A Neural Network with Central Pattern Generators Entrained by Sensory Feedback Controls Walking of a Bipedal Model

Wei Li(✉), Nicholas S. Szczecinski, Alexander J. Hunt, and Roger D. Quinn

Department of Mechanical and Aerospace Engineering, Case Western Reserve University, Cleveland, OH 44106-7222, USA
wxl155@case.edu

Abstract. A neuromechanical simulation of a planar, bipedal walking robot has been developed. It is constructed as a simplified musculoskeletal system to mimic the biomechanics of the human lower body. The controller consists of a dynamic neural network with central pattern generators (CPGs) entrained by force and movement sensory feedback to generate appropriate muscle forces for walking. The CPG model is a two-level architecture, which consists of separate rhythm generator (RG) and pattern formation (PF) networks. The presented planar biped model walks stably in the sagittal plane without inertial sensors or a centralized posture controller or a "baby walker" to help overcome gravity. Its gait is similar to humans' with a walking speed of 1.2 m/s. The model walks over small obstacles (5 % of the leg length) and up and down 5° slopes without any additional higher level control actions.

Keywords: Biologically inspired · Central pattern generator · Sensory feedback · Bipedal walking

1 Introduction

It is generally accepted that basic rhythmic motor signals driving walking and other forms of locomotion in various animals are generated centrally by the neural networks in the spinal cord (reviewed by [1–3]). These neural networks, capable of producing coordinated and rhythmic locomotor activity without any input from sensory afferents or from higher control centers are referred to as central pattern generators (CPGs). Cats with transection of spinal cord and removal of major afferents can still produce rhythmic locomotor patterns [4, 5]. Despite lacking clear evidence of CPGs in humans, observations in patients with spinal cord injury provide some support [6]. Sensory input also plays an important role in locomotion. Locomotion is the result of dynamic interactions between the central nervous system, body biomechanics, and the external environment [7]. As a closely coupled neural control system, sensory feedback provides the information about the status of the biomechanics and the relationship between the body and the external environment to make locomotion adaptive to the real environment. Sensory input could reinforce CPG activities, provide signals to ensure correct motor output for all body parts, and entrain the rhythmic pattern to facilitate phase transition when a

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proper body posture is achieved [8, 9]. There is strong evidence [10, 11] to suggest that sensory feedback affects motoneuron activity through common networks, rather than directly through reflex pathways acting on specific motoneurons. It implies sensory feedback and CPGs are highly integrated networks in the spinal cord.

Several 2D bipedal walking models have been previously developed with controllers containing CPGs and reflexes. Some simulation models [12, 13] had over-simplified multi-link structures driven by motors at joints, which lack the viscoelasticity of musculoskeletal structures. This lack provides poor mechanical properties that may reduce stability. Some models [14, 15] adopt more realistic musculoskeletal structures, but their CPG activities are not affected by sensory feedback. Instead, sensory afferents are connected directly with motoneurons. This is contradictory to findings that sensory feedback could strongly affect CPG activities [11, 12]. Geyer et al. [16] produced realistic human walking and muscle activities in a 2D model. They claim that their model only relies on muscle reflexes without CPGs or any other neural networks. It is realized by two sets (swing phase and stance phase) of coupled equations for every muscle to produce human walking and muscle activities. However, these equations could be viewed as abstract mathematical expressions of neural networks controlling muscle activities and the transition between two phases works like a finite-state machine. More recently, CPGs were added to a similar muscle-reflex model to control muscles actuating the hip joint [17]. Both of these works rely on precisely tracking muscle and other biomechanical parameters and provide limited insight into how the central nervous system is structured in humans. CPGs have also been applied to physical bipedal robots [18, 19], but usually these CPGs are abstract mathematical oscillators tuned to generate desirable trajectories of joints angles or torques, and the phases of the oscillators could simply be reset by reflexes. Klein and Lewis [20] claimed that their robot is the first that fully models human walking in a biologically accurate manner. But it has to rely on a baby walker-like frame to prevent it from falling down in the sagittal plane. Its knees remain bent distinctly before the foot touches the ground and its heels are always off the ground. It looks like it is pushing a heavy box, fundamentally different from a human's normal gait.

