A Neural Model for Generation of Some Behaviors in the Fictive Scratch Reflex

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We have studied the scratch reflex in order to better understand the strategy that the spinal neural structures use to program limb movements. A network is proposed for the positioning and rhythmic portions of the scratch reflex in the deafferented preparation. This model is based on Berkinblit's hypotheses regarding organization of neurons constituting the spinal central pattern generator (CPG) for this behavior. We offer a mechanism by which the initial position of the hindlimb can influence the output of the CPG in the immobilized animal.

1 Introduction

The supraspinal control of a set of reflexes whose circuitries lie in the spinal cord may be the basic mechanism by which voluntary movements are generated (Berkinblit, Feldman and Fucksen 1986). The idea that the central control signals are expressible in terms of reflex parameters was the basis for work of Feldman on the "equilibrium point hypothesis" (for a review, see Feldman 1986). The underlying assumption is that afferent signals and central commands are interchangeable and that the nervous system accomplishes point to point movements by imitating inputs from the afferents to the stretch reflex circuitry (Berkinblit, Feldman and Fucksen 1986). This hypothesis leads to an algorithm for the control of redundant multijoint limbs without explicitly computing the inverse kinematics, the trajectory of movement, or the forces necessary for its execution (Berkinblit, Gelfand, and Feldman 1986).

The scratch reflex is an example of a movement that involves several joints and muscle groups and can be programmed by the spinal motor centers. In the vertebrate scratch reflex, an animal responds to irritation of its skin by reaching towards and scratching the irritation site rhythmically with its foot. Since the movement can be evoked in animals with the spinal cord transected at upper cervical segments, networks of neurons residing entirely in the spinal cord probably contain the essential mechanisms for control of this movement (Berkinblit et al. 1978). Understanding the mechanisms by which an animal maps the sensory field (in this case, the site of irritation on the skin) onto the neuronal circuitry
that is capable of generating a temporal pattern of muscular activation may lead to an understanding of voluntary limb movements which rely on the manipulation of the reflex circuitry.

The aim of this paper is to pursue a set of hypotheses on the organization of the neural controller for this reflex (Berkinblit et al. 1978; Deliagina et al. 1975; Deliagina and Orlovsky 1980; Deliagina et al. 1987; Shimanskii and Baev 1987), and to offer a neural model for the generation of the temporal pattern of scratching in the reduced preparation. Our approach is initially to consider the action of the neuronal controller of this reflex in the deafferented preparation: Although this oscillatory output is dependent on sensory feedback from the moving limb (Shimanskii and Baev 1987), when the corresponding limb is deafferented, essential characteristics of the movement remain intact (Berkinblit et al. 1978). We will then introduce limited afferent information to the model and compare the results with the immobilized, but afferent-intact, preparation.

2 The Experimental Data

In the fictitious scratch reflex (fictitious since the responding limb is immobilized), efferent neuromuscular contact is abolished and in response to skin irritation, the following activity in motoneurons is observed: In the initial positioning phase, which lasts about 2 seconds, motoneurons belonging to tibialis anterior (TA, ankle flexor), extensor digitorum longus (EDL, digit flexor), and quadriceps (Q, knee extensor) are gradually depolarized and begin firing, while no change in membrane potential is observed among motor neurons belonging to soleus and gastrocnemius (GS, ankle extensors), and plantaris (P, ankle extensor) (Baev 1981; Berkinblit et al. 1980; Deliagina et al. 1987). By the end of this phase the ankle joint is within reach of the scratch site (Berkinblit et al. 1978). Once it has reached the irritation site, the leg begins an oscillatory pattern of scratching motion (period of \(\sim 300\) msec) which is sustained until the irritation is removed. This second phase is termed the rhythmic phase and is further divided into an aiming (activation of TA, EDL and Q) and wiping phase (activation of GS and P). Almost all muscles active in the positioning phase (for example, TA, EDL and Q) are also active in the aiming phase, and are silent in the wiping phase. Muscles active in the wiping phase (for example, GS and P) are silent during the positioning and the aiming phases. Due to their similar time course of activation, we lump TA, EDL, and Q into group A, and GS and P into group B muscles. In summary then, the positioning phase involves only activity of motor neurons belonging to group A muscles, while the rhythmic phase involves alternation of group A and group B activity.

