CPG Design using Inhibitory Networks *

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***Abstract* – We describe in detail the behavior of an inhibitory Central Pattern Generator (CPG) network for robot control. A four-neuron, mutual inhibitory network forms the basic coordinating pattern for locomotion. This network then inhibits an eight-neuron network used to drive patterned movement. We show that we can get predictable control of important relationships such as the phase of the hip and the knee by adjusting tonic parameters. We demonstrate the basic concept both in a simulation that is used to drive a trotting bipedal robot as well as an aVLSI CPG chip that generates spiking burst patterns. Our results indicate that an inhibitory framework can generate simple, understandable and flexible networks for legged robot control that can be implemented in custom VLSI circuits.**

Index Terms – Central Pattern Generator, Silicon CPG, Bipedal Locomotion, Walking Machines, Biologically Inspired Systems

I. Introduction

CPGs are an attractive means of controlling legged robots because

- They can be implemented in custom VLSI hardware allowing a compact, low power and elegant controller [1-9]
- They are compatible with a dynamic systems approach to robot control which may result in more compact and mechanically efficient movements;
- Movement and perception seem tightly coupled. If we hope to import other biologically inspired aspects of intelligence, we should consider building a movement based system that is biologically motivated.

The drawback of using CPGs is that the mathematical tools for the systematic design of such networks, necessary to meet specific engineering requirements, have not been worked out. Softcomputing methods such as genetic algorithms have been applied in the past to the design of such circuits [10]. Simplified models of CPGs have been designed using Phase oscillators that are amenable to mathematical analysis [11-13].

Our work here concerns building networks using spiking neurons. Spiking neurons are attractive because they more closely reflect biological neurons, and may be more compatible with biomedical applications such as neuroprostheses.

While the biological literature *describes* the behavior of networks, it does not *prescribe* a design. We have discovered a particularly simple network based on inhibitory connections that allow reasonably predictable designs of neural central pattern generators.

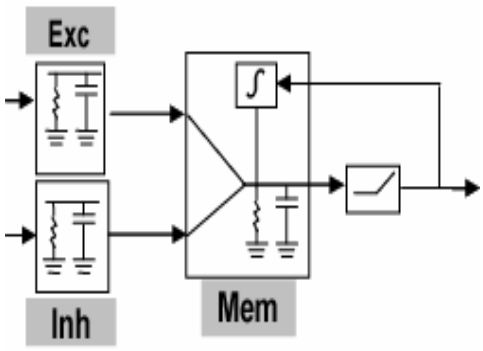

Figure 1. Model of neuron used to construct the motor control network that controls the RedBot Robot.

The core idea we start with is that of a classic half-centered oscillator [14]. The half-centered oscillator is composed of two neurons that have mutual inhibition. These networks can enforce 180° phase difference between neural outputs.

By modulating a tonic drive to this network we can speed up the network, slow it down, cause it to go into a "tenus" phase where both neurons are excited, or shut it off completely.

Next, we use the output of the half-centered oscillator to *inhibit* a network of motor neurons. This is in contrast to the use of an oscillator network to excite a motor neuron network. The advantage of this approach is that when inhibition is removed, the nominal firing rate of the motor neuron is controlled by a descending tonic drive. The inhibition pushes the neuron until it is hyperpolarized. The depth of this hyperpolarization causes the motor neuron to shut off. The time it takes to turn back on depends on the depth of hyperpolarization, which can introduce a phase delay between the release of inhibition and the turn on of the motor neuron. This can be overcome to some extent via an increased tonic drive.

We next turn to the details of the neural model that we used. We have implemented this model in software as well as in an analog Very Large Scale Integration (aVLSI) circuit. We present results in both software and the control of a fast moving biped as well as results in creating appropriate waveforms using the CPG chip.

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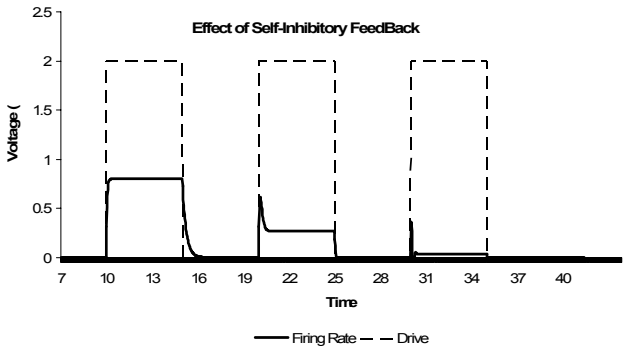

Figure 2. Effect of Self Inhibitory Term. The firing rate is shown as a solid line and the input drive is shown as a dashed line. The input weight is unity. The plot depicts the effect of three self-inhibitory feedback strengths.

