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An unsupervised neural network model for the development of reflex co-ordination

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Abstract. In this paper, we present a model for the development of connections between muscle afferents and motoneurones in the human spinal cord. The model consists of a limb with six muscles, one motoneurone pool, one pooled (Ia-like) afferent for each muscle and a central programme generator. The weights of the connections between the afferents and the motoneurone pools are adapted during centrally induced movements of the limb. The connections between the afferents and the motoneurone pools adapt in a hebbian way, using only local information present at the synapses. This neural network is tested in two examples of a limb with two degrees of freedom and six muscles. Despite the simplifications, the model predicts the pattern of autogenic and heterogenic monosynaptic reflexes quite realistically.

1 Introduction

The nervous system receives information about the outside world through signals from sense organs. It transforms these signals into appropriate motor responses. Both the meaning of afferent signals and the effect of efferent signals changes during life, for instance, due to growth. Thus, the connections in the nervous system have to adapt.

There are several different approaches for studying representations of transformations in the nervous system. A system theoretical approach (e.g. Zisper 1992; Kalveram 1992) lays emphasis on the description of the resulting behaviour. Another approach is to look for an optimum solution for the transformation, given the boundary conditions of the physiological substrates (e.g. Loeb et al. 1989). A third approach is to look for mechanisms that could adapt the behaviour of the nervous system without any supervisor (e.g. Denier van der Gon et al. 1990). These three approaches give different kinds of results and have different limitations. Our approach in this paper is an example of the third category: In which (physiological plausible) way can the nervous system develop appropriate monosynaptic connections between muscle afferents and motoneurones?

An important factor for the enhancement of synaptic efficacy is probably the simultaneous activity of the neurones on both sides of the synapses, as proposed by Hebb (1949). An example will be presented which shows how hebbian mechanisms can form the basis for the development of reflex co-ordination: the formation of connections in the spinal cord leading to an adequate transformation from perturbation of arm position to reflex activation of the arm muscles. For our purpose (the study of the development of connections), this choice has several advantages. There is some knowledge about the (hebbian) mechanism subserving connection formation in the spinal cord (Nelson et al. 1989, 1990; but cf. Webb and Cope 1992), and the activities of motoneurones and sensory afferents during normal behaviour are roughly known. Furthermore, the normal pattern of connections is known: Ia-afferents of a muscle have strong connections with motoneurones of their parent muscle and somewhat weaker connections with synergistic muscles (in cat: Eccles et al. 1957; Fritz et al. 1989; in the human arm: Cavallari and Katz 1989). Moreover, in newborns, connections between Ia-afferents and all motoneurones (including those of antagonistic muscles) are strong. During growth, the connections become weaker, and the radiation to antagonistic muscles disappears (O'Sullivan et al. 1991).

Muscle co-ordination develops in a wide variety of muscles that may have different time constants, motor-unit types, fibre lengths, spindle densities, etc. Inter-individual differences in the strength and attachment positions of muscles also do not seem to hamper the development of muscle co-ordination. Since the learning mechanism we want to model results in co-ordinated activity notwithstanding such a wide range of anatomical and physiological parameters, it will also be effective for a somewhat simplified system. We will therefore use a rather simple model of the anatomy, physiology and mechanics of the human arm.

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2 Elements of the model

2.1 Anatomy

The model contains a description of the mechanics of an arm with two degrees of freedom. In this study we model two configurations (Fig. 1). The first one is the human arm as a planar manipulator (elbow angle about 90°). The more than fifteen muscles in this system are replaced by three antagonistic muscle pairs: one pair around the shoulder (pectoralis-infraspinatus), one pair around the elbow (brachialis-triceps short head) and a biarticular pair (biceps-triceps long head). The second system we study is the elbow with its two degrees of freedom (flexion-extension and supination-pronation of the forearm). For the study of this system, the muscles acting around the elbow are represented as two antagonistic pairs, one for each of the two degrees of freedom (supinator-pronator quadratus and brachialis-triceps), and two muscles which combine a flexion torque with a torque component in the supination (biceps) or pronation (pronator teres) direction.

The anatomical parameters used are listed in Table 1. The parameters describing muscle anatomy are derived from anatomical data found in Yamaguchi et al. (1990). The parameters for the lumped muscles in our model are averages of the parameters of the constituent muscles. The main difference between our model and the anatomical data is that for reasons of simplicity our model has some symmetry: the flexors and extensors are equally strong and have the same moment arms.

