

# Biologically Inspired Neural Controller for Quadruped\*

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**Abstract**—Our study aims at the design and implementation of a general controller for quadrupedal locomotion, allowing the robot to use the whole range of quadrupedal gaits (i.e. from low speed walking to fast running). A general legged locomotion controller must integrate both posture control and rhythmic motion control and have the ability to shift continuously from one control method to the other according to locomotion speed. We are developing such general quadrupedal locomotion controller by using a neural model involving a CPG (Central Pattern Generator) utilizing ground reaction force sensory feedback. We used a biologically faithful musculoskeletal model with a spine and hind legs, and computationally simulated stable stepping motion at various speeds using the neuro-mechanical system combining the neural controller and the musculoskeletal model.

**Index Terms**—Quadruped, Neural Controller, Posture, Rhythmic Motion, CPG, Computational Simulation

## I. INTRODUCTION

Control methods for legged locomotion are generally classified into ZMP-based method and limit-cycle-based method[1]. When we consider legged locomotion with Froude Numbers (FN)  $v^2/gL$  ( $v$ : forward speed,  $g$ : gravity acceleration,  $L$ : leg length), we can point out the following:

- at low FN (low speed), since gravity is dominant, posture control using sensory information such as ground reaction force<sup>1</sup> and/or vestibular information is more important.
- at high FN (high speed), since inertia force is dominant, rhythmic motion control to construct a limit cycle is more important.

Consequently, a general legged locomotion controller must integrate both posture control and rhythmic motion control and have the ability to shift continuously from one control method to the other according to locomotion speed. But, to the authors' best knowledge, implementation of such a controller has never been reported.

In former studies, we realized adaptive walking on irregular terrain using a mammal-like quadruped robot "Tekken." Our approach until now has been to use a control system based on the CPG (Central Pattern Generator) and reflexes paradigm[1]. However, we were not able to realize stable low speed walking with low stepping frequency mainly due to

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<sup>1</sup>ZMP or COP (center of pressure) is such information.

the lack of leg loading sensory feedback to CPGs for posture control[2]. Therefore, we started to design a more sensor-dependent CPG model, following the arguments by Cruse[3] that:

- A central rhythm generator implying a "world model" in the form of a central oscillator could even cause the behavior to deteriorate in unpredictable situations.
- Local rules exploiting feedback loops and the mechanical properties of the body can produce the basic rhythm and can sufficiently explain a considerable part of the coordination.

In other words, while generating the self-excited physical oscillation as a result of local feedback, we might be able to integrate sensor-dependent posture control and sensor-driven rhythmic motion control.

In this paper, we extended and improved the Neural Phase Generator architecture proposed by Wadden et al.[4] to use it with the musculoskeletal model proposed by Ekeberg et al.[5], and simulated stepping motion using a two hind legs model. By doing this, we obtained the original results of being able to induce autonomous speed and stepping pattern modulations according to the change of a single tonic input (modeling the input from the upper neural system), while using that biomorphic musculoskeletal model.

## II. MECHANICAL AND NEURONAL MODELS

As it is broadly admitted that the control system for locomotion in vertebrates is distributed and modular, it is natural to design first the neural controller responsible for the stepping movements of a single leg. This neural controller will be later referred in this paper as the Leg Controller (LC). As the structure of the LC is highly dependent on the structure of the musculoskeletal model, we first define the musculoskeletal model and then detail the design of the LC.

### A. Musculoskeletal model

We are using a slightly modified version of the hind legs musculoskeletal model of [5], including three links and seven muscles (Fig.1). We use the muscle model of Brown et al.(1996) with parameters from Ekeberg et al.[5]. The muscle fascicles are modeled as active contractile elements (CE) (using Hill-type relations) in parallel with passive elastic elements (PE). The force output by the contractile element is scaled by the muscular activation level  $a_m$ , as shown in the following equation for muscle  $m$ :