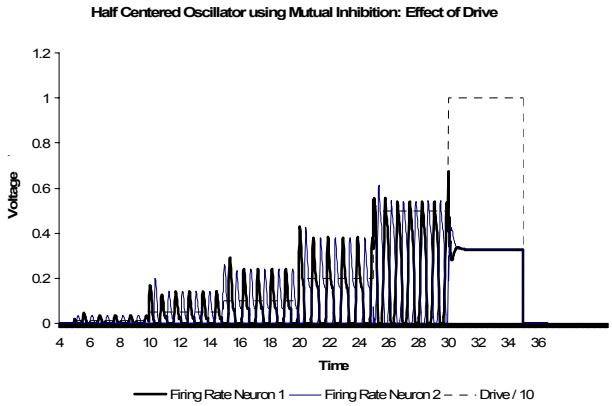

Figure 3. Effect of drive force on a coupled set of neurons with mutual inhibition. Six different levels of drive force are demonstrated.

II. Neural Model Used in Simulation Studies

The neural model is multicompartmental, as shown in Figure 1. For the sake of clarity, some of the details are omitted from the system diagram depicted. The exact equations are given by:

$$\tau_{mem}\frac{d}{dt}V_{mem} = -v_{mem} + (V^+ - V_{mem}) \bullet Syn^+ + (V^- - V_{mem}) \bullet Syn^- - fb \bullet \alpha \quad (1)$$

$$u = \max(0, V_{mem}) \quad (2)$$

$$Syn^+ = \sum S_i^+ \quad (3)$$

$$Syn^- = \sum S_i^- \quad (4)$$

$$\tau_{syn}\frac{d}{dt}S_i^- = -S_i^- + w_i \bullet u_i \quad (5)$$

$$\tau_{syn}\frac{d}{dt}S_i^+ = -S_i^+ + w_i \bullet u_i \quad (6)$$

$$\frac{d}{dt}fb = u \quad (7)$$

where

τ_{mem} =500 ms, the cell membrane time constant

τ_{syn} =100 ms, the synapse time constant

V_{mem} the cell membrane voltage

V^+, V^- the positive and negative voltage drives.

In the simulations described here, Euler integration was used with a time-step of 20 ms.

III. Results

A. Effect of self-inhibitory term

The effect of the self-inhibitory feedback is illustrated in Figure 2. The tonic drive is switched on three times during the time course of this experiment at t=[10,15], t=[20,25] and t=[30,35], and it results in changes in membrane voltage.

In particular, during the first burst of activity, there is no self-inhibitory contribution, i.e.: α=0. Here, the membrane voltage exhibits an exponential charging/discharging as would be expected.

During the second burst of activity we set α =10. In this case, the cell membrane voltage rises sharply as before, but then reduces and finally achieves a steady state rate.

During the third burst of activity, we set α =100. Qualitatively, the response is similar to the second burst of activity. However, the steady state response is further reduced and, more significantly, the time course of the response is compressed. The cell therefore "switches off" more quickly in the case of a stronger feedback contribution.

B. Coupling two neurons to create a half-centered oscillator

The next step consists in building a two cell CPG circuit. This is done by coupling two neurons together and driving inhibitory synapses. The self inhibitory feedback term was set to 20. Referring to Figure 3, the drive force was varied as follows: drive = 0.1 for t=[5.0,10.0), drive = 0.5 for t=[10.0,15.0), drive = 1.0 for t=[15.0,20.0), drive = 2 for t=[20.0,25.0), drive = 5 for t=[25.0,30.0), drive = 10 for t=[30.0,35.0). In all, the drive was varied by two orders of magnitude.

As can be seen, the firing rates of the two cells oscillate 180° out of phase with each other. We also note that the greater the driving force, the greater the amplitude of the output. The output frequency, however, does not change until the drive rate becomes very high and the neurons begin to stay switched on.

Clearly, then, we can conclude that the consequence of an increase in the tonic drive is an increase in the magnitude of the CPG's output.