Figure 1a shows the average change in membrane potential (MP) for a set of neurons belonging to TA (group A) and GS (group B) motor pools during one scratch cycle. MP is drawn with respect to resting
potential of the same motoneuron before the irritation was applied. Note that the potential reached by the TA motoneurons by the end of the positioning phase (MPp) is essentially sustained during the rhythmic phase, except for periodic arrival of inhibition that drops the MP back to the resting level, but not below it. This suggests that during the rhythmic phase, the source of excitation of TA motoneurons is inhibited and not the motoneuron itself. GS motoneurons however do not undergo any change in their MP during the positioning phase and appear to receive only excitatory input during the rhythmic phase.

Berkinblit hypothesized (Berkinblit et al. 1978) that tonic excitatory inflow from the propriospinal neurons relaying information from the sensors at the irritation site activates the CPG. The neurons causing the rhythmic activity are distinguished from the motoneurons and their associated local reflex pathways (that is, Ia inhibitory interneurons and Renshaw cells). By contrast, Miller and Scott’s (Miller and Scott 1977) model of spinal pattern generators uses reciprocal inhibition among motoneurons to generate oscillatory behavior. However, there is some evidence (Pratt and Jordan 1987) that the reflex pathways of Ia inhibitory interneurons and Renshaw cells are not critical for generation of rhythmic activity in the motoneurons, and therefore are not part of the CPG for scratching. Berkinblit’s theory is further supported by the apparent localization of the neurons belonging to this CPG in the third to fifth segments of the lumbar (L3–L5) spinal cord (most hindlimb motoneurons are situated below L5). Many neurons in L3, L4 and L5 segments of immobilized cat fire in bursts that match the rhythmic behavior of the scratch cycle (Berkinblit et al. 1978). Figure 1b shows the averaged activity of these neurons. Berkinblit et al. (1978) named these neurons groups I, II, and III. To avoid confusion with muscle afferents, however, we will refer to these neurons as groups BI, BII and BIII. Activity of neurons in group BI appears to be phase locked with the group A motoneurons active during the aiming phase, while group BIII’s activity is similar to that of group B motoneurons which fired in the wiping phase.

During fictive scratching, activity in the ventral spino-cerebellar tract (VSCT) from the L4 and L5 segments is strikingly similar to that in neurons of groups BI and BII, while activity of spino-reticulo-cerebellar pathway (SRCP) neurons resembles that of group BIII (compare Fig. 202 of Ito 1984). Selective destruction and isolation of various segments of the spinal cord suggests that the main input to VSCT and SRCP neurons is the neuronal network generating the rhythmic oscillations in the L3 to L5 sections (Arshavsky et al. 1984).

The importance of cell groups BI, BII and BIII for generation of rhythmic hindlimb activity is not known. However, neurons in an isolated L5 segment can generate a seemingly unchanged rhythmic behavior when a scratch reflex is evoked (Delagyna et al. 1983). No rhythmic activity is generally observed more caudally (L6 and lower) when neuronal somas in L4 and L5 have been destroyed. Anatomical evidence that a fair
number of neurons located in lamina VII of L4 and L5 segments have axons that terminate on motoneurons of the seventh lumbar (L7) and first sacral (S1) segments (Jankowska and Skoog 1986) further supports the hypothesis of an oscillatory neuronal network residing in the L4 and L5 segments.
3 The Model

Based on this evidence, we propose a neuronal model for pattern generation in the fictive scratch reflex, including a mechanism by which afferent information may be processed in the immobilized preparation. The model (Fig. 2) comprises three groups of neurons (Groups BI, BII and BIII) that constitute the CPG, a set of Ia inhibitory neurons, two motor pools (A and B), and an afferent feedback loop. Each group of neurons in the CPG is modeled by one inhibitory (filled units) and one excitatory (open units) neuron. The three groups of neurons are assumed to reside mainly in lamina VII of L4 and L5 segments.

