Biol. Cybern. 56, 345–353 (1987)

Biological Cybernetics

Mechanisms of Frequency and Pattern Control in the Neural Rhythm Generators

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Abstract. The locomotive motion in animals is produced in some central neural units, and basically no sensory signal from peripheral receptors is necessary to induce it. The rhythm generators do not only produce rhythms but also alter their frequencies and patterns. This paper presents some methematical models of the neural rhythm generators and discusses various aspects of the frequency and pattern control in them.

1 Introduction

Rhythm patterns in the locomotive motion of animals (such as locomotion of quadruped animals, flying of birds, and swimming of fish) are generated in some central neural units, and basically any sensory signal is unnecessary to produce them. Moreover, animals do not only generate the locomotive rhythms but also can alter their speed over a broad range. Some animals even change the rhythm pattern itself; the transition from walking to galloping in the quadruped locomotion is a typical example of such a pattern change. Although the mechanism of the rhythm generation itself is now well understood (Bässler 1986), that of the frequency and pattern alteration is not yet clarified.

Friesen and Stent (1977) showed that a network consisting of three electronic neurons with cyclic inhibition augmented the rhythm frequency along with an increase of tonic excitation to the neurons. The ratio of minimum to maximum duration of cycle period was over 1 : 5. Miller and Scott (1977) also showed that an electronic circuit model of the spinal locomotor generator produced at least four-fold shifts of frequency according to the stimulus intensity. On the other hand, Tsutsumi and Matsumoto (1984) described that a network consisting of five neuron pairs only produced a frequency shift of about twenty percent by computer simulation. These authors, however, gave no or few explanations of why the rhythm frequency varied (or did not vary) with the intensity of the tonic inputs.

Some animals do not only alter the rhythm frequency but also change the rhythm pattern itself. In quadruped mammals, at least four gaits can be seen: the walk, the trot, the pace, and the gallop. The order of the stance and swing phases in the four legs are completely different between these gaits. Also in insects, two different gaits are observed: the slow walk and the run (the tripod gait) (Pearson 1976). An interesting discovery relating to the pattern change is that the pattern can be switched artificially by altering the intensity of the electric stimulus to the rhythm generator. Shik et al. (1965) showed that an increase of electrical stimulation to the midbrain region of the decerebrate cat did not only induce an increase in locomotion rate but also a gait shift.

In this paper we discuss some possible mechanisms in the frequency and pattern control in the neural rhythm generators. We first present a mathematical model representing a general class of neural rhythm generators, called mutual inhibition networks. Next, we investigate some specific networks consisting of a few neurons, which include some interesting networks suggesting the locomotion in quadruped and six-legged animals. Finally a general description is given on the rhythm control in the mutual inhibition networks consisting of more neurons.

Throughout the paper, no proof is given to mathematical propositions. One can prove them in a similar way to Matsuoka (1985).

2 Mutual Inhibition Networks

Although various models have been proposed to demonstrate neural rhythms, the essential feature common in every model is mutual inhibition between neurons (or neuron units). In this paper, therefore, we

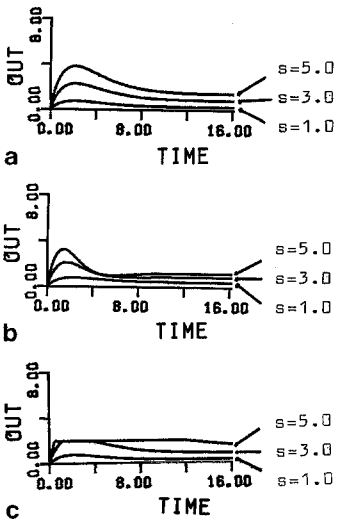

Fig. 1a–c. Step responses of the single neuron to step inputs with different magnitudes. **a** The basic model; $T_r=1$, $T_a=12$, $b=2.5$, $s(t)=0$ for $t<0$ and $=1$, 3 or 5 for $t\geqq 0$. **b** A modified model (7); $q=2$. **c** Another modified model; $x_{max}=2$. In **b** and **c**, the values of other parameters are the same as those in **a**

only consider a class of networks in which the constituent neurons (or neuron units) inhibit each other neuron's activity, and call them mutual inhibition networks.

As a model of individual neurons, we adopt the following continuous-time, continuous-variable neuron model, since the mathematical treatment is easy compared with other neuron models.

$$T_r dx/dt + x = s - bf, \tag{1}$$

$$y = g(x-\theta) \quad (g(x) \equiv \max\{0, x\}), \tag{2}$$

where x is a membrane potential of the neuron body, s an impulse "rate" of the tonic or slowly varying input, y a firing "rate" or output of the neuron, θ a threshold, T_r a time constant (we refer to it as a *rise* time constant since it specifies the rise time when given a step input). The threshold θ can be omitted (or $\theta=0$) without losing generality by replacing $x-\theta$ and $s-\theta$ with new variables x and s, respectively. f is the variable that represents the degree of fatigue or adaptation in the neuron, and b is the parameter that determines the steady-state firing rate for a constant input. If the second term of the right-hand side in (1) is omitted, this model becomes the same as the continuous neuron model adopted by many authors [for example, Morishita and Yajima (1972)].

The adaptation variable f obeys the following equation:

$$T_a df/dt + f = y, \tag{3}$$

where T_a is the time constant that specifies the time lag of the adaptation effect (we refer to it as an *adaptation* time constant). Responses of the single neuron to step inputs are shown in Fig. 1a. Due to the adaptation the neuron has a kind of high-pass-filter characteristic, which is a common property more or less observed in every real neuron.

