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Reinitialization in bipedal locomotor control

By

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Abstract

An important feature of human locomotion is its instant adaptability to unpredictable changes in the conditions affecting gait. Humans can, for example, seamlessly adapt their gait to successive unpredictably fluctuating perturbations, *e.g.*, a series of oncoming collisions with other people in a crowded street. Based on neurophysiological evidence, theoretical studies of bipedal locomotion have revealed that a basic walking gait is generated by a coupled system composed of a central pattern generator (CPG) and the body itself. Modeling studies from a neurophysiological and biomechanichal perspective have referred to the leg posture at the beginning of the stance phase of the step cycle as the *initial state*, since it determines the subsequent behavior of the system with regard to whether or not it continues to walk. Such modeling studies have shown that coordination of the initial state can induce the coupled system composed of a CPG and body to adapt to various strong perturbations. We refer to the initial state coordination according to new conditions *reinitialization*. In this paper, we test the effectiveness of reinitialization in response to successive fluctuating perturbations using computer simulation, and analyze the process of reinitialization in terms of dynamic systems.

§ 1. Introdution

§ 1.1. Flexible locomotor control

Human locomotion in the real world must deal with unpredictable situations at every step. For example, when taking one step forward, the ground may change (*e.g.* become sandy or muddy) or a gust of wind may suddenly blow, the body may also collide with another person in a crowded place, or the legs themselves may be injured. However, by flexibly changing the gait following such successive unpredictable changes of condition, human walking is seamlessly maintained. Such flexibility to various unpredictable perturbations is one of the most significant features of human locomotion.

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The mechanisms underlying such flexible locomotor control are of interest to scholars in neuroscience, as well as in the physical and mathematical sciences.

Neurophysiological experiments concerning animal locomotion have revealed that basic stepping rhythm is controlled by rhythm-generating neural circuits. Such neural circuits are called central pattern generators (CPGs) and exist in the lower level of the central nervous system [1]. Although a CPG could dynamically generate rhythm by itself, the rhythm generated is entrained by the oscillatory motion of the motor apparatus in animals. This strongly suggests that stepping emerges from the dynamic interaction of the oscillations of the two dynamic systems, *i.e.*, the CPG and the body. Theoretical studies of bipedal locomotion [2] have demonstrated that a basic walking gait is generated by mutual entrainment between the CPG and the body, *i.e.*, by these *two coupled dynamic processes*, and a human locomotor CPG has been found in the spinal cord [3, 4]. This coupled pair of dynamic processes is a well-known conventional model of bipedal locomotion.

Two coupled dynamic processes form a dynamic system model. Given initial constraints (the initial state), the evolution of the system over time is governed by the dynamic properties according to which a given state determines its subsequent state. Changes of system parameters perturb the time evolution from the constraints, but do not make fundamental adjustments suited to the new conditions. A given constraint thus forever retains its influence on the time evolution of the system, regardless of parameter changes (changes in condition). We consider this to be the essential reason for the poor robustness of the conventional model.

Theoretical studies of dynamic systems [5, 6] have demonstrated that in the neighborhood of the neutral state, a slight difference in the way the system approaches the neutral state can induce the system to converge to quite different behaviors. In theoretical studies of human walking gaits, Ohgane *et al.* [7, 8] have shown that the neutral state is latent in the two coupled dynamic processes under the condition that no perturbations occur. The neutral state can be easily revealed by modulating the knee joint angle at the beginning of the stance phase (BSP). Because the system state at this phase determines the subsequent behavior of the system, *i.e.*, whether the walking system adapts or falls, this system state may be regarded as the *initial state*. Moreover, Ohgane *et al.* have shown that even when the vector field changes completely, *initial state coordination* can renew the walking pattern so that it suits the new conditions. In this paper, the term *'reinitialization'* is defined as such *initial state coordination* according to new conditions.

From a biomechanical point of view, bipedal locomotion can be regarded as a repetitive inverted pendulum motion [9, 10, 11, 12]. Indeed, the motion of the whole body is left entirely to the inverted pendulum motion of the leg in the stance phase

[7, 8]. As shown in Fig. 1a, the forward motion of the body is naturally decelerated and accelerated in the first and second halves of the stance phase, respectively. As shown in Fig 1b, if the knee joint of the leg is flexed or extended beginning in the stance phase, the natural deceleration phase is shortened or lengthened, respectively. Thus, the knee joint angle ϕ at the BSP significantly affects the total propulsive force for a step; the modulation of the knee joint angle ϕ at the BSP causes the propulsive force for locomotion to be modulated. This suggests that a stepping motion perturbed by external force can be neutralized near the BSP by modulation of the knee joint angle ϕ at the BSP, which may result in the *reinitialization* of the system. Modeling studies on bipedal locomotion [7] have shown that the system state when perturbed by a strong momentary external force applied at the hip joint can be balanced during the motion of several steps controlled by knee angle modulation at the BSP.

Animals are known neurophysiologically to initiate locomotion after posture has been adjusted and posture control is involved until the completion of locomotion [13, 14]. Locomotion control thus involves not only generation of a stepping rhythm but also posture control. Clinical trials on human patients [3, 4] have also shown that while spinal cord activity, including the locomotor CPG, can induce the legs to step rhythmically (on a bed), this motion is definitely different from locomotion where the body walks forward while maintaining balance. Neurophysiological experiments on cats showed that by receiving proprioceptive sensory signals through the mossy fibers system, Purkinje cells in the cerebellum can modulate the activity of the motor neurons in the spinal cord. When the paravermal part of lobules IV and V or the vermal part of lobule V in the cerebellum is partially cooled, excessive flexion and extension (respectively) of the legs is induced around the BSP, which results in a failure to walk [15, 16]. The activity of Purkinje cells in the paravermal part of lobule V or in the vermal zones becomes significantly high around the BSP [17, 18]. These results indicate that locomotor control is achieved with the participation of leg extension control at the BSP by the Purkinje cells [19, 20]. Moreover, as mentioned in the previous paragraph, the biomechanics of bipedal locomotion suggest that modulation of the leg extension at the BSP is the key to achieving a gait that is adaptive to external perturbations. In human bipedal walking, leg extension basically depends only on the angle of the knee joint. In this study, we focus on the relationship between modulation of the knee angle and adaptation to successive external perturbations.

When walking in a crowded street, humans may collide with each other. Likewise, at a gap between tall buildings, a gust of wind may push a walking person strongly backwards. We deal with such cases in this study, and investigate a mechanism for providing instant adaptability of gait in response to successive unpredictable perturbations. Even if such perturbations have crucial affects on the walking conditions, the gait can

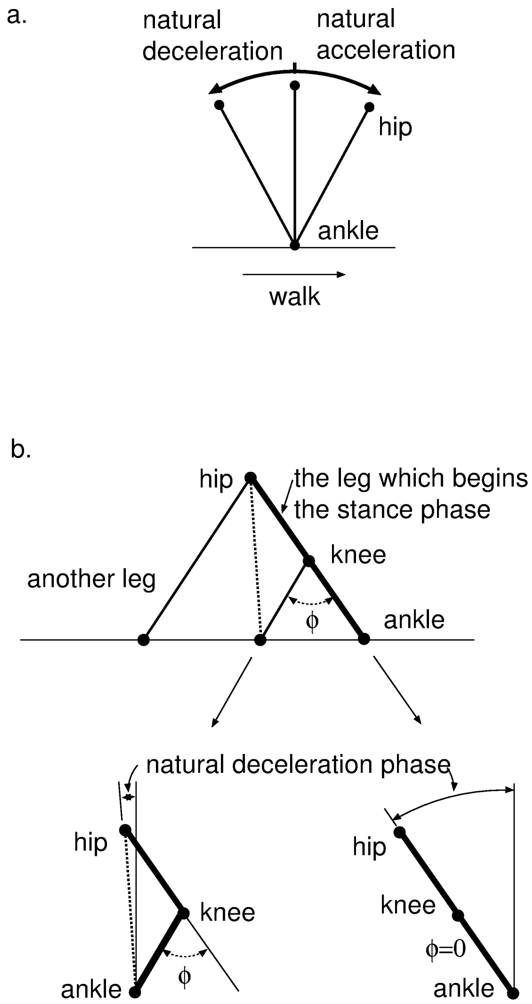

Figure 1. A biomechanical view of bipedal walking. (a) During walking, the whole body takes a step forward utilizing the inverted pendulum motion of the leg in the stance phase. The forward motion of the body is naturally decelerated and accelerated in the first and second halves of the stance phase, respectively. (b) The length of the natural decelerationchanges depending on the knee angle ϕ at the beginning the stance phase. The thick line indicates the leg which begins in the stance phase and ϕ represents the knee angle.

be maintained if each individual step adapts completely to the given conditions. We suppose that such adaptive stepping motion can be generated by modulation of the knee joint angle at the BSP, *i.e.*, by the *initial state* coordination. Initial state coordination has been applied to a single change of condition [7, 8] but not to successive changes of condition. Thus we call initial state coordination in response to new conditions *reinitialization*. In this paper, we demonstrate using computer simulations of our walking model that human gait can adapt to successive perturbations by reconstructing the constraints, *i.e.*, by *reinitialization* at every step. We understand the process of *reinitialization* from a theoretical perspective in terms of dynamic systems. The concept of *reinitialization* entirely includes the structure of the conventional walking model [2] which is adaptive to weak perturbations, and extends its adaptability to include strong perturbations. From a theoretical perspective, the constraints define a hyperplane in phase space and *reinitialization* denotes the restarting of the orbit from the hyperplane. This is explained in detail in the next subsection where gait generation is considered as a dynamic system.