Activity in excitatory neurons of groups BI and BIII activates muscles in the aiming (group A) and wiping (group B) phases, respectively. Interaction between groups BI, BII and BIII neurons leads to the positioning and rhythmic behaviors observed during the fictive scratch. The role of Ia inhibitory interneurons can be described as follows: When dorsal roots are severed and no length information is being used to cause inhibition of the antagonist motor pool, a rhythmic pattern of activity persists in the Ia interneurons during the scratch cycle. The Ia interneurons leading to motoneurons of group A muscles are depolarized in phase with the motoneurons of group B muscles, while the Ia interneurons leading to the motoneurons of group B muscles are depolarized in phase with the later portion of the aiming phase (Deliagina and Orlovsky 1980). The pattern of depolarization in Ia interneurons leading to motoneurons belonging to group A muscles and group B muscles is similar to the pattern of activity seen in groups BIII and BII respectively. In figure 2, we therefore connect groups BII and BIII to these Ia interneurons.

Initially, we consider the operation of the network in the deafferented animal, that is, the afferent neurons are silent. As the irritation is detected, cutaneous input is translated into a tonic activation of group BI neurons which yield sustained contraction of the group A muscles (positioning phase). This afferent tonic excitation eventually activates group BII neurons which are postulated to have a longer time constant so that it takes them longer to reach firing threshold. Activity of the inhibitory neuron in group BII inhibits group BI neurons and serves to end the positioning phase. Activity in group BII's excitatory neuron excites group BIII neurons, but until group BII is successful in ending activity in group BI neurons, group BIII cannot overcome group BI's inhibition. Once group BI is shut down, activation of the excitatory neuron in group BIII leads to activation of the motoneurons in group B motor pool (wiping phase), while the inhibitory neuron in the same group inhibits group BII and ends its own source of excitation. Inhibition of group BII leads to disinhibition of group BI neurons, which leads to start of another aiming-wiping cycle.

The activation history of neurons belonging to groups BI, BII, and BIII is plotted in figure 3. Mathematics used in simulation of this network have been described elsewhere (Shadmehr and Lindquist 1988). Input
Figure 2: Schematic of the neuronal system postulated to control the positioning and rhythmic portions of the fictive scratch reflex. Groups BI, BII, and BIII are neurons located in lamina VII of the L4 and L5 spinal segments, while the Ia inhibitory interneurons are near the motor pools in the more caudal segments. Group A motor pool is a lumped representation for TA, EDL and Q motor pools. Group B represents GS and P pools. Open neurons are excitatory, filled are inhibitory.
arrives at the time marked with an arrow, and is simulated by excitatory input to groups BI and BII. Parameter values for this particular run are given in the appendix of (Shadmehr and Lindquist 1988). The initial period seen in group BI lasts on the order of 1.2 seconds. In this period, group A muscles are activated such that they deflect the hindlimb forward, presumably so that the paw is within reach of the scratch site. Once this positioning period ends, activity in group BI is limited to an oscillatory pattern which lasts 300 msec, followed by an inactivation period that lasts 100 msec.

The network offers a mechanism by which tonic input can be translated into an oscillatory output in a way that matches the data of (Berkinblit et al. 1978; 1980). Further data exists that describes the effects of initial hindlimb position on the output of the CPG in an immobilized animal whose afferents are intact (Baev 1981). Passive pulling of the limb to a more caudal position before eliciting the scratch reflex lengthens the initial positioning phase while pulling to a more rostral position shortens

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**Figure 3:** Activation history (firing rates) of the neurons of groups BI, BII and BIII. Stimulus is tonic excitation of groups BI and BII neurons. The long initial activation in the group BI neuron corresponds to the positioning phase, the period activation in group BI is the aiming phase. The short periodic activation in group BIII corresponds to the wiping phase. Firing rate of each neuron is a sigmoid function bounded by 0 and 1 (Shadmehr and Lindquist 1988).
it, suggesting that afferent information on length of hindlimb muscles in an immobilized animal affects the CPG.