C. Effect of altering the self-inhibitory term

In Figure 4, we examine the effect of changing the self inhibitory feedback term. The term varies from a value of 15 for t=[5.0,10.0), to 20 for t=[10.0,15.0), to 30 for t=[15.0,20.0), to 40 for t=[20.0,25.0), to 50 for t=[25.0, 30.0).

As can be seen, the effect of altering this feedback term is an increase in the frequency of the oscillation. In addition, there is a minor decrease in the amplitude of the response, similar to the increase found as consequence of a stronger tonic drive.

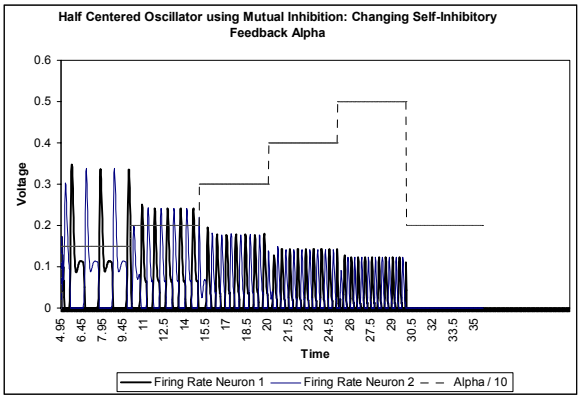

Figure 4. The effect of altering the self-inhibitory feedback term.

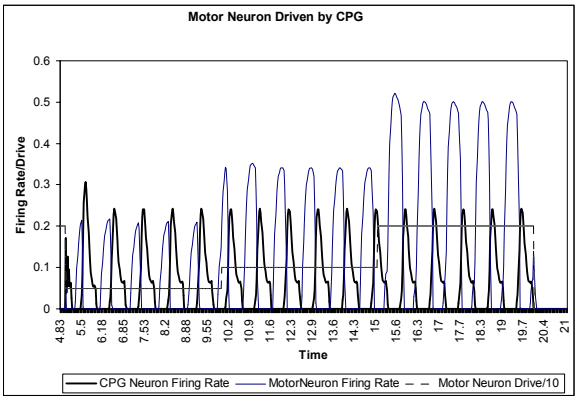

Figure 5. Effect of changing the drive to the motor neuron and phase. The drive to the motor neuron changes both the phase and the amplitude of the driven motor neuron.

Varying the mutual coupling term did result in a similar effect. However, we found that the mutual coupling parameter was very sensitive to variation. This would not be a desirable property in an engineered system. Due to space limitation, we do not present the results of those experiments here.

D. Driving a motor neuron from the CPG

Building on these results, the CPG can now be used to drive a motor neuron. We use inhibitory coupling between the CPG and the motor neuron. A tonic drive to the motor neuron, similar to the one used by the CPG, will activate it. Periodic inhibition from the CPG will then stop this activity. We can separately drive the motor neuron with a tonic input. The effect of changing this tonic input is illustrated in Figure 5. We vary the drive in three steps: 0.5 motorneuron drive t=[5,10), 1.0 motorneuron drive t=[10,15) and motorneuron drive = 2 for t=[15,20).

There are two key effects. The most evident is that increasing drive has the effect of increase the mean firing rate, as would be expected. The second effect is that increasing the drive causes the motor neuron to begin firing sooner, thus *advancing* the phase. By changing this drive, we will show that we can make changes to the phase relationship between joints in a robot walking machine sufficient to allow walking.

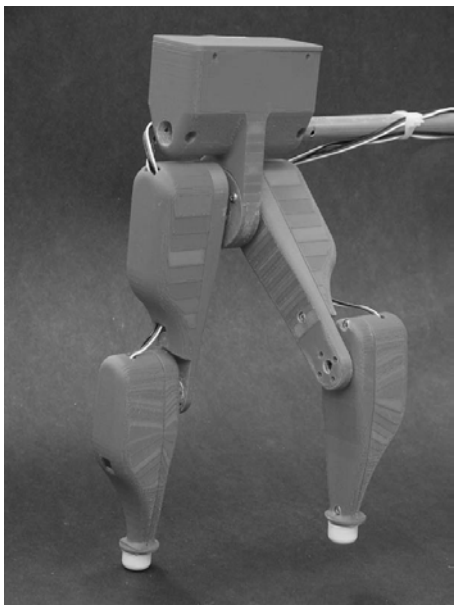

Figure 6. The "RedBot" robot mechanism used in the CPG experiments described here. The robot stands about 28 centimeters tall.