§ 1.2. Theoretical view of reinitialization

Our model is similar to that proposed by [7, 8]. The model is constructed by adding a posture controller to the coupled system composed of a CPG and the body. The activity of the CPG, which is made up of coupled Bonhoeffer-van der Pol (BVP) neurons [21], is represented by differential equations of a vector $\mathbf{u} = (u_1, v_1, \cdots, u_{12}, v_{12})$ which describe 12 neurons since the BVP equation describing a single neuron has two variables. The motion of the body is represented by differential equations of a vector $\mathbf{x} = (x_1, \cdots, x_6)$ describing five links (*cf.* Fig. 8). The coupled system composed of the CPG and body can generate a basic gait. An outline of the time evolution of the coupled system can be described as follows,

$$\begin{cases} \dot{\mathbf{u}}(t) = G_1(\mathbf{u}, \mathbf{E}(\mathbf{x})), \\ \ddot{\mathbf{x}}(t) = G_2(\dot{\mathbf{x}}, \mathbf{x}, \mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}}), F(t)), \end{cases} \tag{1.1}$$

where t denotes the time, $\mathbf{E}(\mathbf{x})$ indicates $\mathbf{R}^{12}$ -valued feedback from the body to the CPG, $\mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}})$ indicates $\mathbf{R}^6$ -valued torque generated by the CPG, and $F(t)$ indicates a scalar perturbation. When the perturbation $F(t)$ on the coupled system is strong, the coupled system immediately converges to an equilibrium, *i.e.*, the walking system falls.

The behavior of the coupled system can be perceived as the movement of a point $(\mathbf{u}(t), \mathbf{x}(t), \dot{\mathbf{x}}(t))$ in the phase space $X := \mathbf{R}^{36}$. The variable x_1 is the position of the hip on the horizontal axis. Here, we set the vectors $\tilde{\mathbf{x}}(t) := (x_2(t), \cdots, x_6(t))$, and

$P(t) := (\mathbf{u}(t), \tilde{\mathbf{x}}(t), \dot{\mathbf{x}}(t))$ which moves in $\tilde{X} := R^{35}$. Two falling states [1] correspond to two lines parallel to x_1- axis in X. The images of the normal projection of the two lines onto $\tilde{X}$ represent two stable equilibriums in $\tilde{X}$. The image of the normal projection onto $\tilde{X}$ of the orbit in X corresponding to a basic gait is a limit cycle attractor. The behavior of $\dot{x}_1$ in the orbit is cyclic. Fig. 2a illustrates the image of the normal projection of the dynamics onto $\tilde{X}$. The attractor basin of the limit cycle appears between the two attractor basins of the stable equilibrium.

As mentioned in Section 1.1, the posture at the beginning of the stance phase (BSP) is important for locomotor control. The heights of the left and right feet, *i.e.*, $y_l(t)$ and $y_r(t)$, are expressed by the following equations:

$$
(1.2) \qquad \begin{cases} y_l(t) = x_2(t) - l_1 \cos x_3(t) - l_2 \cos x_4(t), \\ y_r(t) = x_2(t) - l_1 \cos x_5(t) - l_2 \cos x_6(t). \end{cases}
$$

As indicated in Fig. 8, x_2 is the height of the hip joint, x_3 and x_4 are the angles of the thigh and shank of the left leg, respectively, x_5 and x_6 are the angles of the thigh and shank of the right leg, respectively, and l_1 and l_2 are for both legs the lengths of the thigh and shank, respectively. The heights of the feet $y_l(t)$ and $y_r(t)$ become 0 only at the moment when the stance phase begins or completes because they become lower than of the height of the ground during the stance phase in our model. The inequalities $x_3(t_l) > 0$ and $x_5(t_r) > 0$ represent the situations when the left and right legs, respectively are flexing, *i.e.*, near the BSP. Thus the times of the left and right leg BSPs, *i.e.*, t_l and t_r, are determined by the following equations and inequalities :

$$
(1.3) \qquad \begin{cases} y_l(t_l) = 0 \quad and \quad x_3(t_l) > 0, \\ y_r(t_r) = 0 \quad and \quad x_5(t_r) > 0. \end{cases}
$$

Fig. 2 illustrates the projected dynamics in $\tilde{X}$ instead of the original dynamics in X. As shown in Fig. 2b, a perturbation is translated into a shift of the vector field. Because the attractor on the shifted vector field leads the system to approach it, the orbit deviates from the original limit cycle attractor. A large perturbation causes the solution to cross over the original separatrix. We have called this the *surfacing of the neutral state* just before the BSP [8]. Just after the perturbation, the original vector field returns, as shown in Fig. 2c. At this time, the system is on the side in which a stable equilibrium exists. The system therefore converges to a stable equilibrium, *i.e.*, the walking system falls.

[1] Since the walking body is usually modeled in the sagittal plane in theoretical studies, there are two falling states, forwards and backwards.

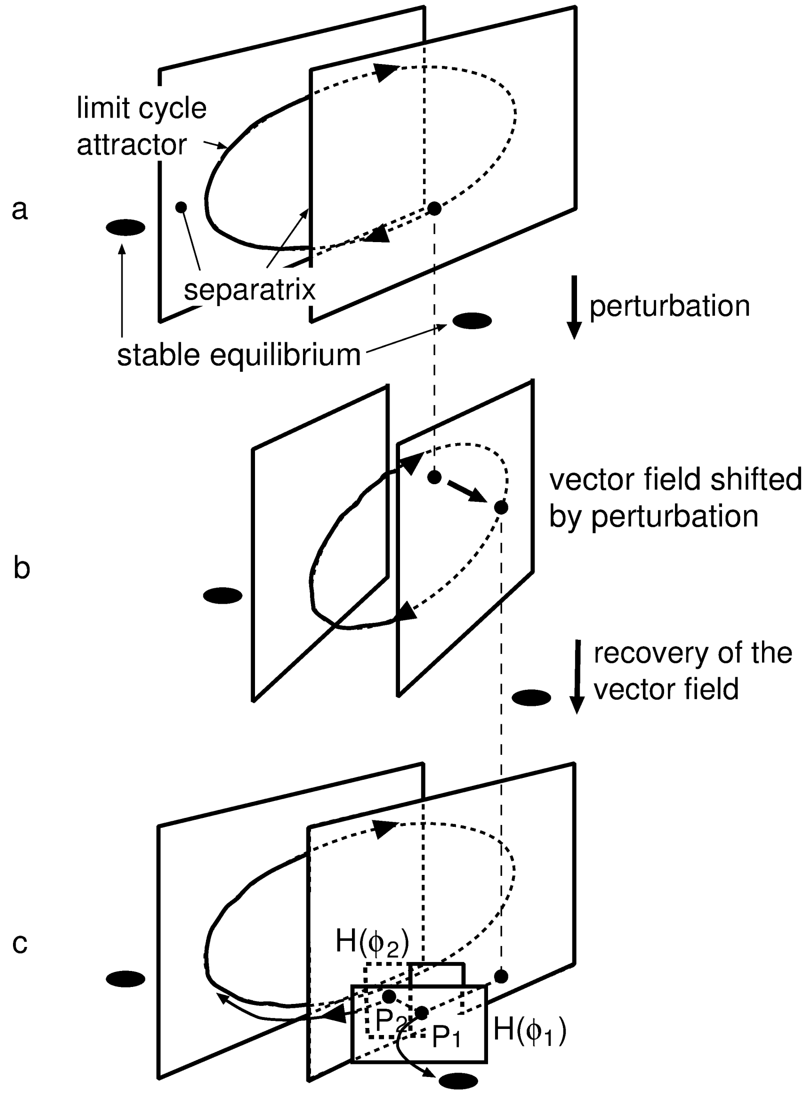

Figure 2. Schematic diagram showing the projection of the walking system's behavior in the phase space X onto the phase space $\tilde{X} := \mathbf{R}^{35}$. (a) A gait cycle and two falling states are translated into a limit cycle attractor and two stable equilibriums, respectively. (b) Schematic diagram illustrating the system motion when a perturbation affects the system ($0.1s$). The perturbation is translated into a shift of the vector field. Because the attractor on the shifted vector field causes the system to approach it, the system motion deviates from the original limit cycle orbit. A large perturbation causes the system to cross over the original separatrix. (c) Schematic diagram illustrating reinitialization. Even if the system is strongly perturbed, the gait can be maintained. At a position P_1 occurring on the path the system takes while approaching a stable equilibrium, the posture controller causes the system state to be reset from P_1 to another position P_2 near the separatrix and on the side where the limit cycle exists. At P_2, the system is immediately attracted to the limit cycle. The positions P_1 and P_2 are on the hyperplanes $H(\phi_1)$ and $H(\phi_2)$, respectively.