A mutual inhibition network consisting of n neurons is represented by:

$$T_r dx_i/dt + x_i = -\sum_{j=1}^{n} a_{ij} y_j + s_i - bf_i, \tag{4}$$

$$y_i = g(x_i), \tag{5}$$

$$T_a df_i/dt + f_i = y_i, \tag{6}$$

where a_{ij} ($\geqq 0$ for $i \neq j$ and $=0$ for $i=j$) is a weight of inhibitory synaptic connection from the j-th neuron to the i-th neuron. (We consider neither excitatory connection nor self-inhibition.) The adaptation effect or the high-pass-filter characteristic of the single neuron plays a very important role in the rhythm generation; if each neuron has a strong adaptation effect, mutual inhibition networks of almost every structure generate stable rhythms if only they include a loop (Matsuoka 1985).

If s_i is replaced by Ks_i for all i, the solution of the differential equations only becomes K times as large as the original solution. Therefore, the uniform increase or decrease of the stimulus intensity induces no change in rhythm frequency or pattern. Thus, if a network has a property such that the rhythm frequency changes with the input intensity, the constituent neuron must have such a mechanism that the time constants or the other parameters virtually alter according to the stimulus intensity.

3 Rhythm Control in Mutual Inhibition Networks Consisting of a Few Neurons

In this section we discuss the rhythm control in several networks consisting of two to six neurons. They all have simple structures, but exhibit various aspects in the rhythm control.

3.1 Two-Neuron Network

There is only one mutual inhibition network that consists of two neurons (reciprocal inhibition network).

Network I. This network is the most simple mutual inhibition network, in which two neurons suppress each other neuron's activity (Fig. 2a). It can be considered a basic model of the stepping of one leg,

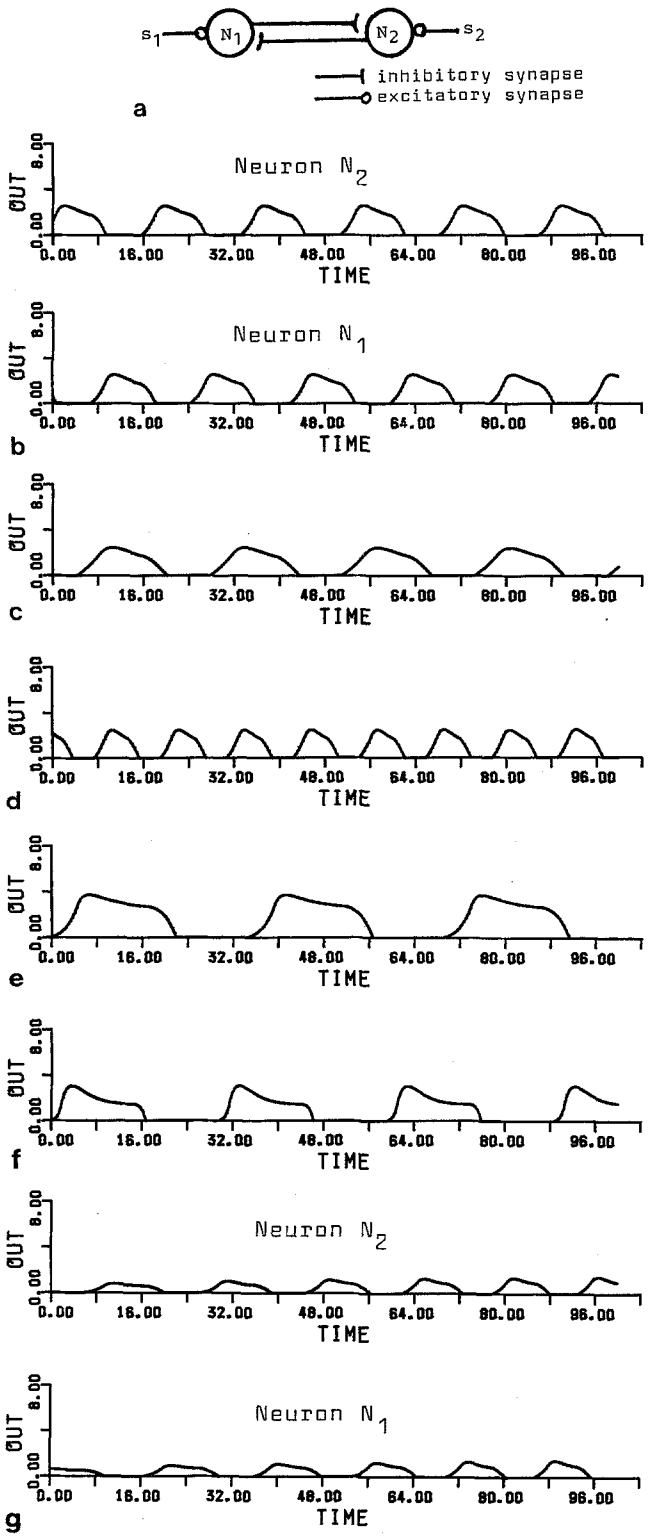

Fig. 2a–g. Network I. **a** Structure. **b** Output or firing rate of each neuron; $T_r=1$, $T_a=12$, $b=2.5$, $a_{12}=a_{21}=1.5$, $s_1=s_2=5$. **c** $T_r=2$. **d** $T_a=6$. **e** $b=1$. **f** $a_{12}=a_{21}=2.5$. **g** Outputs of the modified neurons; $q=2$; inputs are slowly increasing as $s_1(t)=s_2(t)=1$ for $t<0$ and $=0.02t+1$ for $t\geqq 0$. In **c** to **g**, the values of the other parameters are the same as in **b**. In **c** to **f**, the output of neuron N_1 is only shown