In this paper we present a biologically rooted neuromechanical simulation model of a planar, bipedal walker. It has a musculoskeletal structure and is controlled by a two-level CPG neural network incorporating biological reflexes via sensory afferents. The model is developed in Animatlab, which can model biomechanical structures and biologically realistic neural networks, and simulate their coupled dynamics [21]. We demonstrate that natural-looking, stable walking can be achieved by this relatively simple but biologically plausible controller combined with a human like morphological structure.

2 Structure of the Model

We based the biomechanical structure of our model on the human lower body, which has been shaped and selected by millions of years' evolution, and is energetically close to optimal [22]. The planar skeleton is made up of rigid bodies, and is actuated by

antagonistic pairs of linear Hill muscles. The morphology structure employed significantly reduces the control effort with its inherent self-stability and provides a basis for comparison to the real human gait. The simulated biped (Fig. 1) is modeled as a pelvis supported by two legs. Each leg has 3 segments (thigh, shank and foot), which are connected by 3 hinge joints (hip, knee and ankle) and actuated by 6 muscles. The foot is composed of two parts connected by one hinge joint, spanned by a passive spring with damping (spring constant = 16 kN/m, damping coefficient = 20 N·m/s). This makes the foot flexible, which is the basis of the toe-off motion. The foot has a compliance of 10 μm/N to mimic the soft tissue of a human foot. The mass and length ratio of different segments (except the foot length) are based on human data [23]. The total mass of the model is 50 kg and its height is approximately 1 m (leg length is 0.84 m). The mass of the pelvis, thigh, shank and foot are 25 kg, 8 kg, 4 kg and 0.5 kg. The lengths of the thigh, shank and foot are 0.42 m, 0.42 m and 0.24 m.

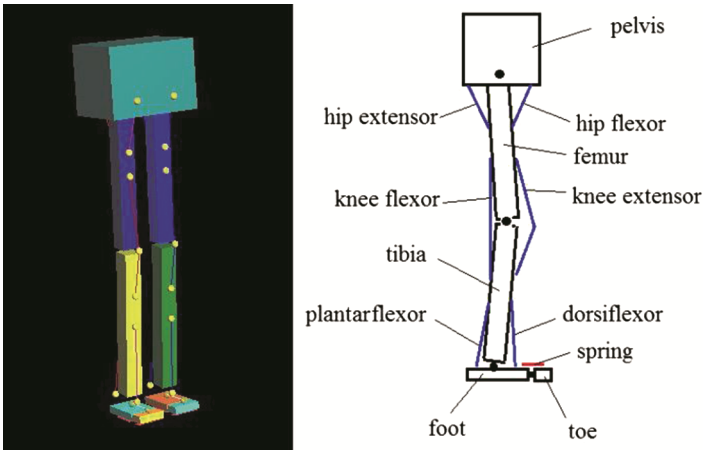

Fig. 1. Left: biomechancal model in Animatlab. Right: the muscle and skeleton system of the simulated biped.

3 Neural Control System

3.1 Neural Model

The neurons are leaky "integrate-and-fire" models. Each neuron is modeled as a "leaky integrator" of currents it receives, including synaptic current and injected current:

$$C_m \frac{dv(t)}{dt} = I_{leak}(t) + I_{syn}(t) + I_{inj}(t) \tag{1}$$

where $v(t)$ is the membrane potential, C_m is the membrane capacitance, $I_{leak}(t)$ is negative current due to the passive leak of the membrane, $I_{syn}(t)$ is the current from total synaptic

input to the neuron, $I_{inj}(t)$ is the current externally injected into the neuron. The leak current is:

$$I_{leak}(t) = g_{leak} \cdot \left(E_{reset} - v(t)\right) \tag{2}$$

where g_{leak} is the leak conductance, E_{reset} is the resting potential. When the membrane potential reaches the threshold value the neuron is said to fire a spike, and is reset to E_{rest}.

Transmitter-activated ion channels are modeled with a time-dependent postsynaptic conductance $g_{syn}(t)$. The current that passes channels $I_{syn}(t)$ depends on the reversal potential E_{syn} of the synapse and the postsynaptic neuron membrane potential $v(t)$:

$$I_{syn}(t) = g_{syn}(t) \cdot \left(E_{syn} - v(t)\right) \tag{3}$$

If $E_{syn} < E_{rest}$, the synapse is inhibitory and it induces negative inhibitory postsynaptic current. If $E_{syn} > E_{rest}$, the synapse is excitatory and it induces positive excitatory postsynaptic current. The postsynaptic conductance $g_{syn}(t)$ increases to a value with a time delay to the arrival of the presynaptic spike and then decays exponentially to zero:

$$g_{syn}(t) = g_{\max} \cdot e^{-\frac{t - t_0 - \delta t_{delay}}{\tau_{syn}}}, \; t \geq t_0 + \delta t_{delay} \tag{4}$$

where $g_{\max}$ is the initial amplitude of the postsynaptic conductance increase, t_0 denotes the arrival time of a presynaptic spike, δt_{delay} is the time delay between the presynaptic spike and the postsynaptic response, and τ_{syn} is the time constant of the decay rate.