On the other hand, gait is recovered from disturbed motions caused by strong perturbations in the model, through the function of the posture controller. The posture controller consists of two BVP neurons. The activity of the posture controller is described by differential equations of a vector $\mathbf{u}_p = (u_{p1}, v_{p1}, u_{p2}, v_{p2})$ describing two neurons. Being entrained by the rhythm of the CPG, around the BSP the posture controller outputs an action potential which gives the posture modulation torque $\mathbf{T}_{BSP}(\mathbf{u}_p, \mathbf{x}, \dot{\mathbf{x}}, \phi_c(t)) = (T_{BSPl}, T_{BSPr})$, which is defined in (2.4). $\phi_c(t)$ is a scalar function depending on the perturbation $F(t)$ and determines the body posture at the BSP. This is expressed in detail below, in (1.5). The interaction between the posture controller and the coupled system is summarized as follows.

$$
(1.4) \qquad \begin{cases} \dot{\mathbf{u}}_p(t) = f(\mathbf{u}_p, \mathbf{u}) \\ \dot{\mathbf{u}}(t) = f(\mathbf{u}, \mathbf{E}(\mathbf{x})), \\ \ddot{\mathbf{x}}(t) = g(\dot{\mathbf{x}}, \mathbf{x}, \mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}}), F(t), \mathbf{T}_{BSP}(\mathbf{u}_p, \mathbf{x}, \dot{\mathbf{x}}, \phi_c(t))). \end{cases}
$$

At some time $t = t_1$, occurring a little before the BSP of the left leg at time t_l, the posture controller neuron $u_{p1}(t)$ begins to fire ($g(u_{p1}(t)) > 0$). The position in $\tilde{X}$ at time t_1 is defined as $P_1 \in \tilde{X}$. When a given perturbation $F(t)$ is strong, P_1 is in the attractor basin of a stable equilibrium, as shown in Fig. 2c. The firing of $u_{p1}(t)$ can cause the production of the torque T_{BSPl}. During resting of neuron activity $u_{p1}(t)$ ($g(u_{p1}(t)) = 0$), and T_{BSPl} are zero. The torque T_{BSPl} forces the solution to be reset from P_1 to another point P_2 on the attractor basin of a limit cycle. The time when the solution arrives at P_2 is defined as t_2. That is, $P(t_1) = P_1$ and $P(t_2) = P_2$, where $P(t)$ denotes the point on the projected orbit in $\tilde{X}$ at time t. The torque T_{BSPl} is configured to be produced until the solution arrives at P_2. Here, the position P_2 corresponds to the body posture at the BSP. Therefore, the time t_2 is equivalent to the time of the BSP t_l, *i.e.*, $t_2 = t_l$. From P_2, the projected system is immediately attracted to a limit cycle. Just after the BSP, the firing of the posture controller neuron ends and $g(u_{p1}) = 0$ (see (2.4)). This resetting process from P_1 to P_2 corresponds to a decrease in the deceleration period of the stance phase (see Fig. 1). This process, which is referred to as *reinitialization*, enables the projected system to restart its dynamic state from P_2. After about a half period from this point, again around the BSP of the right leg at $t_r(= t_2)$, a procedure similar to that above is brought about by the firing of $u_{p2}(t)$ and the torque this yields, T_{BSPr}.

We now present an interpretation of this reinitialization process within the framework of the dynamic system projected in $\tilde{X}$. Let us denote the image of the projection of $\mathbf{x} \in X$ onto $\tilde{X}$ by $\tilde{\mathbf{x}}$, and define a hyperplane as follows.

$$
(1.5)\quad \begin{aligned}
&H(\phi) = \{(\mathbf{u}, \tilde{\mathbf{x}}, \dot{\mathbf{x}}) \in \tilde{X}, \\
&\alpha_1 x_2 + \cdots + \alpha_5 x_6 + \alpha_7 \dot{x}_1 + \cdots + \alpha_{11} \dot{x}_6 + \alpha_{12} u_1 + \alpha_{13} v_1 + \cdots \\
&+ \alpha_{34} u_{12} + \alpha_{35} v_{12} = \phi\} \subset \tilde{X}, \\
&(\alpha_1, \cdots, \alpha_{35} : constants)
\end{aligned}
$$

The coefficients $\alpha_1, \cdots, \alpha_{35}$ are assumed to be set so that the hyperplane $H(\phi)$ is transverse to the separatrix. Describing such a hyperplane which includes the point P_1 as $H(\phi_1)$, the shifted position P_2 belongs to a hyperplane $H(\phi_2)$ which is parallel to $H(\phi_1)$. The constant ϕ_2 is determined by the perturbations applied. The process of shifting from P_1 to P_2 follows the dynamics of the posture controller and the coupled system composed of the CPG and the body, and the perturbations applied, namely (1.4). After the shift to P_2 is completed, the orbit is released from the dynamics of the posture controller and restarts from P_2 on $H(\phi_2)$ following the dynamics of the coupled system composed of the CPG and the body. For the condition that the perturbation strength is zero, $\phi_1 = \phi_2$. This is the procedure we call *reinitialization*.

The constraint produced following a given condition (perturbation) yields the dynamic behavior of the system suited to the condition. The system abandons the dynamics and recomputes the constraint by revising the angle ϕ_2 for newly applied conditions. The reproduction of the constraint, *i.e.*, the *reinitialization*, can thus guarantee instant adaptability to unpredictable environments.

The mechanism of flexible locomotor control has never been investigated in terms of the repetitive reproduction of constraints. According to neurophysiological information, the posture controller should be modeled so that it yields *reinitialization* and integrates it into the coupled dynamic model. We validate the effectiveness of *reinitialization* with such a model through computer simulations.

§ 2. The walking model

The model is constructed by adding a posture controller [7] to the two coupled dynamic processes [2]. The model thus consists of the body and the neural system composed of the CPG and the posture controller, as shown in Fig. 3.

The body consists of an interconnected chain of 5 rigid links in the sagittal plane, as shown in Fig. 8. The motion of the body is represented by differential equations of a vector $\mathbf{x} = (x_1, \cdots, x_6)$ describing five links (*cf.* Fig. 8), *i.e.*, mass point positions of 1 link and inertial angles of 4 links. The equations are described according to the Newton-Euler method (see Appendix A). They are represented in summarized form by the third equation of (1.4).

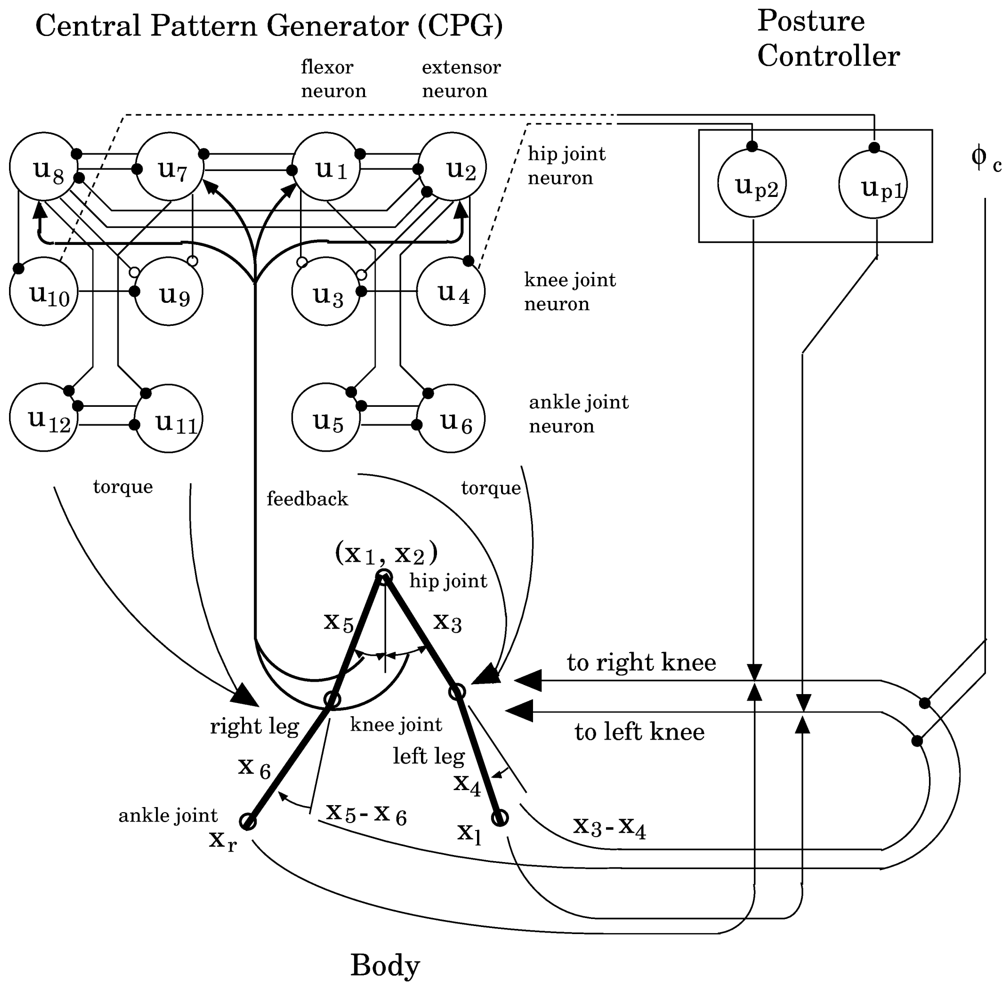

Figure 3. The walking system consists of the posture controller (neurons $p1$ to $p2$) and the coupled system composed of the central pattern generator (CPG) (neurons 1 to 12) and the body. ◦ and • denote excitatory and inhibitory Connections, respectively. The motion of the hip, the knee and the ankle in the left leg is governed by neurons $1-2$, $3-4-p1$ and $5-6$, respectively. Similarly, the motion of the joints in the left leg is governed by neurons 7 to 12 and $p2$. Odd-numbered neurons and even-numbered neurons in the CPG controller represent flexors and extensors, respectively. The posture controller provides the equilibrium angle ϕ_c for the knee joint at the BSP.