$$F_m = F_m^{max} \cdot \max(a_m F_{CE}(x_m, v_m) + F_{PE}(x_m), 0) \quad (1)$$

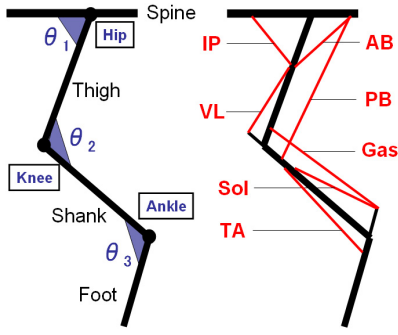


Fig. 1. **Left:** joint angles. **Right:** musculoskeletal model (IP: Iliopsoas, AB, Anterior Biceps, PB: P. Biceps/Semitendinosus, VL: Vastus Lateralis, Gas: Gastrocnemius, TA: Tibialis Anterior, Sol: Soleus)

$$m \in \{IP, AB, PB, VL, Gas, Sol, TA\}$$

where  $x_m$  and  $v_m$  are the relative muscle length and muscle contraction speed respectively, while  $F_m^{max}$  is the maximum isometric force.

### B. Overview of the leg controller

The distributed neural controller originally developed by Wadden et al.[4] consisted of three parts, those were neural phase generators, fast feedback pathways and a higher level controller. We extended their controller by dividing it into other three parts and improving each part for our Leg Controller (LC) in order to simulate more adaptive stepping motion according to sensory inputs and tonic input, while utilizing the dynamic properties of the more complex, animal-like musculoskeletal model.

Our LC (Fig. 2) is made of three parts: the Neural Phase Generator (NPG), the Motor Output Shaping Stage (MOSS) and the Propulsive Force Control Module (PFCM).

- The NPG activity characterizes the current phase of the leg. The functional units of the NPG layer are called “modules.” Transition of activity from one module to another is under heavy sensory control, and all the modules are activated in sequence during one locomotion cycle.
- Associated to each NPG module is a set of so-called “synergies”<sup>2</sup> in the MOSS layer. Initiation and termination of the activity of one synergy obey to a given timing or can be triggered on the basis of sensory information. Each synergy has a feed-forward and a feedback components. The outputs of such components are summed by the motor neurons (MN), which output the muscular activation levels  $a_m$  for the muscular system.
- The PFCM is responsible for the adjustment of the stepping frequency and locomotion speed according to a single tonic input from the upper neural system. In the case of a cat, it is well known that locomotion speed and gait selection can be regulated by the electrical stimulation of a small region in the midbrain, called the Mesencephalic Locomotor Region (MLR)[6], so that

<sup>2</sup>Simultaneous activation of muscles to fulfill a given motor goal

when the stimulation increases, the cat changes its gait from walk to trot and even gallop. It is shown that the MLR stimulation is related to the propulsive force developed by the cat, i.e. the intensity of muscle contraction. Accordingly, in our controller, the PFCM will not act directly on the rhythm generation at the NPG level but instead modulates the intensity of the muscular activations output by the MOSS by modifying the feed-forward levels and the feedback paths gains.

### C. Models of neurons

Various kinds of neurons are used in the LC shown in Fig. 2. The main type is a slightly modified version of the interneuron type used in [4] to design the NPG. We added four other types to achieve various functions in the LC.

1) *Interneurons (I)*: Each neuron represents a population of functionally similar neurons and its output is the mean firing frequency of the population. It is basically a “leaky integrator” with saturating transfer function and thus an output value between 0 and 1 (the maximum firing rate). Each neuron is characterized by three parameters: the time constant ( $\tau$ ), the gain ( $\Gamma$ ) and the activation threshold ( $\Theta$ ). The excitatory and inhibitory synaptic inputs are handled separately (using the presynaptic inputs from sets  $\Psi_+$  and  $\Psi_-$  respectively):

$$\dot{\xi}_+ = \frac{1}{\tau} \left( \sum_{i \in \Psi_+} u_i w_i - \xi_+ \right) \quad (2)$$

$$\dot{\xi}_- = \frac{1}{\tau} \left( \sum_{i \in \Psi_-} u_i w_i - \xi_- \right) \quad (3)$$

where  $w_i$  is the strength of the synapse  $i$  and  $u_i$  the output value from the corresponding presynaptic neuron. We added two features to the original model: the saturation of inhibitory synaptic input ( $\xi_- \leq 1$ ) and the resetting of  $\xi_+$  to 0 when the neuron is completely inhibited ( $\xi_- = 1$ ).  $\xi_+$  and  $\xi_-$  are then recombined to generate the neuron output:

$$u = \begin{cases} 1 - \exp\{(\Theta - \xi_+)\Gamma\} - \xi_- & \text{if positive} \\ 0 & \text{otherwise} \end{cases} \quad (4)$$