Feedback from the hindlimb muscles to the L4 and L5 segments of the spinal cord appears to be mainly via group II afferents (Edgely and Jankowska 1987). These afferents make monosynaptic connections to neurons residing in lamina VII of L4 and L5 (where the CPG is postulated to be located) and can be activated with muscle stretches of less than 100 μm, though little or no innervation of this area of spinal cord from group I afferents has been found. It has also been reported that the intensity of afferent flow to the L5 region of the spinal cord from a hindlimb muscle that is being passively stretched increases (Shimanskii and Baev 1987).

To account for the variations in the network's output when the hindlimb is passively moved before the start of the behavior, we postulate that the new hindlimb position is reported to the neurons that make up the CPG by an increase in the firing rate of group II afferents of ankle and thigh extensors. Since the effect of this initial limb position must be a decrease in the length of the positioning phase, it is postulated that these afferents must excite group BII neurons of figure 2. Using the same approach, it can be supposed that afferents of thigh and ankle flexors increasingly inhibit group BII neurons as the limb is further pulled to a more caudal position. The net effect of synaptic input from these afferents on the group BII neurons must be of a tonic nature since the limb never actually moves after the fictive scratch has been initiated, and group II afferents, as opposed to group Ia afferents, are mainly sensitive to absolute length of the muscle (Carew and Ghez 1985), and not its rate of shortening.

We assume that the tonic source of excitation from irritation that initiated the reflex has remained the same. Figure 4 shows that effect of tonic inhibition on group BII neurons is a lengthening of the initial positioning phase with an increase in the period of the rhythmic phase. The effect of excitation is the reverse. Changing the afferent tonic inflow has a profound effect on the initial positioning phase, while the period of the rhythmic phase quickly reaches asymptotic values, in good agreement with experimental data showing that the location of the hindlimb mainly affects the initial positioning phase (Shimanskii and Baev 1987). The magnitude of change in the period of the rhythmic phase is a readily testable result of this model.

This model has attempted to take into account the role of afferent information only when the hindlimb has been immobilized. It was suggested that the dependence of the motor output on the initial position of the limb can be explained by considering a mapping of afferents belonging to extensor or flexor muscles such that they provide tonic excitation or inhibition to group BII neurons of the CPG, respectively. The role of afferent information on modification of the CPG's output in the intact animal is not known. However, it is generally believed that afferent information is processed at the input into the spinal cord by a mechanism
Figure 4: Length of the positioning phase and the period of the rhythmic phase as functions of the magnitude of input on group BII neurons. Input is defined as the weighted algebraic sum of the inhibitory and excitatory firing rates. Inhibitory input from muscle afferents leads to smaller values on the abscissa, while larger values on the abscissa result from excitatory afferent input on group BII neurons.

of presynaptic inhibition (Baev et al. 1978). The function of this inhibition on the pre-synaptic junction is to modulate the sensitivity of that terminal to incoming action potentials from the afferent neuron, or possibly generate anti-dromic action potentials to collide and cancel action potentials generated by the afferent neuron (Bayev and Kostyuk 1981). Recently, Shimanskii and Baev (1988) have suggested that a model of afferent inflow from the muscles is located in the scratch generator of the intact animal, and serves to correct the trajectory of the limb.

In the one dimensional model presented here, the mapping of the irritation site to positioning of the limb is coded in the time length of the positioning phase. From figure 4, it is possible to arrive at some quantity $h(u)$, which is the sum of tonic afferent inputs required to produce a positioning phase of length $d$. The goal of our future research is to postulate a network that converts the site of irritation and initial position of the hindlimb first to a positional error $e$ (distance from paw to irritation).
and then $h(e)$, which is now supplied as an extra input in parallel to the afferents we have postulated in figure 4.

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