We construct the CPG for bipedal locomotion using the BVP neuron model [21]. It is composed of 12 neurons that have both excitatory and inhibitory connections. The excitatory and inhibitory connections between the neurons can make the relative phases of neuronal activity synchronous and opposite, respectively. Each neuron induces a torque at a specific joint. The CPG is represented by the following differential equations.

$$(2.1)\qquad \begin{cases} \tau_i \dot{u}_i(t) = u_i(t) - v_i(t) - u_i(t)^3/3 + \sum_{j=1}^{12} w_{ij} f(u_j(t)) \\ \qquad\qquad + u_0 + E_i(\mathbf{x}(t)), \\ \tau_i' \dot{v}_i(t) = u_i(t) + a - b v_i(t), \\ f(u) = max(0, u), \qquad (i = 1, \cdots, 12) \end{cases}$$

where u_i is the potential of the ith neuron; v_i is responsible for the accommodation and refractoriness of the ith neuron; w_{ij} is the connecting weight from the ith neuron to the jth neuron; and τ_i and τ_i' are the time constants of the inner state and the accommodation and refractory effects, respectively. $f(u_i(t))$ is the output of the ith neuron; and E_i is the feedback from the body, a and b are positive constants. The natural frequency of each joint's neuron(τ_i, τ_i') is set to a value close to the natural frequency of the joint [2], and the input u_0 activates all of the neurons by the constant value γ, as follows.

$$(2.2)\qquad u_0(x_2(t)) = \begin{cases} 0 & \text{for} \quad x_2(t) \leq \Gamma, \\ \gamma & \text{otherwise.} \end{cases}$$

The variable $x_2(t)$ indicates the height from the ground to the hip position. Γ is a constant parameter. Only when the system is falling is $x_2(t) \leq \Gamma$, and u_0 makes the state of all the neurons equal to their resting state. The resting state of all the neurons makes the system converge to a stable equilibrium.

To modify the basic gait, the posture controller modulates the knee joint angle at the BSP by providing it with the equilibrium angle ϕ_c occurring just before the BSP. The posture controller also consists of two BVP neurons, *i.e.*, u_{p1} and u_{p2}. Neurons u_{p1} and u_{p2} govern the modulation of the left and right knee joint angles, respectively. Their equations are as follows.

$$(2.3)\qquad \begin{cases} \tau_{p1} \dot{u}_{p1}(t) = u_{p1}(t) - v_{p1}(t) - u_{p1}(t)^3/3 + w_{4,p1} f(u_4(t)) + u_0, \\ \tau_{p1}' \dot{v}_{p1}(t) = u_{p1}(t) + a - b v_{p1}(t), \\ \tau_{p2} \dot{u}_{p2}(t) = u_{p2}(t) - v_{p2}(t) - u_{p2}(t)^3/3 + w_{10,p2} f(u_{10}(t)) + u_0, \\ \tau_{p2}' \dot{v}_{p2}(t) = u_{p2}(t) + a - b v_{p2}(t), \\ f(u) = max(0, u). \end{cases}$$

The terms $w_{4,p1}f(u_4(t))$ and $w_{10,p2}f(u_{10}(t))$ represent the entrainment from the 4th neuron in the CPG to the u_{p1} neuron, and from the 10th neuron in the CPG to the u_{p2} neuron, respectively. Through this entrainment each neuron outputs its action potential around each BSP. The equilibrium angle produces the following posture modulation torque in the left and right knee joints, $\mathbf{T}_{BSP} = (T_{BSPl}, T_{BSPr})$. These are defined as follows.

$$
\tag{2.4}
\begin{cases}
T_{BSPl} = f_m(T_l - \alpha f(u_{p1})), \\
T_{BSPr} = f_m(T_r - \alpha f(u_{p2})), \\
T_l = g(u_{p1})g(y_l)g(x_3 - x_4 - \phi_c)(p_{b1}(x_3 - x_4 - \phi_c) - p_{b2}(\dot{x}_3 - \dot{x}_4)), \\
T_r = g(u_{p2})g(y_r)g(x_5 - x_6 - \phi_c)(p_{b1}(x_5 - x_6 - \phi_c) - p_{b2}(\dot{x}_5 - \dot{x}_6)), \\
y_l = x_2 - l_1 \cos x_3 - l_2 \cos x_4, \\
y_r = x_2 - l_1 \cos x_5 - l_2 \cos x_6, \\
f(u) = max(0, u), \\
f_m(z) = \begin{cases} \delta & \text{for} \quad z > \delta, \\ z & \text{for} \quad |z| \le \delta, \\ -\delta & \text{for} \quad z < -\delta, \end{cases} \qquad g(z) = \begin{cases} 0 & \text{for} \quad z \le 0, \\ 1 & \text{otherwise.} \end{cases}
\end{cases}
$$

u_{p1} and u_{p2} are the neuron potentials of posture controller governing the knee angle modulation in the left and right legs, respectively. These neurons only generate their action potentials around each BSP [8]. $g(u_{p1})$ and $g(u_{p2})$ output 1 during the firing periods of u_{p1} and u_{p2}, *i.e.*, $u_{p1} > 0$ and $u_{p2} > 0$, respectively. They output 0 during the resting periods of u_{p1} and u_{p2}, *i.e.*, $u_{p1} \le 0$ and $u_{p2} \le 0$, respectively. y_l and y_r represent the distance from the ground to the ankle joint of the left and right legs, respectively (1.2). $g(y_l)$ and $g(y_r)$ output 1 during the swing phase of the left and right legs, respectively, and output 0 during the stance phase of the left and right legs, respectively. (x_1, x_2) represents the position of the hip joint, while x_3 and x_4 represent the angle of the left shank and thigh, respectively. x_5 and x_6 represent the angle of the right shank and thigh, respectively. α, p_{b1}, and p_{b2} are constant coefficients. Because $(-\alpha f(u_{p1}))$ and $(-\alpha f(u_{p2}))$ function as antagonists acting against T_l and T_r, respectively, the knee angles are forced to converge to the equilibrium angle ϕ_c during the firing periods of u_{p1} and u_{p2}. $f_m(z)$ is a function that restricts the amplitude of the torque needed to maintain walking to a realistic level, δ. This function may be realized by coactivation of the agonists and the antagonists at the joint. Thus the torques T_{BSPl} and T_{BSPr} modulating the left and right knee angles are produced during the periods when $g(u_{p1})g(y_l) > 0$ and $g(u_{p2})g(y_r) > 0$, respectively, *i.e.*, both knee joints are modulated during both firing of the ipsilateral neuron in the posture controller and the swing phase of the ipsilateral leg. The entrainment from the CPG to the posture controller is designed so that the posture controller neuron begins to fire a little before the ipsilateral leg ends the swing phase. The production of the posture modulation torque is therefore triggered at $t = t_1$ by the firing of the posture controller neuron and is stopped at $t = t_l(= t_2)$ or $t = t_r(= t_2)$ by the beginning of the stance phase (BSP). Consequently, as mentioned in Section 1.2, T_{BSPl} and T_{BSPr} are produced during the intervals from t_1 to t_l and t_r, respectively. Since the constant ϕ_c corresponds to ϕ_2 as described in Section 1.2, the posture modulation torques (T_{BSPl}, T_{BSPr}) force the knee angle at t_1 ($\phi_1 = x_3(t_1) - x_4(t_1)$ or $x_5(t_1) - x_6(t_1)$) to become $\phi_2 = \phi_c$ at t_2. Then, at t_l or t_r, the legs are released from this constraint by the equilibrium angle ϕ_2, and the dynamics of the coupled system restart from the BSP. The posture modulation torque ($\mathbf{T}_{BSP}$) thus implements *reinitialization*.

Receiving the sensory signals from the legs and the outputs from the CPG, the posture controller neurons govern the moderate extension of the legs in the BSP. It is known that Purkinje cells in the cerebellum receive proprioceptive sensory signals and the outputs of the CPG [22, 23]. Also, as mentioned in introduction, Purkinje cells in the cerebellum strongly participate in the moderate extension of the legs at the BSP. The posture controller we present may thus correspond to the action of Purkinje cells in lobule V of the paravermal and the vermal areas of the cerebellum, *etc.*

§ 3. Simulations and Results

During walking, humans may collide many times with each other in a crowded street. At gaps between tall buildings, a gust of wind may also push a walking person backward. We considered the situation when such unpredictable and successive external forces applied to the body cease. The successive perturbations may end immediately, or may continue for some time so that perturbed motions overlap and may result in failures to walk. In such cases, it is necessary to compensate immediately for perturbations, and to recover normal walking within a single step. It was tested whether or not such flexible locomotor control can be realized by the *reinitialization* strategy.

We assume that an external force makes a backwards effect on the hip joint over an interval of $0.1[s]$ during walking. We refer to this force simply as a perturbation. We define normal walking to be walking generated without posture modulation, *i.e.*, walking generated using $\phi_c = 0$ at the BSP. Firstly, it was confirmed that for steady walking achieved in the normal case, *i.e.*, without the posture controller ($\phi_c = 0$ at the BSP), perturbations smaller than $280[N](Newtons)$ could be accommodated. Secondly, a successive sequence of perturbations was prepared. The perturbation level in this sequence (under $500[N]$)is realistic from the following perspective. The usual speed of an adult human walk is about $2[m/s] \cong 6[k/h]$. For a walking human with a weight of $70[kg]$, when a speed of $2[m/s]$ is decelerated to $1.5[m/s]$ by a perturbation with duration $0.1[s]$, the external force F may be estimated as $F = 70 \times (2.0 - 1.5)/0.1 = 350[N]$.