fluttering of bird wings, chewing and other simple rhythms, and was first investigated by Reiss (1962) by computer simulation. As for our continuous neuron model we can obtain the mathematical condition for the network to produce a stable rhythm; *(1)* $a_{12}/(1+b)<(s_1/s_2)$, $a_{21}/(1+b)<(s_2/s_1)$ and *(2)* $\sqrt{(a_{12}a_{21})}>1+T_r/T_a$. This condition claims that b or the adaptation must be large to evoke the rhythm; one can easily prove that if there is no adaptation or $b=0$, then any values of a_{12} and a_{21} do not satisfy the above condition. This network is a kind of relaxation oscillator; only one neuron can fire at a time due to the mutual inhibition, and the alternation of the firing neuron is caused by the adaptation in the firing neuron and the recovery of the activity in the resting neuron.

The rhythm frequency depends upon the values of the parameters included in the model. Figure 2b–f shows the rhythms generated for different parameters (with $a_{12}=a_{21}$ and $s_1=s_2$). It can be seen that the rhythm frequency is positively correlated to the parameter b, and is negatively correlated to the rise time constant T_r, the adaptation time constant T_a, and the synaptic weights of the mutual inhibition, a_{12} and a_{21}.

Meanwhile, the change of either or both stimuli yield little or no change in the rhythm frequency. However, if the neuron has a property such that its "virtual" time constants vary according to the input level, the rhythm frequency will depend on the intensity of the inputs. For example, we consider the following equation instead of (6):

$$T_a df_i/dt+f_i=y_i^q, \tag{7}$$

where q is an exponent greater than one. It implies that the adaptation effect increases in proportion of a power of the firing rate. Responses of the single neuron to step inputs of various magnitudes are shown in Fig. 1b ($q=2$). As the magnitudes of the input increases, the transient burst in the beginning is pronounced; i.e., the "virtual" adaptation time constant increases, although T_a is fixed. A rhythm generated by the network consisting of this modified neurons is shown in Fig. 2g, where the inputs ($s_1=s_2$) are slowly increased. One can see that the rhythm frequency increases with the increase of the input. Although we shall not deal with this modified neuron model henceforth, the same characteristic can apply to every network in which the alternation of the firing neuron is caused by the adaptation.

3.2 Three-Neuron Networks

There are five mutual inhibition networks consisting of three neurons. Four networks are only discussed,

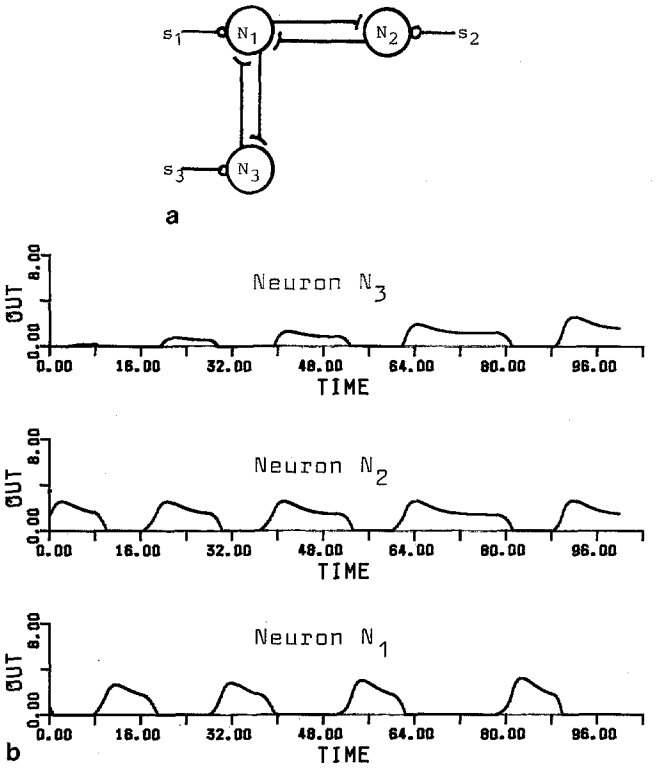

Fig. 3a and b. Network II. **a** Structure. **b** Outputs; $a_{ij}=1.5$, $s_1=s_2=5$, $s_3(t)=0.05t$; the other parameters are the same as in Fig. 2b