This is the default output function. However, for the sake of simplicity, this is sometimes replaced by the following simpler linear model:

$$u_{lin} = \begin{cases} \min((\xi_+ - \Theta)\Gamma, 1) - \xi_- & \text{if positive} \\ 0 & \text{otherwise} \end{cases} \quad (5)$$

2) *Sensory neurons (SN)*: The neuronal model used for sensory signals transduction is the same as above except for two features. First, the excitatory synaptic input is function of  $\varphi$  (the monitored quantity) and  $\dot{\varphi}$  (its derivative). Second, the output function is simpler (similar to  $u_{lin}$ ).

$$\dot{\xi}_+ = \frac{1}{\tau} (K_p(\varphi - \varphi_{off}) + K_v \dot{\varphi} - \xi_+) \quad (6)$$

$$u_{SN} = \begin{cases} \min(\xi_+, 1) - \xi_- & \text{if positive} \\ 0 & \text{otherwise} \end{cases} \quad (7)$$

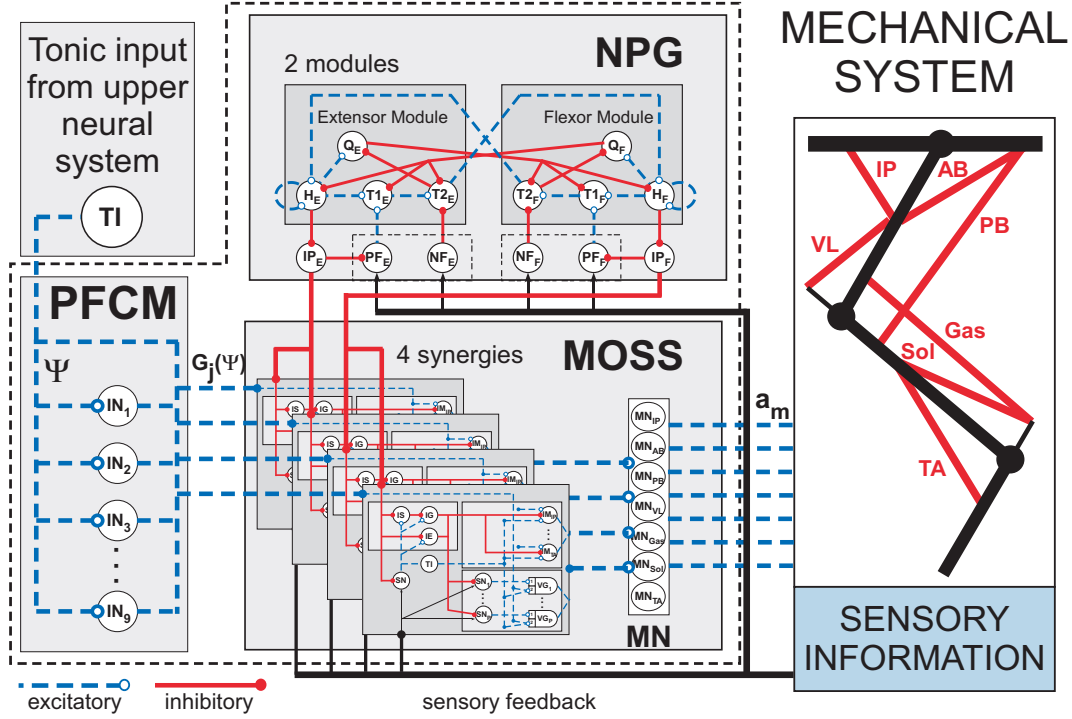


Fig. 2. Overview of the Leg Controller

where  $\varphi_{off}$  is an offset value. Currently, four kinds of sensory information coming from the musculoskeletal model are available to the LC: the muscular force, the muscle length and the muscle contraction speed of each muscle, as well as contact with the ground status for each leg. The first three sensory information will be noted, for a muscle  $m$ ,  $f_m$ ,  $x_m$  and  $v_m$  respectively, and the sensory neurons handling those signals  $SN_s$  (with  $s \in \{x_m, f_m\}$ ).