A sequence of corresponding equilibrium angles for the knee joints ϕ_c at the BSP was thus designed. In simulations using these conditions, the model was clearly shown to overcome the external perturbations.

As shown in Fig. 4, we find that the walking pattern changes at every step as long as the successive perturbations are applied. Fig. 5 shows the neuron activities of the posture controller, the angular motion of the knees, the prepared sequence of ϕ_c, and the time series of the successive perturbations. The series of ϕ_c is only effective during the firing of the posture controller neurons (u_{p1}, u_{p2}). Even in the case that the series of perturbations suddenly ceases, the model could immediately recover normal walking. When the series of perturbations ceases, walking could be maintained by not modulating the knee angle at the BSP ($\phi_c = 0$). For example, even when only the first perturbation takes effect (no second or third perturbations are applied), the model can recover normal walking immediately by setting ϕ_c to 0. This demonstrates that each of the values in the prepared sequence of ϕ_c can induce each of the perturbations to be completely compensated within one step. During the firing of the posture controller neurons u_{p1} and u_{p2}, the following process may be seen in the motion of the variables. The firing of the posture controller neurons u_{p1} and u_{p2} causes the production of the torques T_{BSPl} and T_{BSPr}, respectively. The torques T_{BSPl} and T_{BSPr} cause the knee angles $x_3 - x_4$ and $x_5 - x_6$ to attain at ϕ_c within a very short time. Next, the angles of the knees change a little near ϕ_c until the end of torque production, *i.e.*, until the BSP (see Fig. 5). Each of other variables of the system changes significantly within its usual range during the firing of the posture controller neurons. After the firing of the posture controller neurons u_{p1} and u_{p2}, the respective angles of the knee joints $x_3 - x_4$ and $x_5 - x_6$ also change significantly.

Setting ϕ_c to a constant value also facilitates overcoming of the successive perturbations. However, the range of such a constant value of ϕ_c was somewhat limited, and it is too difficult to find such a constant value. This result shows that changing the system state at the BSP is sufficient to overcome unpredictably fluctuating successive perturbations. The *reinitialization* process was thus shown to have an affect on unpredictably fluctuating successive perturbations.

Since the system overcomes perturbations by forming the knee joint posture at the BSP, this strategy generates a load on the knee joint at the BSP. The torque generated around the BSP is almost limited to the active torque (actively generated torque), and the strategy is realized by the active torque. We therefore check the level of the active torque.

The active torque at the knee joint of the model during walking was computed. The active torques of the left and the right legs are given by T_{r2} and T_{r5}, respectively (see Appendix). Figure 6 shows sequential changes in the active torque of the left knee

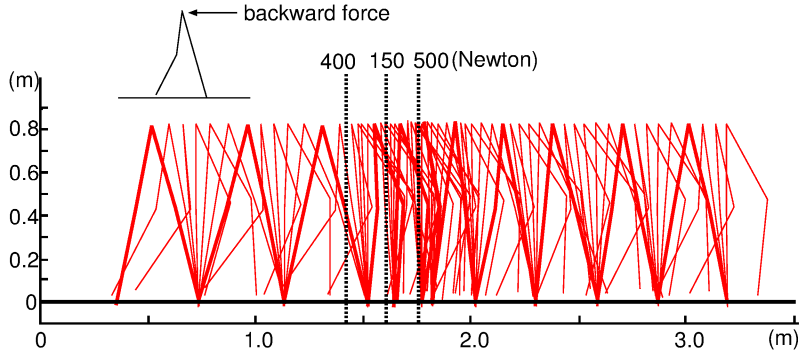

Figure 4. Stick figure of the walking motion adapting to successive perturbations (backward forces) affecting the hip joint. The dotted line denotes the position of the hip joint on the horizontal axis when each of the perturbations is applied. This motion is generated using the planned series of ϕ_c values which is shown in Fig. 5. Re-establishing the leg posture at the BSP just after a perturbation enabled the walking system to overcome this series of strong perturbations.

joint (T_{r2}). The model realized normal walking using a torque level within about 0.1 [torque $[Nm]$/weight $[N]$],since the body weight was assumed to be $70[kg] \cong 700[N]$. This agrees with experimental results obtained from biomechanical studies of normal human walking [24]. Adapting the gait to the perturbations required just over one and a half times as much torque as the maximum torque level of normal walking, $0.1[Nm/N]$. Similar results to this were also found in the right knee joint torque level. On the other hand, the torque produced at the other joints of the model was always within a realistic level as revealed by biomechanical studies [24]. That is, the sequential values of the active torque at the hip and ankle joints during steady walking were within $0.15[Nm/N]$ and $0.2[Nm/N]$, respectively. This torque level was not affected by the perturbations (data not shown).

§ 4. Reinitialization interpreted from the perspective of dynamic systems

The process of *reinitialization* may be viewed theoretically according to the motion of the model. Since bipedal locomotion systems have a bilaterally symmetrical structure, the model executes the reinitialization process twice per period of motion. We focus on the system motion in the half period that includes the BSP of the left leg. Figure 7 is a schematic diagram illustrating the image in $\tilde{X}$ of the process which is considered to be reproduced exactly during simulations of the model.

Before the BSP, the motion of the system obeys the dynamics of the coupled system (1.1) and perturbation applied. The onset of the action potential of u_{p1} defines $H(\phi_1) \subset \tilde{X}$, which the perturbed orbit intersects at P_1.

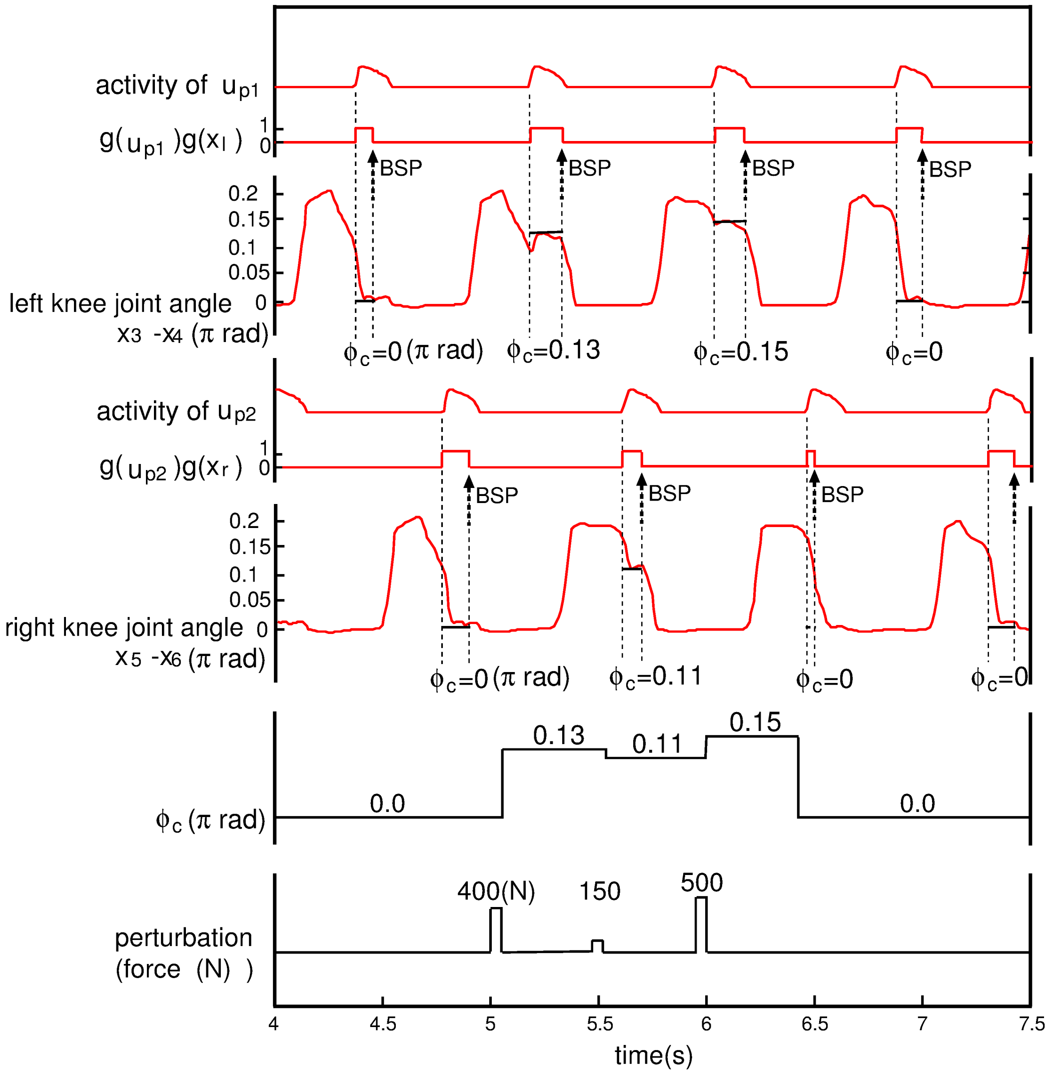

Figure 5. The motion of the knee joint angles around the period of successive perturbations. The series of equilibrium angles ϕ_c prepared for the series of perturbations only works when the firing of the posture controller neuron falls during the swing phase, *i.e.*, when $g(u_{p1})g(x_l) > 0$ and $g(u_{p2})g(x_r) > 0$ (see (2.4)) for the left and right knees, respectively. At the BSP the knee joints are released from the equilibrium angle constraint ϕ_c.