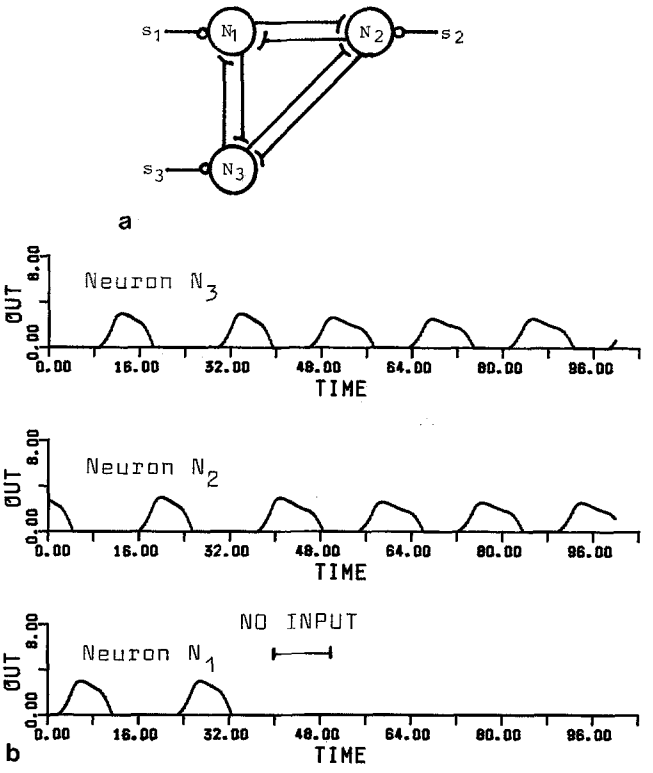

Fig. 4a and b. Network III. **a** Structure. **b** Output; $a_{ij}=1.5$ for every i and j; $s_1(t)=0$ for $40<t<50$, otherwise $s_1(t)=5$; $s_1=s_2=5$ at every t; the other parameters are the same as in Fig. 2b

since the other one has almost the same property as one of the network presented.

Network II. We connect another neuron N_3 to N_1 in Network I with a reciprocal connection (Fig. 3a). If the mutual inhibition is symmetric or $a_{ij}=a_{ji}$ for every i and j, a sufficient condition for a stable rhythm can be obtained as: *(1)* $a_{21}/(1+b)<s_2/s_1$, $a_{31}/(1+b)<s_3/s_1$, $a_{12}s_2+a_{13}s_3<(1+b)s_1$ and *(2)* a_{12} or $a_{13}>1+T_r/T_a$.

Now we suppose that the inputs, s_1 and s_2, are nearly equal and fixed, while s_3 is variable with time. For small s_3, the network is substantially the same as Network I, since the signal from N_3 to N_1 diminishes. If s_3 becomes comparable with s_1 or s_2, the behavior of the network turns to a reciprocal inhibition between N_1 vs. a pair of N_2 and N_3. This case is also equivalent to Network I with large synaptic weights of a_{21} and $2a_{12}$, leading to a decrease in the rhythm frequency. Figure 3b shows the decrease of the rhythm frequency when the magnitude of s_3 is slowly increased.

Network III. If we further assume a reciprocal inhibition between N_2 and N_3 in Network II and give all synaptic weights equal values ($a_{ij}=a$ for every i and j), we obtain a completely symmetric structure in terms of graph theory (Fig. 4a). The condition for the network to produce a stable oscillation is: *(1)* $a/(1+b)\leqq s_2/s_1$ and *(2)* $a>1+T_r/T_a$, where we assume $s_1\geqq s_2\geqq s_3$ without losing generality. This network is also a kind of relaxation oscillator, requiring the adaptation for the rhythm generation.

An interesting feature of this network is that it can generate five different rhythm patterns:

$N_1>N_2>N_3>N_1>N_2>N_3\ldots,$

$N_1>N_3>N_2>N_1>N_3>N_2\ldots,$

$N_1>N_2>N_1>N_2\ldots,$

$N_1>N_3>N_1>N_3>\ldots,$

$N_2>N_3>N_2>N_3>\ldots$

(> indicates the time order of firing neurons). In the last three cases one neuron's activity is always suppressed by the other neurons' firing.

Due to the multimode characteristic of this network, a rhythm pattern can be switched to another pattern by temporarily changing the stimulus pattern. An example is shown in Fig. 4b.

Network IV. Now we consider a network including a cyclic inhibition (Fig. 5a). If the synaptic weights are all the same ($a_{12}=a_{23}=a_{31}=a$), a condition for the

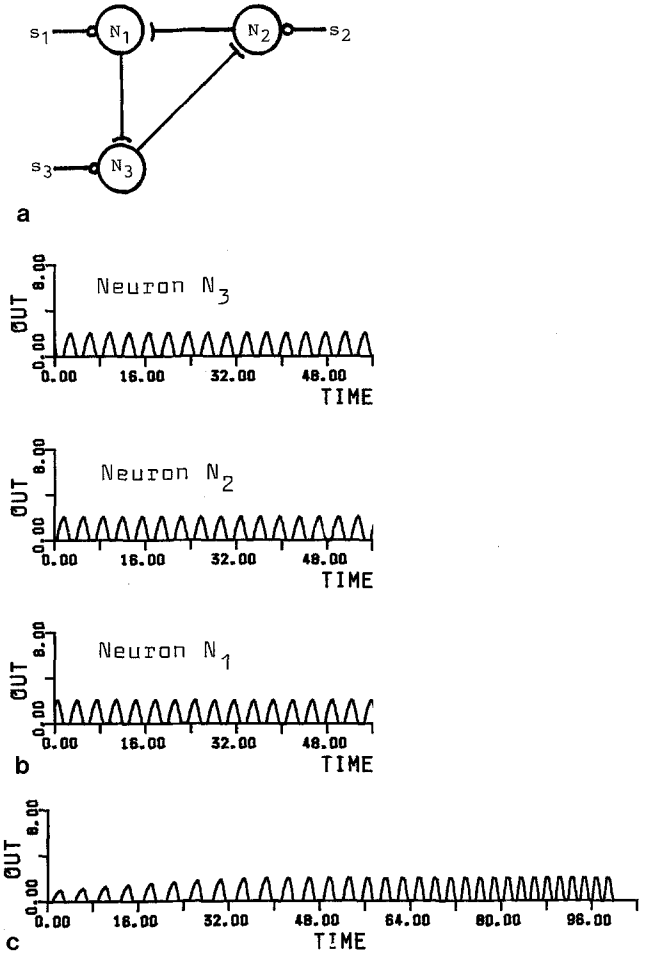

Fig. 5a–c. Network IV. **a** Structure. **b** Output; $a_{12}=a_{23}=a_{31}=2.5$, $s_1=s_2=s_3=5$. **c** The modified neuron model with a saturation; $a_{12}=a_{23}=a_{31}=4$; $x_{max}=2$; $s_1(t)=s_2(t)=s_3(t)=0.06t+2$; the output of one neuron is only shown. The values of the other parameters are the same as in Fig. 2b