E. Additional Results

We also noted that when we drive the CPG network with a higher drive input, we create a higher firing rate for the CPG neuron. This has the consequence of inhibiting the Motor Neurons more. Thus it takes longer for them to start firing. This has the effect of *retarding* the phase relationship between the CPG neurons and the motor neurons.

IV. Experiments in a Robot

A. Robot Platform

The robot platform used is the "RedBot" biped sold by AlegROBOT, Inc. of Urbana, Il, and is shown in Figure 6. The robot is made from lightweight plastic and uses high speed servo motors for actuation. It is capable of walking frequencies as high as 4-5 Hz.

A network sufficient to control the robot, and constructed using the principles described above, is illustrated in Figure 7. The four CPG neurons used allow us to allocate 2 neurons to control the major movements of each hip joint. As the neurons have only positive firing rates, two neurons per joint are needed to specify backward and forward movement. In a system composed of two legs, with two neurons required per joint, a CPG network of a minimum of four neurons is necessary.

We then use a Motor Neuron network to construct the proper commands for hips and knees. We note that this is not the only architecture. Others have suggested that there might be a chain of unit CPGs. One unit CPG might control the knee joint, one the hip joint etc. This has been suggested by Grillner [15] more than 20 years ago. We have found that this idea is not compatible with robot control. Another approach is to transition from the CPG-based approach to a more kinematic-based robot control scheme for specification of the knee and hip joints. We found that this

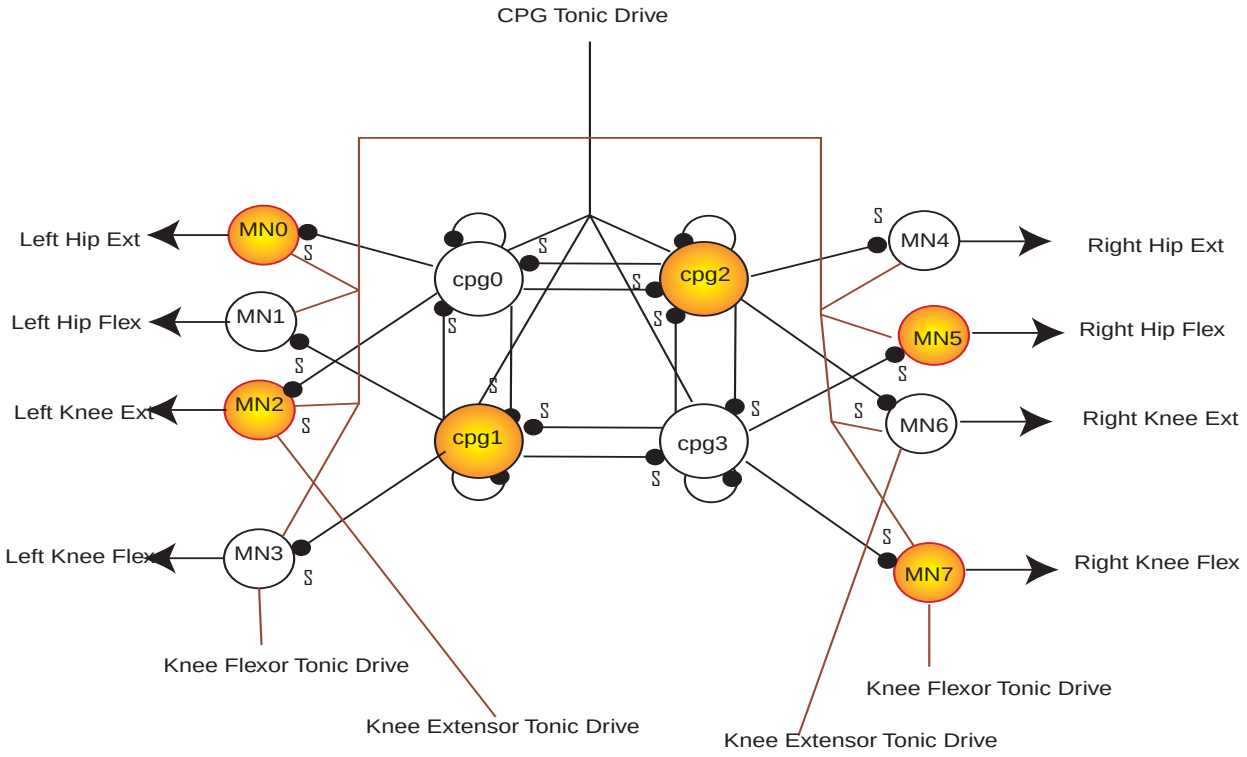

Figure 7. Network used to control robot.

was not necessary and creates an unnecessarily complex motor controller.