3.2 Half-Center Oscillator

A basic neural pattern generator is a half-center neural oscillator composed of two reciprocally inhibited neurons. When one neuron spikes, it inhibits the other. Due to the facilitation of the synapse [24] the repeated spiking of the presynaptic neuron gradually hampers the synaptic transmission and reduces the postsynaptic conductance so the amplitude of the postsynaptic current gradually declines. The postsynaptic neuron would spike again as soon as the total current input recovers to the threshold value. When it starts to spike it would inhibit the other neuron in the same way.

3.3 CPG

Rybak et al. [25] proposed a two-level CPG architecture composed of a central rhythm generator (RG) network controlling several coupled unit pattern formation (UPF) modules in the pattern formation (PF) level. Sensory afferents could access and affect RG and PF networks separately. In this model the CPG network adopts a similar architecture as shown in Fig. 2. The RG is a half-center oscillator that generates the basic periodic stance signal for both legs. The PF network contains half-center oscillators as

UPF modules and each joint of each leg (hip, knee, and ankle) has one. Each half-center in the UPF module is connected to the corresponding extensor and flexor motoneurons to drive the extensor and flexor muscles for each joint. All half-center oscillators consist of two reciprocally inhibited "integrate-and-fire" neurons as described previously.

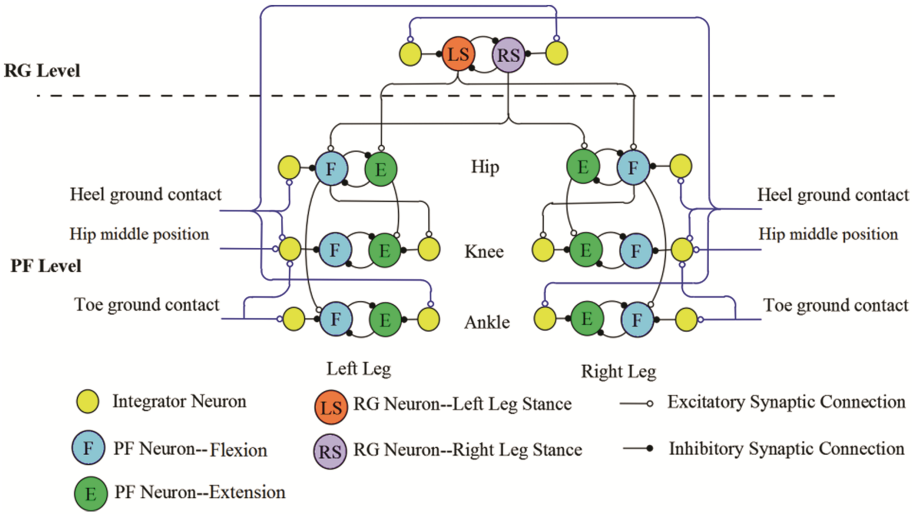

Fig. 2. The architecture of the CPG network. Black lines are synaptic connections between CPG neurons. Blue lines are synaptic connections from sensory neurons. (Color figure online)

Each neuron in the RG has an excitatory connection with a hip extension neuron and the other leg's hip flexion neuron. It encourages the legs to step in antiphase. At the PF level the hip flexion neuron has an excitatory connection with the ankle dorsiflexion neuron and an inhibitory connection with knee extension neuron. The hip extension neuron has an excitatory connection with the knee extension neuron. This controller uses a layer of interneurons to combine and filter afferent inputs from different receptors. These interneurons have inhibitory connections to the interneurons in the RG and PF networks.

3.4 Sensory Afferents

It is generally agreed that sensory input affects CPG timing directly since stimulation of corresponding afferents entrains or resets the locomotor rhythm [26]. In legged animals, there are three main categories of afferents that satisfy this requirement; group I muscle afferents in extensor muscles, cutaneous afferents in feet, and muscle afferents around the hip joint signaling its position. The first two largely depend on leg loading information, and the last is based on movement.