3) *Motor neurons (MN)*: Motor neurons simply sum the presynaptic inputs and output the sum (if it is positive).

$$\xi_+ = \sum_i u_i w_i \quad (8)$$

$$u_{MN} = \max(\xi_+, 0) \quad (9)$$

One motor neuron  $MN_m$  corresponds to each muscle  $m$ , and its output ( $u_{MN}$ ) is used as the muscular activation level  $a_m$  of eq.(1).

4) *Variable gains (VG)*: Variable gains are building units that output the product of their two inputs (the first one being the signal and the second one the gain).

5) *Tonic input neurons (TI)*: These neurons output a constant value.

#### D. Neural Phase Generator

1) *Neuronal Structure*: We improved the NPG module proposed by Wadden et al.[4] in order to cope with more sensory information. Our NPG is made of two modules, the extensor and flexor modules (Fig.3). Each module is made of seven neurons (all the following neurons are interneurons, except  $PF$  and  $NF$  which are sensory neurons):

- $H$  neuron is the neuron representative for the activity of the module. It has excitatory connections with itself

(self-excitation), as well as  $Q$  and  $T_1$  neurons. On the other hand, it inhibits the  $IP$  neuron.

- $Q$  neuron ensures that only one module is active at the time by inhibiting the  $H$  and  $T$  neurons of the other module.
- $T_1$  and  $T_2$  neurons are responsible for the transition to the other module. In Wadden and Ekeberg (1998), one module contained only one  $T$  neuron, receiving only excitatory sensory inputs. In our model, we divided it into two: a slow  $T_1$  neuron which receives excitatory inputs from both  $H$  neuron and sensory feedback pathways (through the  $PF$  neuron), and a fast  $T_2$  neuron which is under inhibitory sensory influence (through the  $NF$  neuron).  $T_1$  excites  $T_2$  neuron which then promotes transition by exciting the  $H$  neuron of the other module, while reducing its inhibition by inhibiting the  $Q$  neuron of its own module. This structure allows to set the time constant  $\tau$  of  $T_1$  in order to achieve long duration of module activity without slowing down the transition when inhibitory sensory input acting on  $T_2$  disappears.
- $IP$  neuron is the complementary neuron of  $H$ : it is active when the module is inactive. It has inhibitory connections with the interneurons of the synergies in the MOSS corresponding to the module it belongs and with  $PF$  neuron (gating role). When  $H$  becomes active, it inhibits  $IP$ , hence it activates the previously inhibited neurons.
- $PF$  and  $NF$  neurons are the sensory neurons which relay the sensory information to the transition neurons.  $PF$  is used for the sensory feedback which promotes the transition and  $NF$  for the sensory feedback which

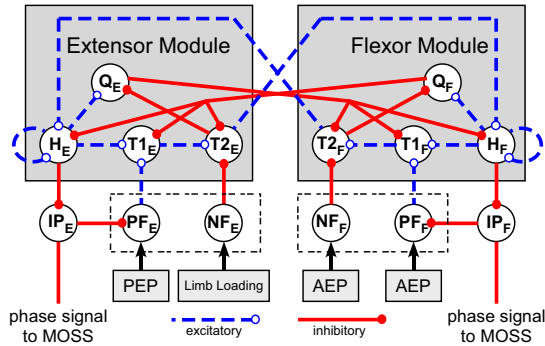


Fig. 3. Internal structure of the NPG

prevents it.

2) *Sensory feedback*: We used the same kinds of sensory information as Ekeberg et al.[5] for the sensory feedback to the NPG:

- **Anterior Extreme Position (AEP)** proximity, evaluated using the length of AB muscle, is used to control the transition of activation from the flexor to the extensor module through excitatory and inhibitory influences on the transition neurons of the flexor module.
- **Posterior Extreme Position (PEP)** proximity, evaluated using the length of IP muscle, promotes the transition of activation from the extensor to the flexor module.
- **Leg Loading (LL)**, evaluated using the muscular force developed by the Soleus, prevents the transition of activation from the extensor to the flexor module, as long as the leg loading is over a given threshold. This kind of sensory feedback was not used in [4] and the transition from stance to liftoff depended only upon feedback about PEP proximity.

### E. Motor Output Shaping Stage

The MOSS is under inhibitory influences from the NPG, coming from the IP neurons of each module (Fig. 2, 3). These signals are used to select the synergies during the active period of the corresponding NPG module.