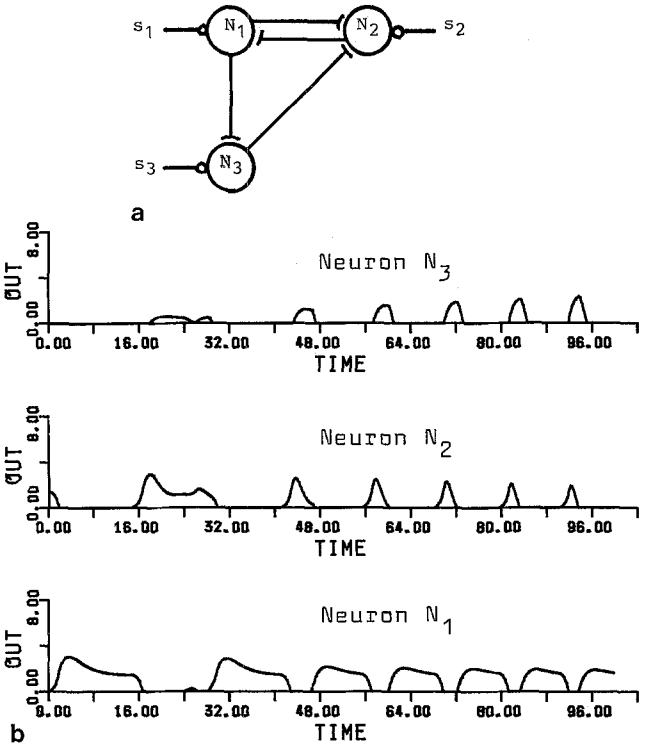

Fig. 6a and b. Network V. **a** Structure. **b** Output; $a_{ij}=2.5$; $s_1=s_2=5$; $s_3(t)=0$ for $t<0$ and $=0.04t$ for $t\geqq 0$; the other parameters are the same as in Fig. 2b

network to sustain a rhythm is: *(1)* $a/(1+b)\geqq s_1/s_2$, s_2/s_3, s_3/s_1, and *(2)* $a>1+T_r/T_a$.

The mechanism of the rhythm generation in this network is completely different from that in the last three ones. If N_1 is firing, N_3's activity will be suppressed. So N_2 will become firing, N_1 will be suppressed, and so on. Thus, the alternation of the firing neuron is caused by the negative feedback loop of this network, not by the adaptation or fatigue of the individual neurons. This network, therefore, does not necessitate adaptation but only strong cyclic inhibition for the rhythm generation (one can see that, large "a" satisfies the above condition for $b=0$).

Due to this behavior, the rhythm frequency is mainly determined by the rise time constant T_r, not by the adaptation time constant T_a. If T_r is much smaller than T_a, the rhythm frequency will become considerably higher than that in the last three networks (Fig. 5b).

The frequency is independent of the stimulus strength even for the modified model (7), since the rise time constant does not depend upon the stimulus strength. Here, we consider another modification of the original model; the membrane potential of the neuron body has a maximum or saturation value x_{max}, leading to a saturation of the firing rate. Step responses of the single neuron of this model are given in Fig. 1c. One can see that the "virtual" rise time constant varies with the magnitude of the step input. If this type of neurons are incorporated into Network IV, the rhythm frequency will alter according to the stimulus intensity (Fig. 5c).

Incidentally, Friesen and Stent (1977) showed that a three-neuron network of the same structure increased the rhythm frequency with an increase of tonic inputs. Their neuron model must have had a similar property to the present model.

Network V. This network (Fig. 6a) can be considered a combination of Network I and Network IV. For small s_3 it is nothing but Network I (generating a low-frequency rhythm), and for s_3 as large as s_1 and s_2 it behaves as Network IV (generating a high-frequency rhythm). Thus the rhythm frequency can be regulated by altering the magnitude of s_3. Figure 6b shows an increase of the rhythm frequency when input s_3 is

slowly augmented. The frequency seems to be changed discontinuously in the midpoint.

3.3 Four-Neuron Networks

Although there are more than a hundred four-neuron networks, we only discuss a few networks which are interesting from a biological point of view.

Network VI. This network (Fig. 7) is a relative of Network I. If the weights of mutual inhibition are given symmetric values, or $a_{ij}=a_{ji}$ for all i and j, a sufficient condition for the rhythm generation is obtained as: for some i and k $(i \neq k)$, *(1)* $s_i > \sum_j a_{ij}s_j/(1+b)$ and $s_k > \sum_j a_{kj}s_j/(1+b)$, and *(2)* $a_{ik} > 1 + T_r/T_a$ or b. If a_{ij} all have the same value, the behavior of this network becomes essentially equivalent to the reciprocal inhibition between two pairs, N_1-N_4 and N_2-N_3, in each of which two neurons fire in phase. This rhythm pattern might suggest the trot in the quadruped locomotion.

