The circuitry is able to generate rhythmic signals controlling the muscles and creating stable walking gaits that can be changed by variation of intrinsic neural parameters. These variations can be controlled by signals coming from an upper level circuitry. Following this way we assume that the neuro- musculoskeletal simulator presented here will be able to simulate impacts of PD disorders on human walking like observed in the medical studies. Though, further work will be aimed at taking into account the Hamstrings and Rectus muscles, simulation of altered walking gaits due to PD, like FoG, based on patients' data, and at developing a model of nervous links between Basal Ganglia region and this simulator.

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