1. Ground Contact Afferents

Studies show that increasing the load on an infants' feet while they walk with support significantly increases the duration of the stance phase. It suggests that load receptor reflex activity can strongly modify and regulate gait timing and resides within the spinal circuits [27]. In our model, separate force sensors located on the plantar surface of the heels and toes separately detect and measure the ground contact force. As shown in Fig. 2, heel ground contact sensors excite afferent neurons which make inhibitory connections to the hip flexion, knee flexion, and ankle plantarflexion neurons at the PF level. They also apply inhibition to the other leg's stance neuron in the RG. Toe ground contact sensors inhibit the knee flexion and ankle dorsiflexion neurons at the PF level.

2. Hip Middle Position Afferents

During normal walking the knee extends in swing before the foot touches the ground. The knee reaches its peak flexion between 25 % and 40 % of the swing phase before extending. As shown in Fig. 2 in our model the hip middle position afferent is connected to the knee flexion neuron at the PF level and inhibits it when hip movement passes the predetermined hip angle during hip flexion.

4 Experiment and Results

In software simulation experiments, we confined the movement of the body to the sagittal plane, but it has no gravitational or pitching support, so it can fall forward or backward.

4.1 Normal Walking

The model walks at a moderate speed (1.2 m/s) on a rigid surface. Figure 3(a) shows a frame by frame comparison to that of a human. It is interesting to observe that they appear similar at corresponding phases of the cycle. Figure 3(b) compares hip, knee, and ankle angles between the model and human data during one gait cycle. The model's motion matches a human's in several aspects. The range of motion and general shapes of hip, knee and ankle angle curves are similar to a human's, although the model's knee motion lacks initial flexion at early stance phase, and the ankle dorsiflexion is insufficient. The knee reaches maximum flexion at about 75 % of the cycle and begins to extend before the hip reaches the anterior extreme position to prepare for ground contact. The transition from stance to swing is at about 60 % of the cycle, when the hip and knee begins to flex, and ankle reaches maximum plantarflexion. Figure 3(a) also compares the motion of the model's foot with a human's foot at early, middle and late stance phases.