The structure of each synergy is shown in Fig. 4, where *IM* and *VG* are output neurons of the feedforward part and the feedback part, respectively. For both the feed-forward and the feedback parts, the muscular output levels depend on the connectivity of the *TI* neuron to the output neurons (*IM* and *VG*) and on the input from the PFCM. The first component will be called the *basic activation levels*, while the second is the *modulation from the PFCM*.

Currently, two synergies are implemented for each module: liftoff (**LO**) and swing (**SW**) synergies for the flexor module, and touchdown (**TD**) and stance (**ST**) synergies for the extensor module. These synergies are the same as the ones used in [4] and [5] (Fig. 5). Each synergy is responsible for the simultaneous feed-forward activation of a certain number of muscles and the opening of specific feedback paths in order to fulfill a given motor function. The active *IM<sub>m</sub>* and *VG<sub>s-m</sub>* neurons in each synergy are shown in TABLE I. The details

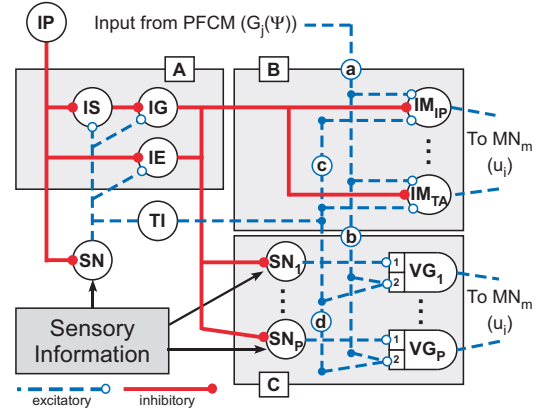


Fig. 4. Synergy structure. [A]: Initiation and termination part. [B]: feedforward part. [C]: feedback part (*P*: number of sensory feedback pathways of the synergy).

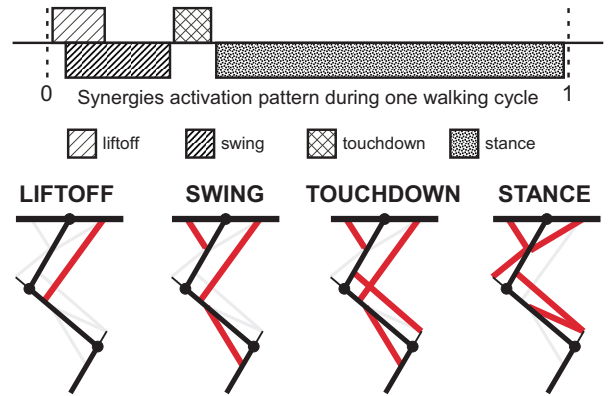


Fig. 5. **Upper part**: Example of synergies activation pattern during one stepping cycle. Liftoff starts right after the beginning of the flexor module active phase and is shut down after around 50 ms, while swing starts around 20 ms after the beginning of the flexor module activation and last until its end. Touchdown starts right after the beginning of the extensor module active phase and ends when the foot touches the ground, triggering the initiation of the stance which lasts until the end of the extensor module active phase. **Lower part**: active muscles for each synergy.

of the open sensory feedback pathways in each synergy are eliminated in this paper because of limited space.

### F. Propulsive Force Control Module

The PFCM is the part of the LC responsible for adjusting the propulsive force according to the intensity of the tonic input from upper neural system.

As the results of simulations at various speeds of steady walking, it appeared that speed adjustment could be carried out by modifying the value of only a few number of parameters. Using such results, we aggregated the values of control parameters to change walking speed into relationships implemented using interneurons (Fig. 2).

We used interneurons with the simplified linear output (eq.(5)) to implement the right factor of the right side of the relationship. The outputs of these interneurons become the inputs to *IM<sub>m</sub>* and *VG<sub>s-m</sub>* (Fig. 4) while being calculated