It has been shown (Tax and Denier van der Gon 1991) that, assuming a homogeneous activation of the motoneurone pool, muscle force increases linearly with the overall input to the motoneurone pool. The motoneurone pool (the motoneurones of a muscle) can therefore be represented by one linear unit. Muscle spindles are innervated by both β-motoneurones and γ-motoneurones. Although there are numerous experiments which show that γ-motoneurones can be activated independently of the α-motoneurones, α-γ coactivation is frequently observed (Prochazka 1989). For simplicity, the pool of α-, β- and γ-motoneurones of each muscle is modelled by one (β-like) motoneurone pool innervating both intrafusal and extrafusal muscle fibres. The ensemble of spindle afferents of a muscle is modelled as one (Ia-like) afferent that signals the sum of spindle activities (Fig. 2).

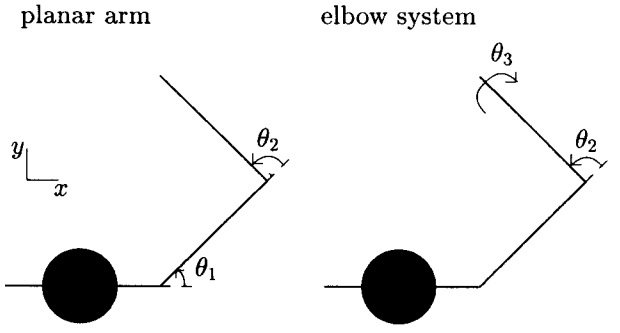

Fig. 1. The two systems used for the simulations. In the planar arm (*left*) both the shoulder (θ_1) and the elbow (θ_2) can flex and extend. In the elbow system (*right*), the shoulder is fixated, and the elbow has an extra degree of freedom supination–pronation of the forearm (θ_3)

2.2 Mechanics

Torques about the joints, moment arms and joint velocities are expressed as two-dimensional vectors in joint coordinates (Hogan 1985). The activity of the motoneurone pools (M_i) results in torques ($\vec{T}$), which depend on the maximal forces (F_i) that the $K(=6)$ muscles

Table 1. The values of the anatomical parameters used in the model. The strength of the muscles (F) is proportional to their physiological cross-sectional area. Moment arms ($\tau_{1,2,3}$) are given in mm. For the planar arm, the degrees of freedom are flexion-extension of the shoulder (θ_1, τ_1), and of the elbow (θ_2, τ_2); for the elbow system, flexion-extension of the elbow (θ_2, τ_2) and supination-pronation (θ_3, τ_3). For the definitions of these angles, see Fig. 1. All muscles which have the same combination of action about the joints are pooled in one muscle. The "brachialis" of the planar arm, for instance, represents both the "brachialis" and "pronator teres" of the elbow system. For lumped muscles that are present in both systems, the same anatomical parameters are used. The values of the jacobian and inertia tensors are derived for $\theta_1 = \frac{1}{4}\pi$ and $\theta_2 = \frac{1}{2}\pi$. s.h., short head; l.h., long head

Planar arm				Elbow			
Muscle	F	τ_1	τ_2	Muscle	F	τ_2	τ_3
1. brachialis	50	0	30	1. brachialis	25	30	0
2. biceps	25	45	30	2. biceps	25	30	7
3. pectoralis	150	45	0	3. supinator	15	0	7
4. triceps s.h.	50	0	−30	4. triceps	75	−30	0
5. triceps l.h.	25	−45	−30	5. pronator quad.	15	0	−7
6. infraspinatus	150	−45	0	6. pronator teres	25	30	−7
Inertia (kg m²)	$\begin{pmatrix} .17 & .07 \\ .07 & .07 \end{pmatrix}$			Inertia (kg m²)	$\begin{pmatrix} .07 & 0 \\ 0 & .0014 \end{pmatrix}$		
Jacobian (m)	$\begin{pmatrix} .42 & -.21 \\ 0 & -.21 \end{pmatrix}$			Jacobian (m)	$\begin{pmatrix} .3 & 0 \\ 0 & .06 \end{pmatrix}$		