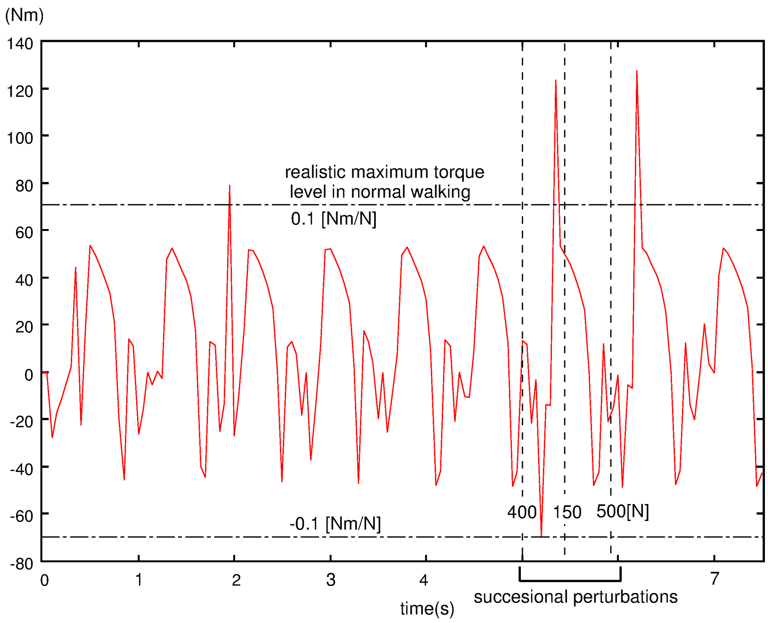

Figure 6. Sequential changes of the torque acting in the left knee joint (T_{r2} in the appendix) in the case where the hip is perturbed by external forces. The dotted line denotes the times when perturbations are applied.

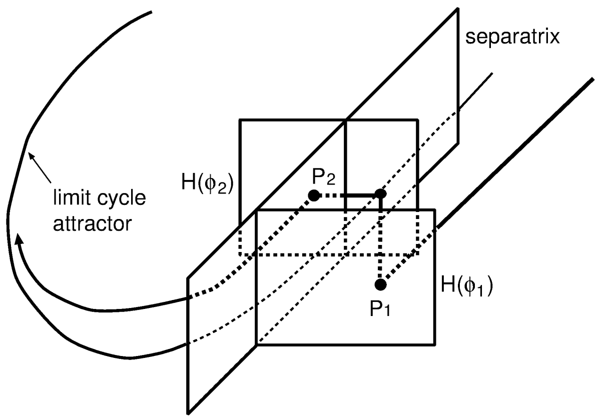

Figure 7. Schematic diagram illustrating the image in $\tilde{X}$ of the reinitialization process which is considered to be reproduced exactly in simulations of the model. The shift of the moving point $P(t)$ from P_1 to P_2 can be understood to embody the following two characteristics. 1) First the moving point $P(t)$ arrives at $H(\phi_2)$ in a very short time. Next, 2) the point $P(t)$ moves along $H(\phi_2)$ until it arrives at P_2. Arriving at P_2, the orbit immediately restarts from this position, obeying the dynamics of the coupled system. The orbit is then attracted by the limit cycle attractor.

(4.1) $$H(\phi_1) = \{(\mathbf{u}, \tilde{\mathbf{x}}, \dot{\mathbf{x}}) \in \tilde{X}; x_3 - x_4 = \phi_1\},$$

$\phi_1 = x_3(t_1) - x_4(t_1)$. Let $P(t) = (\mathbf{u}(t), \tilde{\mathbf{x}}(t), \dot{\mathbf{x}})(t) \in \tilde{X}$. As mentioned in Section 1.2, the time when the posture controller neuron $u_{p1}(t)$ begins to fire is defined as $t = t_1$ ($g(u_{p1}(t_1)) > 0$) such that $P(t_1) \in H(\phi_1)$. The position in $\tilde{X}$ at this time t_1 is defined as $P_1 \in \tilde{X}$. $P(t_1) = P_1$ where $P(t)$ denotes the point on the projected orbit in $\tilde{X}$ at time t. The perturbed orbit from P_1 to a stable equilibrium corresponds to the motion of the walking system in the case that u_{p1} is a zero function. Following (1.4), the torque T_{BSPl} is produced during the interval beginning with the onset of the action potential of $u_{p1}(t)$ ($g(u_{p1}(t_1)) > 0$) to the BSP of the left leg (2.4), *i.e.*, from t_1 to $t_2 = t_l$, where t_l is defined by (1.3). This torque forces the moving point $P(t)$ to shift from P_1 on $H(\phi_1)$ to P_2 on $H(\phi_2)$. The constant ϕ_2 is determined by the given perturbation ($\phi_2 = \phi_c$ according to (2.4)) so that the position P_2 is on the attractor basin of a limit cycle. Because the walking system does not only follow the u_{p1}-triggered force T_{BSPl} but also the dynamics of the coupled system while it moves during this time interval, the value of $H(\phi_2)$ automatically determines the shifted state $P_2(t_2)$. For the no perturbation condition, $P_1 = P_2$ because $\phi_1 = \phi_2$. In this model, ϕ_1 and ϕ_2 correspond to the knee angle $x_3 - x_4$. $H(\phi_1)$ and $H(\phi_2)$ are therefore parallel. By forcing only the knee joint angle $x_3(t_2) - x_4(t_2)$ to become ϕ_2, the torque T_{BSPl} compensates for the motion of all variables. From the simulation results, this shifting process can be understood to embody the following two characteristics. 1) First, the moving point $P(t)$ arrives on $H(\phi_2)$ in a very short time. Next, 2) the point $P(t)$ moves along $H(\phi_2)$ according to the dynamics of (1.4) until it arrives at P_2. Arriving at P_2, the orbit immediately restarts from this position, obeying the dynamics of the coupled system. The orbit is then attracted by the limit cycle attractor. The hyperplane $H(\phi_2)$ might be partially included in the attractor basin of the stable equilibrium. The characteristic motion 2) above is thus considered to ensure that the restart position of the orbit becomes P_2 and is truly in the attractor basin of a limit cycle (Fig. 7). Just after the BSP, the action potential of the posture controller neuron ceases ($g(u_{p1}(t_1)) \leq 0$).

The importance of *reinitialization* is summarized from a theoretical viewpoint as follows. 1) The values of ϕ_2 needed for correct reinitialization are limited. 2) In phase space, setting ϕ_2 corresponds to restricting the motion of the point $P(t)$ to a specific hyperplane. 3) This hyperplane is transverse to the separatrix. 4) The moving point $P(t)$ can only cross over the separatrix along a specific hyperplane. 5) Such a specific hyperplane is established by the posture controller.

We believe that the concept of *reinitialization* provides basic perspectives from which to investigate the mechanisms of flexible biological motor control. A theoretical understanding of reinitialization should be advanced.

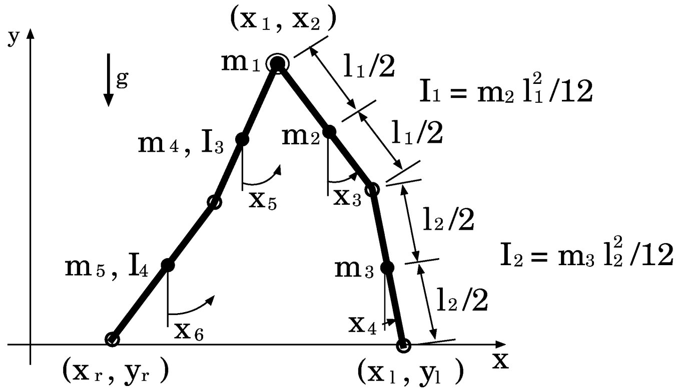

Figure 8. Model of a bipedal body as an interconnected chain of 5 rigid links (a point mass m_1 on the hip and 4 rigid bodies $I_i (i = 1, 4)$).

Acknowledgements

We would like to thank Dr Masato Iida and Mr Friedrich Meyer for helpful discussion and technical assistance.