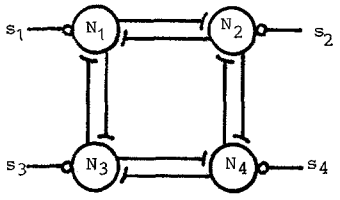

Fig. 7. Structure of Network VI

Network VII. This network (Fig. 8a) has a completely symmetric structure as Network III does. If a_{ij} all have the same value, the condition for the rhythm generation is just the same as that in Network III. Again it can produce many rhythm patterns (at least 18 patterns). However, if some synaptic weights are given large values relative to other ones, one rhythm pattern will become more stable than other patterns. One example is given in Fig. 8b, in which four synaptic weights are somewhat decreased so that the network will behave in a similar manner to the following Network VIII. One might see that it resembles the walk gait in the quadruped locomotion.

Network VIII. This network is a relative of Network IV and also produces a rhythm of high frequency (Fig. 9a). The condition for the rhythm generation becomes a similar one to that in Network IV.

Here, we suppose that the values of s_1 and s_2 are equal and fixed, while s_3 and s_4 are equal and changeable. When the magnitude of s_3 or s_4 is small, the network is equivalent to Network I, producing a low-frequency rhythm. For large s_3 and s_4, on the other hand, it produces a high-frequency rhythm. Thus, the frequency can be changed over a broad range by regulating the intensity of s_3 and s_4 (Fig. 9b).

Miller and Scott (1977) showed that a network model for the spinal locomotor generator changed the rhythm frequency according to the magnitude of a pair of inputs. The essential structure of their network and the mechanism of rhythm generation are just the same as those of the present model, although their model was composed of six neurons.

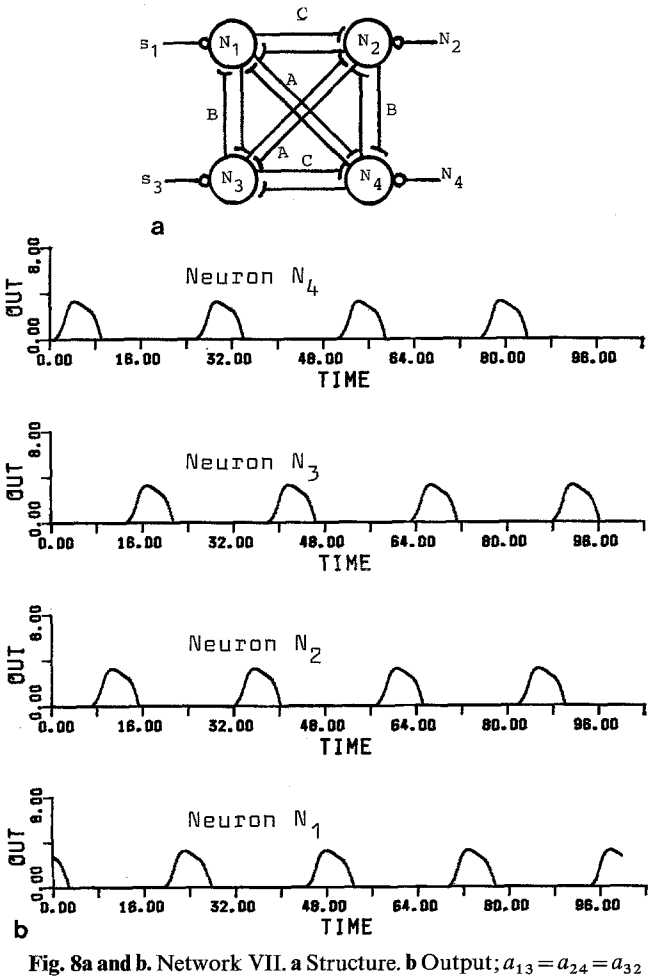

Fig. 8a and b. Network VII. **a** Structure. **b** Output; $a_{13}=a_{24}=a_{32}=a_{41}=1.5$ and other $a_{ij}=2$; the other parameters are the same as those in Fig. 2b

3.4 Six-Neuron Models

Finally we present a network consisting of six neurons, suggesting the six-legged locomotion in insects.

Network IX. This network is also a relative of Network I (Fig. 10); reciprocal inhibition between two triplets, $N_1-N_4-N_5$ and $N_2-N_3-N_6$. Two triplets fire with a phase difference of 180 deg to each other while in each triplet three neurons fire just in phase. It might suggest the running gait (the tripod gait) in insects.