TABLE I  
ACTIVE  $IM_m$  (FEEDFORWARD COMPONENT) AND  $VG_{s-m}$  (FEEDBACK COMPONENT) FOR EACH SYNERGY

Synergy	Active $IM_m$	Active $VG_{s-m}$	
LO	$IM_{PB}$	$x$	
		$f$	
SW	$IM_{IP}, IM_{TA}$	$x$	$VG_{xPB-PB}, VG_{xPB-TA}$
		$f$	
TD	$IM_{IP}, IM_{Gas}, IM_{TA}$	$x$	$VG_{xPB-PB}$
		$f$	
ST	$IM_{VL}, IM_{Sol}$	$x$	$VG_{xIP-IP}, VG_{xIP-VL}, VG_{xIP-Sol}, VG_{xAB-AB}, VG_{xVL-VL}$
		$f$	$VG_{fVL-VL}, VG_{fGas-Sol}, VG_{fSol-Gas}$

by the following equation.

$$G_j(\Psi) = K_j * \max(0, \min(1, (\Psi - \psi_{th,j}))) \quad (10)$$

where  $K_j$  are the gains and  $\psi_{th,j}$  is the threshold values for the  $j^{th}$  variable parameter. The gains  $K_j$  correspond to the connection weights ( $w_{PFCM-IM}$  and  $w_{PFCM-VG}$ ) between the PFCM interneurons and the MOSS output neurons ( $IM$  and  $VG$ ). Using this implementation, we can change the walking speed by the single control input  $\Psi$  while driving the LC.

### G. Numerical value of the parameters

The numerical value for all the parameters of the LC (NPG neurons and sensory feedback paths, MOSS basic activation levels and modulation from the PFCM) can be found in [7].

## III. SIMULATION RESULTS

In order to validate our Leg Controller (LC) architecture, we tested it on the two hind legs model represented on Fig. 6. First, we confirmed that the results of Ekeberg et al.[5] could be reproduced. Next, we simulated autonomous speed and stepping pattern modulations of the stepping motion of the two hind legs model according to change of a single tonic input from the upper level.

### A. Generation of stepping motion

Using the simulation model of Fig.6, we were able to generate stable alternative stepping at various speeds. As there is no explicit interaction between two LCs, the coordination of the legs is an emergent property of the system, induced by the way the LC are interacting with the environment through the sensory feedback. This was however a predictable result since the LC uses the information about the load supported by the leg as the main sensory signal to trigger the transition between the extensor and the flexor modules. Nevertheless, our results show that this conclusion can be extended to a much broader range of speed than the one investigated in that previous study.

### B. Adjustment of locomotion speed

The PFCM makes the model be able to walk at different speeds by simply modifying the value of  $\Psi$  of two LCs. Values of the control input ranging from 0.05 to 0.45 allowed to evoke locomotion speed ranging from around 0.15 to 0.80 m/s (Fig. 7).

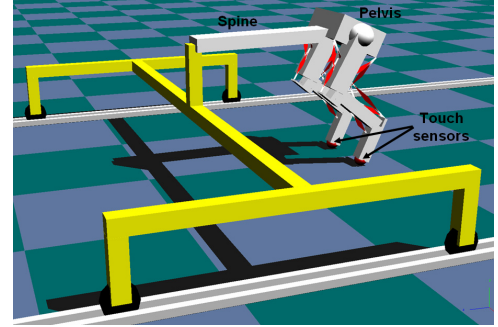


Fig. 6. Simulation Model. The spine can rotate around the pitch axis.

### C. Characteristic parameters of the walking gait: comparison with biological data

We investigated the characteristic parameters of the simulated walking patterns at various speeds and we compared them to biological data we could find in the literature about cat walking in the same range of speed (from [8]). We found that the stance and swing phases durations of the walking patterns generated in simulation have the same global behavior as the one observed in animals, i.e. the swing period is more or less constant with respect to the speed, and the stance period drastically drops as the speed increases (Fig. 8). However, in general, the duration of the stance phase in simulation is lower than the corresponding duration found in biological data for the same speed. As a consequence, the same tendency can be observed for the stepping period (upper part of Fig. 9). The behavior of the duty ratio with respect to the speed is respected as well (lower part of Fig. 9).