Appendix

A. The equations of motion for the body

All variables and conventions correspond to those shown in Fig. 8. By using the Newton-Euler method also used in [2], the motion of the body can be written as follows.

$$P(\mathbf{x})\ddot{\mathbf{x}} = \mathbf{Q}(\mathbf{x}, \dot{\mathbf{x}}, \mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}}), \mathbf{T}_{BSP}(\mathbf{u}_p, \mathbf{x}, \dot{\mathbf{x}}, \phi_c), F),$$

therefore,

$$\ddot{\mathbf{x}} = [P(\mathbf{x})]^{-1}\mathbf{Q}(\mathbf{x}, \dot{\mathbf{x}}, \mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}}), \mathbf{T}_{BSP}(\mathbf{u}_p, \mathbf{x}, \dot{\mathbf{x}}, \phi_c), F),$$

where,

$$\mathbf{x} = (x_1, x_2, x_3, x_4, x_5, x_6)^{\mathrm{T}},$$

$$P(x) = (p_{ij})_{i=1\cdots 6, j=1\cdots 6},$$

$$\mathbf{Q}(\mathbf{x}, \dot{\mathbf{x}}, T_r(\mathbf{u}), F) = (q_1, q_2, q_3, q_4, q_5, q_6)^{\mathrm{T}},$$

$$\mathbf{T}(\mathbf{u}, \mathbf{x}, \dot{\mathbf{x}}) = (T_{r1}, T_{r2}, T_{r3}, T_{r4}, T_{r5}, T_{r6})^{\mathrm{T}},$$

$p_{11} = \sum_{n=1}^{5} m_n,$ $p_{21} = 0,$
$p_{12} = 0,$ $p_{22} = \sum_{n=1}^{5} m_n,$
$p_{13} = (0.5m_2 + m_3)I_1\cos(x_3),$ $p_{23} = (0.5m_2 + m_3)I_1\sin(x_3),$
$p_{14} = 0.5m_3l_2\cos(x_4),$ $p_{24} = 0.5m_3l_2\sin(x_4),$
$p_{15} = (0.5m_4 + m_5)l_3\cos(x_5),$ $p_{25} = (0.5m_4 + m_5)l_3\sin(x_5),$
$p_{16} = 0.5m_5l_4\cos(x_6),$ $p_{26} = 0.5m_5l_4\sin(x_6),$
$p_{31} = (0.5m_2 + m_3)I_1\cos(x_3),$ $p_{41} = 0.5m_3l_2\cos(x_4),$
$p_{32} = (0.5m_2 + m_3)I_1\sin(x_3),$ $p_{42} = 0.5m_3l_2\sin(x_4),$
$p_{33} = 0.25m_2l_1^2 + m_3l_1^2 + I_1,$ $p_{43} = 0.5m_3l_1l_2\cos(x_3 - x_4),$
$p_{34} = 0.5m_3l_1l_2\cos(x_4 - x_3),$ $p_{44} = I_2 + 0.25m_3l_2^2,$
$p_{35} = 0,$ $p_{45} = 0,$
$p_{36} = 0,$ $p_{46} = 0,$
$p_{51} = (0.5m_4 + m_5)I_3 cos(x_5),$ $p_{61} = 0.5m_5l_4\cos(x_6),$
$p_{52} = (0.5m_4 + m_5)I_3 sin(x_5),$ $p_{62} = 0.5m_5l_4\sin(x_6),$
$p_{53} = 0,$ $p_{63} = 0,$
$p_{54} = 0,$ $p_{64} = 0,$
$p_{55} = (0.25m_4 + m_5)l_3^2 + I_3,$ $p_{65} = 0.5m_5l_3l_4\cos(x_5 - x_6),$
$p_{56} = 0.5m_5l_3l_4\cos(x_6 - x_5),$ $p_{66} = 0.25m_5l_4^2 + I_4,$

$$\begin{aligned}
q_1 &= (0.5m_2 + m_3)l_1\sin(x_3)\dot{x}_3^2 + 0.5m_3l_2\sin(x_4)\dot{x}_4^2 \\
&\quad + (0.5m_4 + m_5)l_3\sin(x_5)\dot{x}_5^2 + 0.5m_5l_4\sin(x_6)\dot{x}_6^2 + F_{g1} + F_{g3} + F \\
q_2 &= -(0.5m_2 + m_3)l_1\cos(x_3)\dot{x}_3^2 - 0.5m_3l_2\cos(x_4)\dot{x}_4^2 \\
&\quad - (0.5m4 + m5)l_3\cos(x5)\dot{x}_5^2 - 0.5m_5l_4\cos(x_6)\dot{x}_6^2 + F_{g1} + F_{g2} - \textstyle\sum_{n=1}^{5} m_n g \\
q_3 &= 0.5m_3l_1l_2\sin(x_4 - x_3)\dot{x}_4^2 + F_{g1}l_1\cos(x_3) + F_{g2}l_1\sin(x_3) \\
&\quad - (m_2 + 2m_3)0.5gl_1\sin(x_3) + T_{rp1} + T_{r1} - T_{r2} - T_{r4} - T_{BSPl} \\
q_4 &= 0.5m_3l_1l_2\sin(x_3 - x_4)\dot{x}_3^2 - 0.5m_2gl_2\sin(x_4) + F_{g1}l_2\cos(x_4) \\
&\quad + F_{g2}l_2\sin(x_4) + T_{rp2} + T_{r2} - T_{r3} + T_{BSPl} \\
q_5 &= 0.5m_5l_3l_4\sin(x_6 - x_5)\dot{x}_6^2 - 0.5(m_4 + 2m_5)gl_3\sin(x_5) + F_{g3}l_3\cos(x_5) \\
&\quad + F_{g4}l_3\sin(x_5) + T_{rp3} + T_{r4} - T_{r5} - T_{r1} - T_{BSPr} \\
q_6 &= 0.5m_5l_3l_4\sin(x_5 - x_6)\dot{x}_5^2 - 0.5m_4gl_4\sin(x_6) \\
&\quad + F_{g3}l_4\cos(x_6) + F_{g4}I_4\sin(x_6) + T_{rp4} + T_{r5} - T_{r6} + T_{BSPr}
\end{aligned}$$

F is a perturbation. Horizontal and vertical forces on the ankles are given by:

$$F_{g1} = \begin{cases} -k_g\left(x_l - x_{l0}\right) - b_g\dot{x}_l & \text{for } y_l - y_g\left(x_l\right) < 0 \\ 0 & \text{otherwise,} \end{cases}$$

$$F_{g2} = \begin{cases} -k_g\left(y_l - y_{l0}\right) + b_g f\left(-\dot{y}_l\right) & \text{for } y_l - y_g\left(x_l\right) < 0 \\ 0 & \text{otherwise,} \end{cases}$$

$$F_{g3} = \begin{cases} -k_g\left(x_r - x_{r0}\right) - b_g \dot{x}_r & \text{for } y_r - y_g\left(x_r\right) < 0 \\ 0 & \text{otherwise,} \end{cases}$$
$$F_{g4} = \begin{cases} -k_g\left(y_r - y_{r0}\right) + b_g f\left(-\dot{y}_r\right) & \text{for } y_r - y_g\left(x_r\right) < 0 \\ 0 & \text{otherwise,} \end{cases}$$

where $y_g(x)$ is a function which represents the terrain. When the ground is level $y_g(x) = 0$. (x_l, y_l) and (x_r, y_r) represent the positions of the ankles, which are given by:

$$(x_l, y_l) = (x_1 + l_1 \sin x_3 + l_2 \sin x_4,\ x_2 - l_1 \cos x_3 - l_2 \cos x_4),$$
$$(x_r, y_r) = (x_1 + l_1 \sin x_5 + l_2 \sin x_6,\ x_2 - l_1 \cos x_5 - l_2 \cos x_6).$$

Passively generated torques at each joint are given by:

$$T_{rp1} = k_r f(x_4 - x_3) - b_r f(x_4 - x_3) - b(\dot{x}_3 - \dot{x}_5) - b(\dot{x}_3 - \dot{x}_4),$$
$$T_{rp2} = -k_r f(x_4 - x_3) + b_r f(x_4 - x_3) - b(\dot{x}_4 - \dot{x}_3) - b\dot{x}_4,$$
$$T_{rp3} = k_r f(x_6 - x_5) - b_r f(x_6 - x_5) - b(\dot{x}_5 - \dot{x}_3) - b(\dot{x}_5 - \dot{x}_6),$$
$$T_{rp4} = -k_r f(x_6 - x_5) + b_r f(x_6 - x_5) - b(\dot{x}_6 - \dot{x}_5) - b\dot{x}_6,$$

and where k and b are the positive constants.

$$f(z) = \begin{cases} 0, & \text{for } \quad z \le 0 \\ z & \text{otherwise.} \end{cases} \qquad g(z) = \begin{cases} 0, & \text{for } \quad z \le 0 \\ 1 & \text{otherwise.} \end{cases}$$

Actively generated torques at each joint are given by:

$$T_{r1} = p_1 f(u_1) - p_2 f(u_2) + T_{hl},$$
$$T_{r2} = p_3 f(u_2) - p_4 f(u_4) + T_{kl},$$
$$T_{r3} = p_5 f(u_5) - p_6 f(u_6) g(-y_l),$$
$$T_{r4} = p_1 f(u_7) - p_2 f(u_8) + T_{hr},$$
$$T_{r5} = p_3 f(u_9) - p_4 f(u_{10}) + T_{kr},$$
$$T_{r6} = p_5 f(u_{11}) - p_6 f(u_{12}) g(-y_r),$$

where,

$$T_{hl} = g(u_1) g(x_3 - x_5 - x_h)(p_{e1}(x_3 - x_5 - x_h) - p_{v1}(\dot{x}_3 - \dot{x}_5)),$$
$$T_{kl} = g(u_1) g(x_3 - x_4 - x_k)(p_{e2}(x_3 - x_4 - x_k) - p_{v2}(\dot{x}_3 - \dot{x}_4)),$$
$$T_{hr} = g(u_7) g(x_5 - x_3 - x_h)(p_{e1}(x_5 - x_3 - x_h) - p_{v1}(\dot{x}_5 - \dot{x}_3)),$$
$$T_{kr} = g(u_1) g(x_5 - x_6 - x_k)(p_{e2}(x_5 - x_6 - x_k) - p_{v2}(\dot{x}_5 - \dot{x}_6)),$$

where p_* are positive constants. The torques T_{hl} and T_{hr} roughly confine the maximum angle of the hip joint to a positive constant x_h around the BSP of both legs. The torques T_{kl} and T_{kr} roughly confine the maximum angle of the left and right knee joints to a positive constant x_k during each swing phase. The other equations and parameters used in the model presented here and those in the previous model [8] are identical.