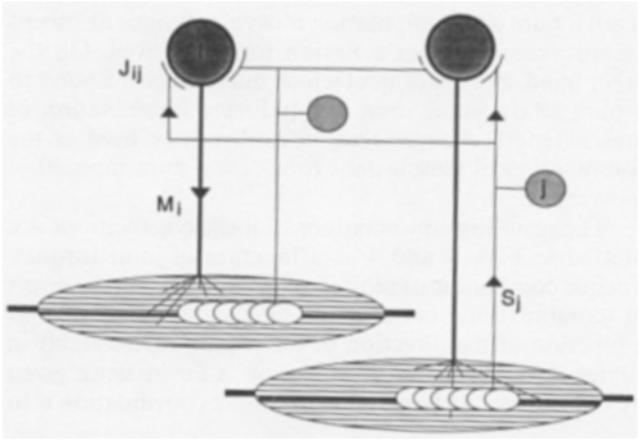

Fig. 2. Scheme of the model for learning the connections J_{ij} between the motoneurone pool i and afferent j. The efferent signal M_i of motoneurone pool i (activating both intrafusal and extrafusal muscle fibres) and the afferent signal S_j are indicated. Only two of the six muscles, spindles, motoneurone pools and afferents used in the model are shown

can exert, and on their moment arms ($\vec{r}_i$):

$$\vec{T} = \sum_{i=1}^{N_a} M_i F_i \vec{r}_i \tag{1}$$

These torques cause movements of the arm. For the elbow system, the two degrees of freedom are independent, so the equation of motion is

$$\vec{T} = \mathbf{I}\ddot{\vec{\theta}} \tag{2}$$

with $\mathbf{I}$ the inertia tensor in joint coordinates. The equation of motion for the planar arm (see for instance Smeets et al. 1990) is somewhat more complicated. In this paper we use only movements over a relatively short distance (implying also low velocities), so that we can approximate these equations by taking only inertial forces into account and by making $\mathbf{I}$ constant. So (2) holds also for this system. By writing (1) we assumed that all time dependence in the relation between muscle-activation and the resulting muscle-force can be neglected (see Discussion). The joint velocities $\dot{\theta}$ are calculated for both systems by integration of the acceleration:

$$\dot{\vec{\theta}}(t) = \int \ddot{\vec{\theta}} \mathrm{d}t = \int \mathbf{I}^{-1} \vec{T} \mathrm{d}t \tag{3}$$

From these velocities we can compute the resulting lengthening velocity of each muscle j

$$v_j = \dot{\vec{\theta}} \cdot \vec{r}_j \tag{4}$$

The relationship between the joint velocities and the velocity of the end-effector (hand) in cartesian coordinates ($\dot{\vec{x}}$) is given by the Jacobian $\mathbf{J}$:

$$\dot{\vec{x}} = \mathbf{J}\dot{\vec{\theta}} \tag{5}$$

The values for the Jacobian and inertia given in Table 1 were derived from anthropometric data (Winter 1979). For the elbow system, however, it is not evident from anthropometric data what the position of the end-effector is for the supination-pronation direction. We chose for this position a point 6 cm from the supination-pronation rotation axis. In this way, the position of the end-effector is scaled relative to moment-arms and inertias: a change in the length of the biceps corresponds to a supination movement and a flexion movement over the same distance, and maximum activation of the supinator and brachialis will lead to about the same (linear) acceleration of the end-effector in the supination and flexion directions.

2.3 Physiology of neurones

During movements, the output of Ia-afferents depends on the γ-activity, the length of the muscle and the stretching velocity. As the movements in this paper are over a relatively short distance, the position dependence will be neglected. The output of the afferent of muscle j is modelled as being linearly dependent on the stretching velocity v_j and the activation M_j of this muscle:

$$S_j = \begin{cases} (1 + M_j)(v_j/v_j^{\max} - v_0) & \text{if } v_j/v_j^{\max} > v_0 \\ 0 & \text{otherwise} \end{cases} \tag{6}$$

with $v_j^{\max}$ the maximum stretching velocity of muscle j in the simulations and v_0 the fraction of the maximum velocity at which the afferent starts firing. As the value for v_0 we chose -0.25, so that the spindles also fire at low shortening velocities. The effect of the activation M_j in our model corresponds to the effect of a combined γ_s and γ_d activation of physiological spindles: an increase of M_j increases both the dynamic index of the response and the response in the absence of movement. We choose the effect of the spindle activation to be moderate: the maximum effect of the activation is a doubling of the spindle output.