## IV. DISCUSSION

The design of the NPG structure and the determination of the sensory signals used to trigger the transition of activation between the NPG modules are grounded on the following biological facts:

- *After deafferentation, oscillatory patterns of extensor and flexor muscular activity still occur under pharmacological excitation* [9]: in our model, even without sensory feedback, transitions of activation between modules can occur if the synaptic weights of the connection between the H and  $T_1$  neurons are high enough to activate  $T_1$ . In that case, the NPG has an oscillatory behavior and the duration of the activity of each module depends

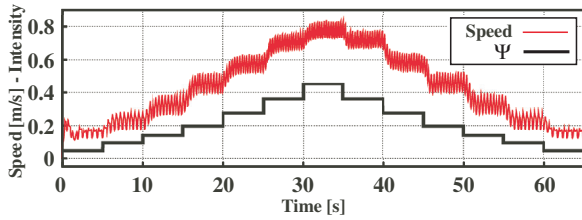


Fig. 7. Walking speed change (starting from rest, increasing  $\Psi$  (resp. decreasing  $\Psi$ ) increases (resp. decreases) the walking speed). High frequency oscillations of the speed are due to the variation of the speed during the step

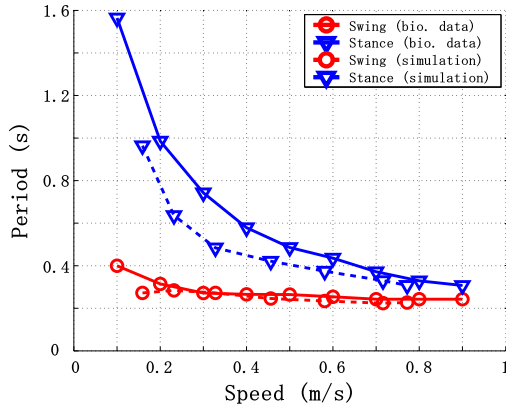


Fig. 8. Changes of the swing and stance phases durations with respect to the locomotion speed, in real animals and in our simulation.

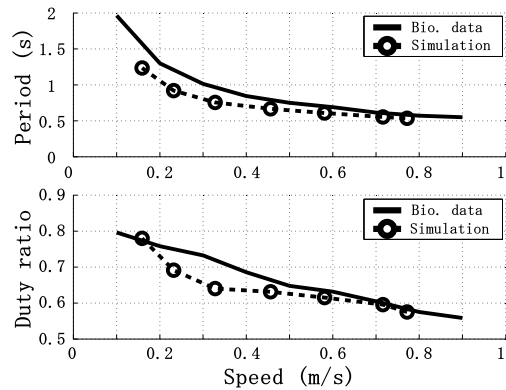


Fig. 9. Changes of the stepping period and the duty ratio with respect to the locomotion speed, in real animals and in our simulation.

on the synaptic weights between the neurons of the transition path and their time constants. By changing these parameters, the oscillation frequency and duty ratio could be tuned by the upper neural systems. However, this function was not implemented here and the values of the connection weights were set under the threshold allowing oscillations.

- *Transition from stance to swing is prevented as long as the hip angle is lower than a given threshold* [10]: in the condition stated above, the intrinsic excitation from  $H_E$  to  $T_{E1}$  is too weak to evoke the transition and it is the excitatory input coming from the PEP feedback (through

$PF_E$ ) which is really responsible for  $T_{E1}$  activation.

- *The extensor module active phase is indefinitely prolonged if the force developed by the ankle extensor muscle is over a given threshold* [11]: as long as the leg is loaded,  $T_{E2}$  is inhibited by the LL feedback (through  $NF_E$ ) which prevents the transition to the flexor module.
- In agreement with the conclusion of [5], leg loading is used as the decisive sensory information for the transition of activation from the extensor to the flexor module as, even if  $T_{E1}$  is activated by PEP sensory feedback signal, it can not trigger the transition as long as  $T_{E2}$  is inhibited by the LL sensory feedback signal.

The better integration of the sensory feedback (especially the leg loading sensory information) in the rhythm generation part of the CPG allows us to hope for more adaptability to the environment than the CPG we used for Tekken[1] and even for basic integrated postural control.

## V. CONCLUSIONS AND FUTURE WORK

In this paper, we reported the design and validation in simulation of a single leg neural controller for a biomorphic model of the hind legs of a cat. The controller was able to induce stepping of the mechanical model at speed from low to medium speed walking. Moreover, the main parameters of the stepping patterns were behaving in a similar way as in the biological data of real cat walking. In the next phase, we intend to investigate whether we can reproduce the walking motion from the dynamical point of view by using a 3D quadrupedal model[12] while comparing data of ground reaction force and so on. In the future, we try to implement the control system on a real robot for validation of our simulation results.

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