This is how the circuit works. The left and right halves of the network are about 180° out of phase. It is an approximate value and might depend to some degree on the exact parameters chosen. We assume that there is a distribution of weights and that all weights are not symmetric. Further, we assume a random distribution of state variables (the membrane voltages) when the system starts. These two conditions prevent the system from "locking up", i.e., if the network were completely symmetric, the membrane voltages would all be increasing at the same rate and it would not be possible to achieve out of phase oscillations.

There are two neurons on each side of the CPG: neurons CPG0 and CPG1 constitute the right side of the network and CPG2 and CPG3 constitute the left side of the network.

All four of these neurons receive a tonic drive input. As noted before, this tonic drive can start and stop the network and control the amplitude of the CPG output.

CPG0 then inhibits the left hip extensor and the left knee extensor. The phase relationship between the hip and the knee can be varied by providing the tonic drive input to the knee extensor. This tonic drive advances the phase of the knee versus the hip.

Likewise, CPG1 inhibits the flexors, and in this case also we can control the relationship between hip and knee by modulating the tonic drive.

It is also very important to note that this network gives us synergies for free. That is, the extensors tend to be activated together and the flexors are activated together. Thus, hip and knee are coordinated.

We have implemented this network on the Redbot and the robot can be made to trot at very fast speeds.

We varied the tonic drive to the knee flexor and extensor motor neurons as shown in Figure 8. In the condition labelled "A" the drive to the knee flexor was 3.0 and the knee extensor was 6.0. In condition "B" the flexor drive was 6.0 and the extensor was 3.0.

Thus, we see the change in the phase relationship between the hip and knee drive.

B. Silicon CPG network implementation

With reference to Figure 7, a CPG-based network, equivalent to one half of the network shown in the figure, was implemented in hardware using four Integrate-and-Fire (spiking) neurons of an aVLSI chip[8, 9]. The chip contains 10 neurons each with 19 synapses. In particular, the neuron has one synapse that charges the membrane just as the tonic drive used in the previous sections, 8 synapses that allow altering of the neuron output through external circuitry, and finally each neuron has the capability of feeding back its output signal to itself and/or to all the other neurons on the chip. Furthermore, since each synapse can be set to be excitatory or inhibitory and each synaptic input can be easily weighted by modifying a synaptic weight table, it is possible to build sophisticated neural networks such as the one shown in figure 7. The network built here, therefore, consists of 2 CPG neurons and the equivalent of four motor neurons. The CPG neurons, just as the neurons described in the previous sections, are characterized by the self-inhibitory synaptic connections upon themselves and the mutual inhibitory connections towards the antagonist CPG neuron. This allows the creation of alternating bursting activity between the two neurons. The situation is depicted

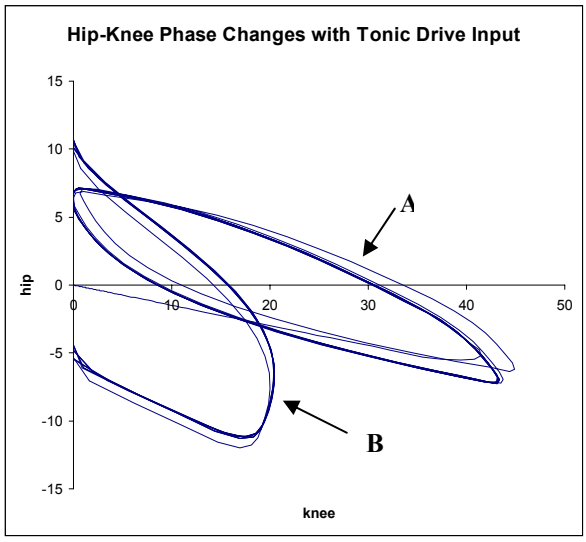

Figure 8. Hip/Knee phase. Effect of changing drive to knee flexor and extensor motor neuron. Condition "A" the knee flexor drive > knee extensor drive and in condition "B" the opposite was true: the knee extensor drive > knee flexor drive.