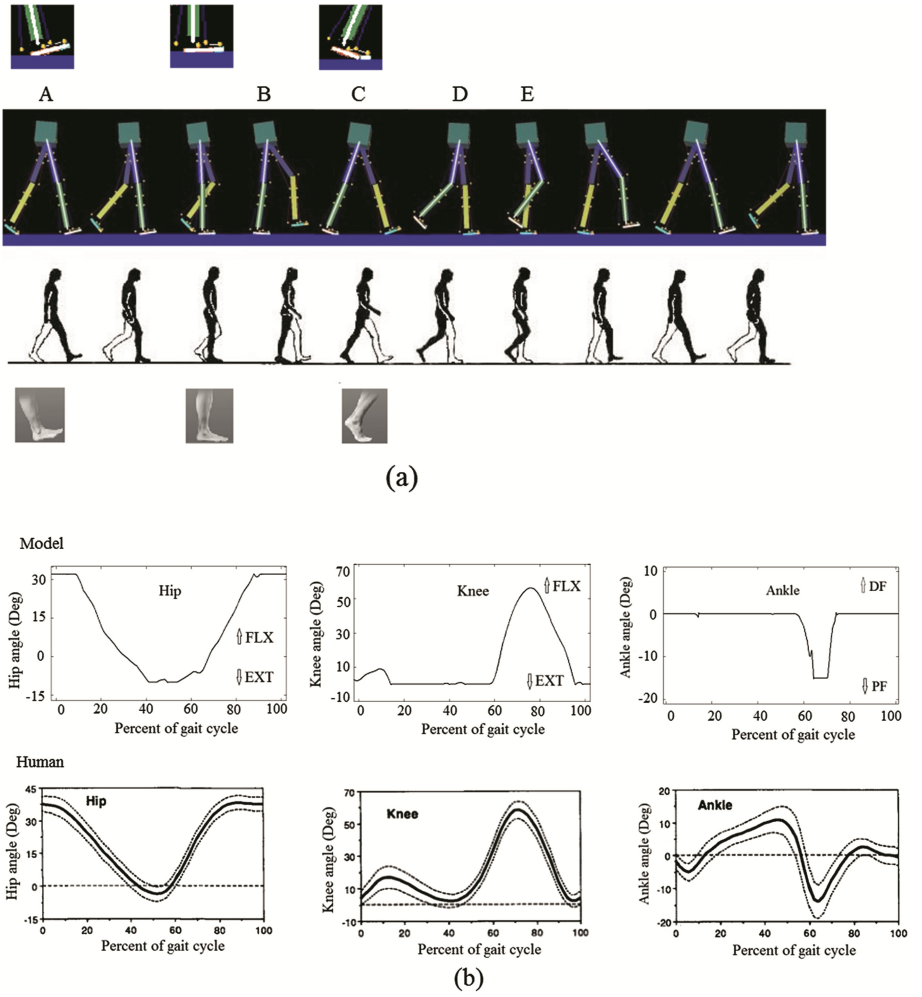

Fig. 3. Comparison of gait between the model and human. (a) Series of frames of the model and human at the same points in the gait cycle. (b) Model's hip, knee, and ankle angles compared with human's in the sagittal plane [28].

4.2 Interaction Between Sensory Feedback and CPGs

When the right leg enters stance (frame A in Fig. 3(a)) the right heel load sensor causes the heel ground contact sensor neuron to spike. This triggers the right leg stance RG neuron, causing right leg hip and knee UPF extension neurons to spike and initiate stance (time A in Fig. 4(c)). At time B in Fig. 3(a) the right heel just leaves ground and right heel ground contact sensor neuron stops spiking (time B in Fig. 4(a)). The right ankle UPF plantarflexion neuron is released from inhibition and begins to spike, causing right ankle plantarflexion (time B in Fig. 4(c)). The double support phase can be seen just following time C in Fig. 3(a). At this point, both the right toe and the left heel are in

contact with the ground. The left heel ground contact sensor neuron begins to spike (time C in Fig. 4(b)) and inhibit the right leg stance RG neuron (time C in Fig. 4(c)). The left leg enters stance and begins to take load from the right leg. Loading of the left leg triggers right leg swing and further encourages stance in the left leg. At time D in Fig. 3(a) the right foot toe part leaves the ground. Right toe sensor neuron stops spiking (time D in Fig. 4(a)), releasing right knee UPF flexion neuron and right ankle UPF dorsiflexion neuron from inhibition (time D in Fig. 4(c)). Right knee further flexes and right ankle goes into dorsiflexion. Toward the end of the right leg's swing phase (time E in Fig. 3(a)) the right hip middle position sensor neuron spikes (time E in Fig. 4(a)), inhibiting the right knee UPF flexion neuron from spiking (time E in Fig. 4(c)). This causes the knee to extend and prepare for ground contact.

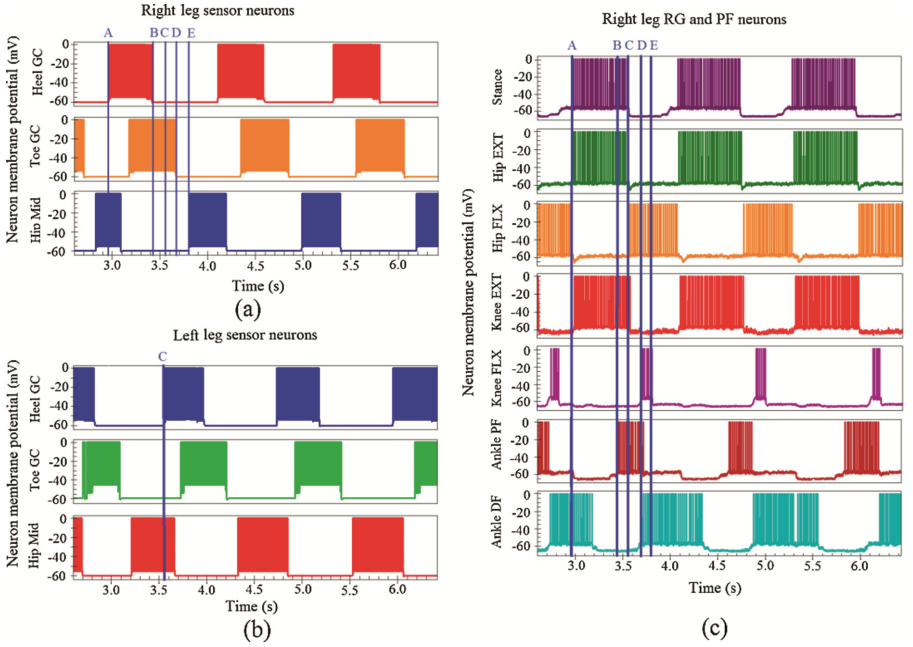

Fig. 4. Neuron membrane potentials in the gait cycle. (a) Right leg sensor neuron membrane potentials. GC: ground contact, Mid: middle position. (b) Left leg sensor neuron membrane potentials. (c) Right leg RG and PF neuron membrane potentials. EXT: extension, FLX: flexion, PF: plantarflexion, DF: dorsiflexion.