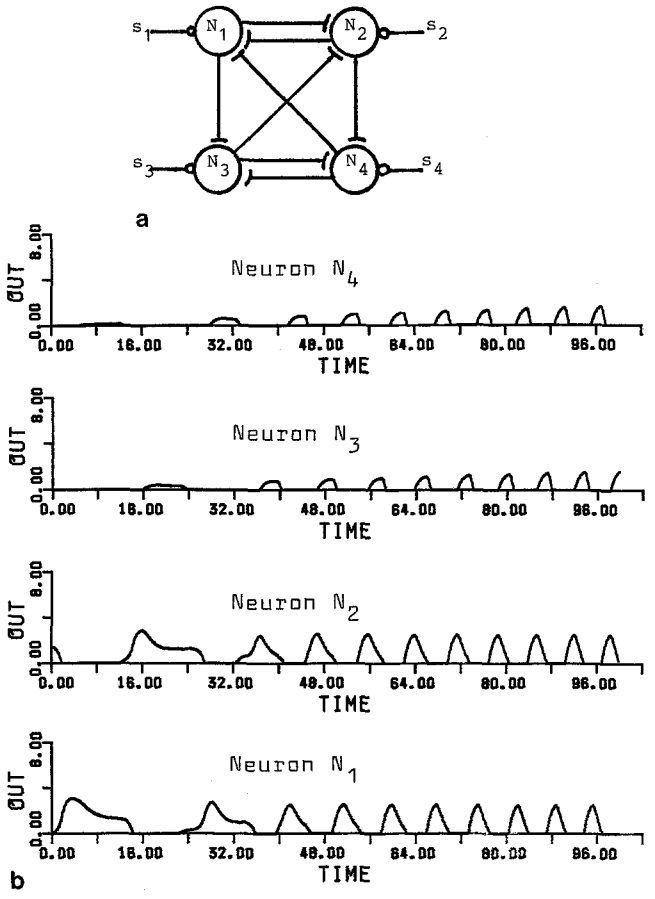

Fig. 9a and b. Network VIII. **a** Structure. **b** Output; $a_{ij}=2.5$ for all i and j; $s_1=s_2=5$; $s_3(t)=s_4(t)=0$ for $t<0$ and $=0.03t$ for $t\geqq 0$; the other parameters are the same as those in Fig. 2b

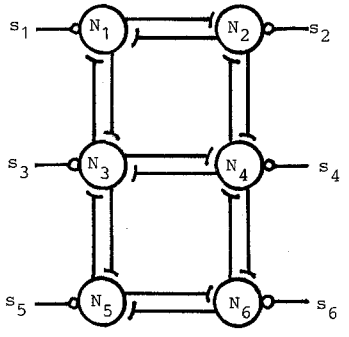

Fig. 10. Structure of Network IX

Network X. This network can be obtained by adding a cyclic inhibition into each triplet in Network IX (Fig. 11a). Between the two triplets, $N_1-N_4-N_5$ and $N_2-N_3-N_6$, a low frequency rhythm occurs, while in each triplet a high-speed cyclic rhythm appears. Thus, the entire network generates a rhythm as $N_1>N_4>N_5>N_2>N_3>N_6>N_1\ldots$ (Fig. 11b). This pattern resembles the slow walk in the locomotion of insects.

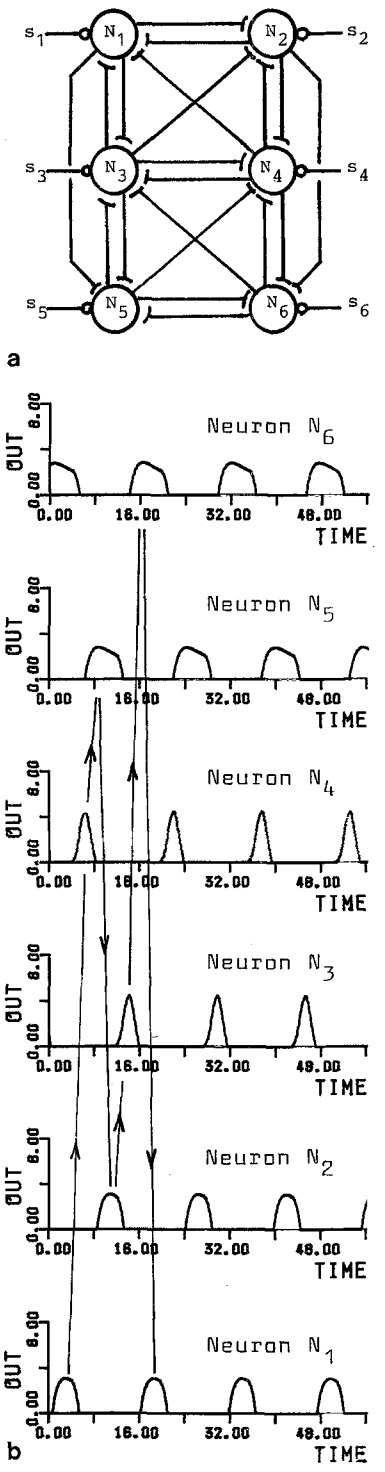

Fig. 11a and b. Network X. **a** Structure. **b** Output; $a_{ij}=2.5$; the other parameters are the same as in Fig. 2b

4 General Discussion on Rhythm Control

In the last section we investigated rhythm control in some networks consisting of a few neurons. Although they were all simple, they included various aspects in

rhythm generation and control. In this section we discuss the rhythm control in mutual inhibition networks in a general manner. Since the control strategy is very different between the types of the networks, we first investigate three typical types of mutual inhibition networks.

4.1 Three Typical Types of Networks

Bässler (1986) pointed out that neural networks generating rhythms can be divided into three types (excepting endogenously oscillating neurons); negative feedback type, positive feedback type, and their combination. Here we discuss this categorization somewhat mathematically. So as to avoid too complicated description we assume that the weights of the synaptic weights and inputs are all equal; $a_{ij}=a$ or 0, and $s_i=s$ for all i and j. Since it is obvious that a network including no loop generate no rhythm, we assume that a network includes at least one loop.

We first define a *structurally unstable* network as follows. Let D be a subset of neurons in a network. If D satisfies the following property, it is referred to as a D-subset:

(1) there is no synaptic connection within D;

(2) every neuron other than D receives at least one inhibitory signal from neurons in D.