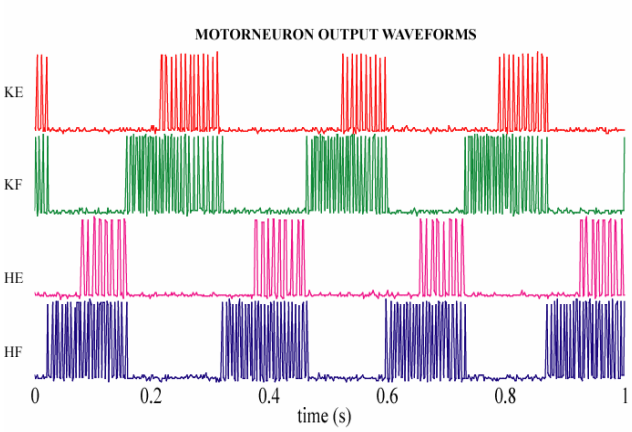

Figure 9. Output motorneuron waveforms. Note that each signal is ±90° out-of phase with the adjacent signal. These signals allow a robotic biped such as RedBot to walk in a humanlike fashion.

in Figure 9, where the hip extensor (HE) and flexor (HF) outputs behave identically to the two CPG neurons. To understand how this alternating activity is formed it is necessary to describe what happens inside each one of the two neurons. As the first bursting sequence displayed by the hip flexor in Figure 9 begins, the hip extensor is shut down due to the strong mutual inhibition signal coming from the flexor. At the same time, however, the self-inhibitory synapse progressively slows down the charging of the neuron's membrane potential, thereby making it harder and harder for that neuron to spike. (With reference to Figure 9, this phenomenon is more readily seen at the end of the hip extensor's first and third bursting patterns). At a certain point, the tonic input which drives the extensor overcomes the inhibition coming from the flexor and allows the extensor neuron's membrane potential to start charging. When it finally reaches threshold, a new mutual inhibition term from the extensor is added to the large self-inhibition value that the flexor neuron had accumulated to bring the flexor neuron's membrane potential low. Then the self-inhibition of the extensor starts to grow until the flexor can spike again and the process starts anew.

Depending on the weight set on the tonic input to the knee flexor or extensor we can then modulate the time-lag between the beginning of the firing of its respective hip signal and the beginning of its own firing. This means that with a strong weight, the two signals will begin the bursting at the same time (or, if the weight is particularly strong, then the knee signal can actually precede to some extent its respective hip signal), whereas if it is weak, the knee bursting will start late with respect to the hip signal. To attain a proper walking motion, the knee and hip extensors have to be 90° out-of-phase of each other, as do the knee and hip flexors. This situation is depicted in figure 9, where the time-lag between the various signals can easily be seen if the figure is viewed from the hip flexor signal towards the knee extensor signal: it is easy to see that each signal is +90° out-of phase with respect to the next one.

V. Summary and Conclusions

We first described a multi-compartmental model of a neuron with self-inhibitory feedback. We showed that by intra and inter inhibitor connections, an oscillator circuit could be built. In addition, we showed that increased self-inhibition leads to increased oscillation rate and that an increased tonic drive leads to a greater firing rate for the CPG neurons but little or no change in the burst frequency. We found that a four neuron network could be made to drive eight motor neurons and generate locomotor patterns necessary for bipedal trotting. Further, we found that by adjustment of tonic input to the motor neuron network, we could adjust the phase relationship between motor neurons driving the hip and motor neurons driving the knee.

We demonstrated that the same network architecture that worked in a mean firing rate simulation model was also valid in a silicon spiking neuron chip. We showed results in a real bipedal robot.

We found that our network creates synergies naturally, and that the coordination between joints is achieved without the need for a chain of oscillators as suggested by Grillner.

The network generated here relies heavily on inhibition in order to work. Indeed, the only excitation found in the network is the tonic excitatory drives. We found the network to behave very well: most of the parameters of the network could be varied over a wide range of parameters and the network would still exhibit a well controlled behaviour. We feel confident, because of this robustness to parameter variation, that our network will be easily implemented in a custom aVLSI chip.

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