The sensory feedback E_i to the ith neuron is given as follows:

$$\begin{cases} E_i(i = 1 \cdots 12) \ \ are \ \ given, \\ E_1 = E, \ \ E_2 = E', \ \ E_7 = E', \ \ E_8 = E, \ \ E_i = 0 \ (otherwise), \\ Here, \\ E = f(-x_3) - f(-x_5), \qquad E' = f(-x_5) - f(-x_3), \end{cases}$$

and,

$$f(z) = \begin{cases} 0, & \text{for} \quad z \le 0 \\ z & \text{otherwise.} \end{cases} \qquad g(z) = \begin{cases} 0, & \text{for} \quad z \le 0 \\ 1 & \text{otherwise .} \end{cases}$$

B. Simulation parameters

1. Body

$m_1 = 48.0$ kg, $m_2 = 7.0$ kg, $m_3 = 4.0$ kg,
$m_4 = 7.0$ kg, $m_5 = 4.0$ kg,
$l_1 = 0.4$ m, $l_2 = 0.5$ m, $l_3 = 0.4$ m,
$l_4 = 0.5$ m, $I_i = m_{i+1}{l_i}^2/3$ kgm^2 $(i = 1, 2, 3, 4)$,
$k_g = 30000.0$ kg/s^2, $k_r = 2000.0$ kg/s^2, $b_g = 3000.0$ kg/s^2,
$b_r = 200.0$ kg/s^2, $b = 1.0$ kg/s^2,
$p_1 = 19.0$ kg rad s^{-2}V^{-1}, $p_2 = 19.0$ kg rad s^{-2}V^{-1}, $p_3 = 24.5$ kg rad s^{-2}V^{-1},
$p_4 = 19.0$ kg rad s^{-2}V^{-1}, $p_5 = 18.0$ kg rad s^{-2}V^{-1}, $p_6 = 5.0$ kg rad s^{-2}V^{-1},
$p_{e1} = 300.0$ kg rad s^{-2}V^{-1}, $p_{v1} = 30.0$ kg rad s^{-2}V^{-1},
$p_{e2} = 400.0$ kg rad s^{-2}V^{-1}, $p_{v2} = 40.0$ kg rad s^{-2}V^{-1},
$p_{b1} = 400.0$ kg rad s^{-2}V^{-1}, $p_{b2} = 40.0$ kg rad s^{-2}V^{-1},
$\alpha = 50.0$ NmV^{-1}, $\delta = 90.0$ kg rad^2 s^{-2},
$x_h = 0.11\pi$rad, $x_k = 0.3\pi$rad, $g = 9.8$m/s^2.

2. Neural system (Central pattern generator and Posture controller)

$\tau_i(i = 1 \cdots 12), \tau_{p1}$, and τ_{p2} are given and,
$\tau_4 = \tau_{10} = \tau_{p1} = \tau_{p2} = 1/50, \ \ \tau_i = 1/30$ (otherwise).
$\tau'_i(i = 1 \cdots 12), \tau'_{p1}$, and τ'_{p2} are given and
$\tau'_4 = \tau'_{10} = \tau'_{p1} = \tau'_{p2} = 20/3, \ \ \tau'_i = 10/3$ (otherwise).
$\gamma = 0.3, \Gamma = 0.1, a = 0.7, b = 0.8,$

$$w_{1\ 5},\quad w_{2\ 6},\quad w_{2\ 4},\ w_{4\ 3}\ =-1.0,$$
$$w_{5\ 6},\quad w_{6\ 5},\quad w_{1\ 7},\ w_{7\ 1}\ =-1.0,$$
$$w_{7\ 12},\ w_{8\ 12},\ w_{8\ 10}, w_{10\ 9}\ =-1.0,$$
$$w_{11\ 12}, w_{12\ 11}, w_{2\ 8},\ w_{8\ 2}=-1.0,$$
$$w_{4\ p1}, w_{10\ p2}\ =-1.0,$$
$$w_{1\ 2}, w_{2\ 1}, w_{7\ 8}, w_{8\ 7}=-2.0,$$
$$w_{1\ 3}, w_{2\ 3}, w_{7\ 9}, w_{8\ 9}=1.0,$$

otherwise $w_{ij}=0.0$.

3. Initial condition

$$x_1=0.0, x_2=l_1+l_2,$$
$$x_3, x_4, x_5, x_6=0.0,$$
$$\dot{u}_i=0.0,\quad \dot{v}_i=0.0,$$
$$\dot{u}_{p1}=0.0, \dot{v}_{p1}=0.0,$$
$$\dot{u}_{p2}=0.0, \dot{v}_{p2}=0.0.$$

References

[1] Grillner, S., Neurobiological bases of rhythmic motor acts in vertebrates, *Science*, **228** (1985), 143–149.

[2] Taga, G., Yamaguchi, Y. and Shimizu, H., Self-organized control of bipedal locomotion by neural oscillators in unpredictable environment, *Biol. Cybern.* **65** (1991), 147–159.

[3] Calancie, B., Needham-Shropshire, B., Jacobs, P., Willer, K., Zych, G. and Green, B., Involuntary stepping after chronic spinal cord injury: evidence for a central pattern generator for locomotion in man, *Brain*, **117** (1994), 1143–1159.

[4] Dimitrijevic, M., Gerasimenko, Y. and Pinter, M., Evidence for a spinal central pattern generator in humans, *Ann. NY. Acad. Sci.*, **860** (1998), 360–376.

[5] Teramoto, T., Ueda, K. and Nishiura, Y., Phase-dependent output of scattering process for traveling breathers, *Phys. Rev. E*, **69** (2004), 056224.

[6] Nishiura, Y., Teramoto, T. and Ueda, K., Scattering and separators in dissipative systems, *Phys. Rev. E*, **67** (2003), 056210.

[7] Ohgane, A., Ohgane, K., Mahara, H. and Ohtsuki, T., Strategy for Flexible Walking, *Forma*, **19** (2004), 427–425.

[8] Ohgane, K., Ohgane, K., Ei, S., Mahara, H., and Ohtsuki, T., 'Initial satate' coordinations reproduce the instant flexibility for human walking, *Biol. Cybern.*, **96** (2005), 426–435.

[9] Donelan, J. M., Kram, R. and Kuo, A. D., Mechanical work for step-to-step transitions in a major determinant of the metabolic cost of human walking, *J. Exp. Biol.*, **205**(23) (2002), 3717–3727.

[10] Saibene, F. and Minetti, A. E., Biomechanical and physiological aspects of legged locomotion in human. *Europ. J. Appl. Physiol.*, **88**(4-5), (2003), 297–316.

[11] Kokshenev, V. B., Dynamics of human walking at steady speeds, *Phys. Rev. lett.*, **93**(20), (2004), 208101,

[12] Ulrich, B. D., Haehl, V., Buzzi, U. H., Kubo. M. and Holt, K. G, Modeling dynamic resource utilization in populations with unique constraints: Preadolescents with and without Down syndrome, *Human Mov. Sci.*, **23**(2), (2004), 133-156.

[13] Sherrington, C. S., *The integrative action of the nervous system*, Yale University Press, New Haven, 1906.

[14] Sherrington, C. S., Flexion reflex on the limb, crossed extension-reflex and reflex stepping and standing, *J. Physiol.*, **40**, (1910), 28–121.

[15] Udo, M., Matsukawa, K., Kamei, H. and Oda, Y., Cerebellar control of locomotion : Effects of cooling cerebellar intermediate cortex in high decerebrate and awake walking cats, *J. Neurophysiol.*, **44**, (1980), 119–134.

[16] Udo, M., Matsukawa, K, and Kamei, H., Effects of partial cooling of cerebellar cortex at lobules V and IV of the intermediate part in the decerebrate walking cats under monitoring vertical floor reaction forces, *Brain Res.*, **160**, (1979), 559–564.

[17] Armstrong, D. M. and Edgley, S. A., Discharges of Purkinje cells in the paravermal part of the cerebellar anterior lobe during locomotion in the cat, *J. Physiol.*, **352**, (1984), 403–424.

[18] Udo, M., Matsukawa, K., Kamei, H., Minoda, K. and Oda, Y., Simple and complex spike activities of Purkinje cells during locomotion in the cerebellar vermal zones of decerebrate cats, *Exp. Brain. Res.*, **41**, (1981), 292–300.

[19] Udo, M., Oda, Y., Tanaka, K. and Horikawa, J., Cerebellar control of locomotion : discharges from Deiters neurons, EMG and limb movements during local cooling of the cerebellar cortex, *In. Progress in Brain Res.* 44: 445-459 **44**, (1976), 445–459.

[20] Chambers, W. W., and Sprague, J. M., Functional localization in the cerebellum. II. Somatotopic organization in the cortex and nuclei, *Arch. Neurol. Psychiatr.*, **74**, (1955), 653–680.

[21] Fitzhugh, R., Impulses and physiological states in theoretical models of nerve membrane, *Biophys. J.*, **1**, (1961), 445–466.

[22] Arshavsky, Y. I., Gelfand, I. M. and Orlovsky, G. N., The cerebellum and control of rhythmical movements, *Trends. Neurosci.*, **6**, (1983), 417–422.

[23] Ito, M., *The cerebellum and neural control*, Ravan, New York, 1984.

[24] Elfman, H., Forces and energy changes in the leg during walking, *Am. J. Physiol.*, **125**, (1939), 339–356.