If there is no D-subset in a network, we call the network a structurally unstable network. In the last section, Networks IV and VIII are structurally unstable networks. A sufficient condition for a structurally unstable network to produce a rhythm is given by:

Proposition 1. *A structurally unstable network generates stable oscillation (not necessarily periodic), if the synaptic weight, a, is large enough.*

Here the term "stable" means that the network continue oscillating for any large disturbance; strictly speaking, the system of the differential equations, (4), (5), and (6), has no stable equilibrium state on the above condition. It does not demand any adaptation but only strong mutual inhibition.

Next, we define a *bilaterally symmetric* network. Suppose that a network can be divided into two D-subsets,

$$D_1=\{N_1, N_2, \ldots, N_m\} \quad \text{and} \quad D_2=\{N'_1, N'_2, \ldots, N'_{m'}\},$$

and has a symmetric structure; i.e., if there is a connection from N_i to N'_j, there is also a connection from N'_i to N_j and vice versa. We call such a network a bilaterally symmetric network. Networks II, III, VI, and IX are of this type. A *necessary* condition for the network to generate an oscillation is:

Proposition 2. *For a bilaterally symmetric network to generate a stable oscillation, large adaptation or b is necessary. (Of course the synaptic weight, a, must be large for the rhythm generation, but must be small compared with b).*

The mechanism of rhythm generation is completely different between these two types. In a structurally unstable network, the alternation of firing neurons is caused by the negative feedback loop in the network. It, therefore, necessitate no adaptation of the single neurons. In a bilaterally symmetric network, on the other hand, the adaptation is necessary for the generation of stable rhythms, because the alternation of the firing neurons is caused by the adaptation of individual neurons. In most cases, the rhythm patterns will become the alternation between D_1 and D_2, in each of which all neurons fire in phase.

The last extreme case is a *completely symmetric* network. It is defined as a network in which there is a connection from any neuron to any neuron. The condition for a completely symmetric network to produce a sustained oscillation is:

Proposition 3. *If* $a/(1+b)\leqq 1$ *and* $a>1+T_r/T_a$, *then the network produces a stable oscillation.*

Due to the symmetry of the network this type of networks have many rhythm patterns, which depends on the initial state. Also in this case, the alternation of the firing neuron is attributed to the adaptation in the individual neurons.

4.2 Four Strategies in Rhythm Control

Uniform Change in Intensity of the Whole Inputs. It was shown that, in Network I and IV, the rhythm frequency varies with a uniform change of the tonic inputs, if the neuron model has a property such that the virtual time constants (rise or adaptation) change with the intensity of the inputs. Since the mechanism of rhythm generation is different between the structurally unstable networks and the (bilaterally and completely) symmetric ones, the dependence to the two time constants of the rhythm frequency is completely different; in the former type, the frequency is determined mainly by the rise time constant, while in the latter type by the adaptation time constant.

Temporal Change of the Stimulus Pattern in Multimode Rhythm Generators. As the connection between neurons becomes complicated, the network could have more than one rhythm patterns. The extreme case occurs in completely symmetric networks. In such a network, the rhythm pattern can be switched by temporarily changing the input patterns.

Alteration of Part of Inputs. In some networks, the rhythm frequency can be regulated by altering the

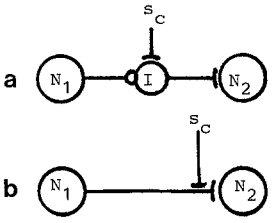

Fig. 12a and b. Two mechanisms for alteration of synaptic weights. **a** Interneuron. **b** Presynaptic inhibition

strength of some inputs. The regulation of the stimuli functions as a kind of gating of some paths in the network. In Network V, for example, putting the stimulus s_3 to null is equivalent to cutting the path $N_1 \rightarrow N_3 \rightarrow N_2$. For slowly varying inputs, the transition from a low-frequency rhythm to a high-frequency rhythm, or its inverse, can occur continuously or discontinuously, depending upon the network structure and the parameters.

Alteration of Synaptic Weights. The alteration of the synaptic weights also causes the change in rhythm frequency and pattern. This control of synaptic weight could be realized by some interneuron receiving other control signals or presynaptic inhibition (Fig. 12). The weight control also functions as a gate.

Using this mechanism, we can build a (speculative) model of the pattern change in the quadruped locomotion. First we consider Network VII in Fig. 8a. If the mutual inhibition is all large, the network will produce a slow rhythm resembling the walk gait as shown in Fig. 8b; $N_1 > N_4 > N_2 > N_3 > N_1 \ldots$. If the reciprocal inhibition A in Fig. 8a is not allowed to work, the rhythm pattern will turn to a reciprocal inhibition between two pairs, $N_1 - N_4$ and $N_2 - N_3$, giving the trot. If the reciprocal inhibition B is removed instead, it becomes the reciprocal inhibition between $N_1 - N_3$ and $N_2 - N_4$, giving the pace. Finally if the reciprocal inhibition C is eliminated, it will induce an alternation between $N_1 - N_2$ and $N_3 - N_4$, leading to the gallop. This strategy can also apply to the switching between the slow walk and the run in the six-legged locomotion; transition between Networks IX and X.

5 Concluding Remarks

We have seen four control mechanisms in the rhythm control. They are:

(1) the regulation of the stimulus intensity of the whole inputs;

(2) the temporary change of input in the networks potentially producing more than one rhythm;

(3) the alteration of part of stimuli;

(4) the change of synaptic weights.

Our mutual inhibition network model is imperfect in two respects:

(1) the model includes no excitatory synapse, which might have some important functions;

(2) it does not take into account the role of sensory signals from peripheral nerve receptors.

However, our model will be suggestive also when one constructs a more complicated model including these functions.

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Received: February 5, 1987

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