4.3 Walking on Irregular Terrain

The model can walk on some irregular terrains with the CPG network alone. No higher control actions are needed. It can walk over a 45 mm high obstacle, approximately 5 % of the leg length (Fig. 5(a)). It can also walk up a 5° incline (Fig. 5(b)) and walk down a 5° decline (Fig. 5(c)).

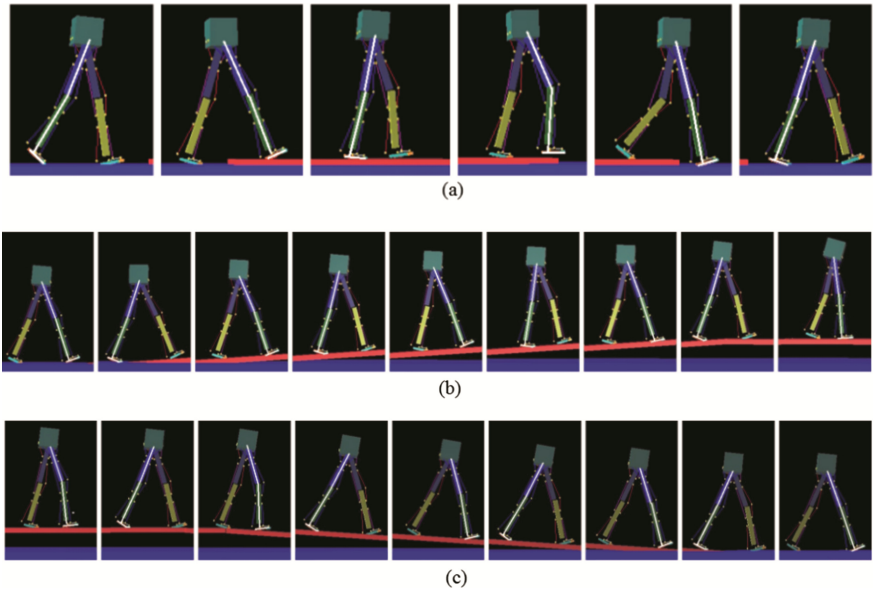

Fig. 5. Walking on irregular terrain (a) Walking over a 45 mm high obstacle. (b) Walking up a 5° incline. (c) Walking down a 5° decline.

5 Conclusions

In this paper we present a bipedal simulation model that can walk naturally and stably in the sagittal plane controlled by a biologically inspired neural network. It notably does not have inertial sensors or a central posture controller, nor does it use a "baby walker" to oppose gravity. Its structure and actuators are modeled after a human's lower body. Our model uses Hill's muscle model as actuators instead of motors at joints, which models the force profile of muscles and stores and dissipates energy during walking similar to humans. A two-component foot structure not only enhances the leg's ground contact late in the stance phase but also makes it possible to provide accurate dynamic sensory information of the relation between the foot and ground during stance by using individual sensors on toe and heel.

Using a neural network as a controller provides a more tangible and consistent imitation of the human central nervous system related to walking than switching between two sets of abstract equations to coordinate muscle reflexes. Compared to other bipedal models or robots adopting CPGs, our neural network has several more biologically plausible features. The rhythmic patterns are generated by oscillators composed of biological neuron models, not by mathematical oscillators tuned to reproduce the trajectory of joint movement. In our model sensory inputs are all biologically realistic and they access the CPG network and entrain its output instead of directly modifying activities of motoneurons. In the two-level architecture the movement of hip joints are

coordinated under a higher level rhythm generator instead of directly coupling hip pattern generators of two legs.

Our developed controller and simulation not only demonstrates that stable bipedal walking in the sagittal plane can be achieved through a biologically based neural controller, but can also be used as a platform to help researchers test hypotheses related to the neural control of human locomotion.

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