2.4 Central pattern generator

The activation pattern used to generate a movement is a two-burst pattern: an activation of the agonists to accelerate the limb, followed by an activation of the antagonists. The overall activation levels were chosen such that in all directions the movement of the end-effector had the same amplitude and average speed, and that the acceleration phase and deceleration phase had the same duration. Which combination of muscles acts as agonists and which as antagonists is a problem which is easily overlooked, but important in a study of muscle coordination.

To generate a torque about a joint, the nervous system has an ample choice of combinations of muscle activation. For instance, to generate an elbow flexion torque, it could activate m. brachialis, m. brachioradialis, m. biceps (combined with the activation of muscles which counteract other torques generated by m. biceps), or any combination of these three. Our pattern generator uses a solution to this redundancy problem originally proposed by Jongen (1989b). For the model of a limb with two degrees of freedom and six muscles, the reasoning is as follows.

A movement of the limb results in length changes of all six muscles. As the limb moves in a two-dimensional

space, the possible combinations of changes in length of the six muscles are restricted to two degrees of freedom. If the nervous system uses these possible combinations of length changes of the muscles (that are known from afferent signals) as combinations of activations of these muscles, it has a solution for the redundancy problem. For instance, during a displacement in the supination direction, all muscles which act as an elbow supinator will shorten; the amount of shortening will depend on the moment arm relative to the elbow. According to Jongen's model, the activation of these muscles proportional to their shortening (and inhibition of the muscles that lengthen) will be used for making a movement. The movement resulting from this combination of activations is not a pure elbow supination movement, since m. biceps for instance generates a flexion torque as well. On the other hand, a displacement which does not correspond to a pure elbow supination will induce a combination of muscle length changes that, in turn, will be used as the combination of muscle activations for a pure supination movement.

The resulting combinations of muscle activations are plotted in Figs. 3 and 4 as a function of joint torques. Torque combinations plotted as iso-activity curves result in (combinations of) straight lines; activity plotted as a function of the direction of the (equal) forces result in circles. A derivation of this relation is for instance given by Goossens et al. (1993). The resulting coordination is in

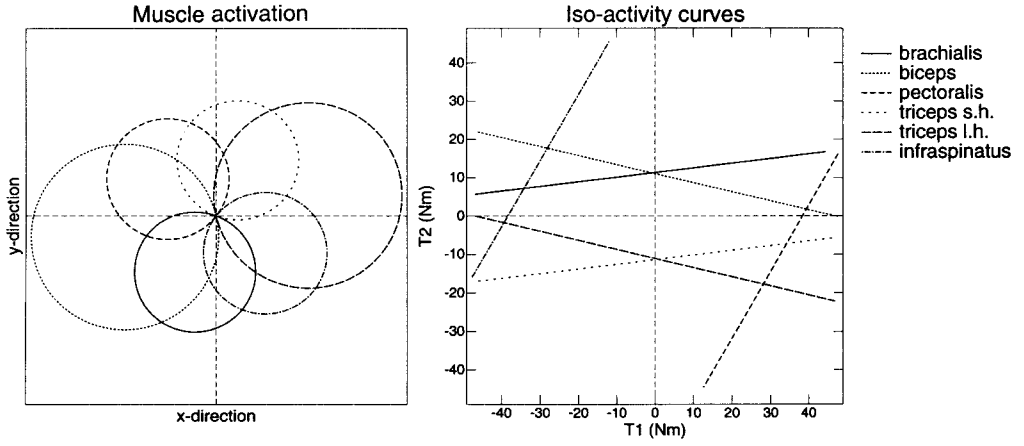

Fig. 3. The coordination of the muscles in our model of the planar arm. In the *right part* (iso-activity curves), we have connected for each muscle all combinations of torques which correspond with the same level of activation M_i of that muscle. The (positive) torques along the axes correspond to shoulder flexion (T_1) and elbow flexion (T_2). These curves correspond to experimentally measured recruitment curves or iso-electromyography (EMG) curves. In the *left part*, a polar plot is shown of the muscle activations M_i that are needed for a movement (constant acceleration) as a function of the movement direction. The x-direction is parallel to the frontal plane and the y-direction, perpendicular to this plane (see Fig. 1). These curves are comparable to EMG measurements as a function of (movement) direction

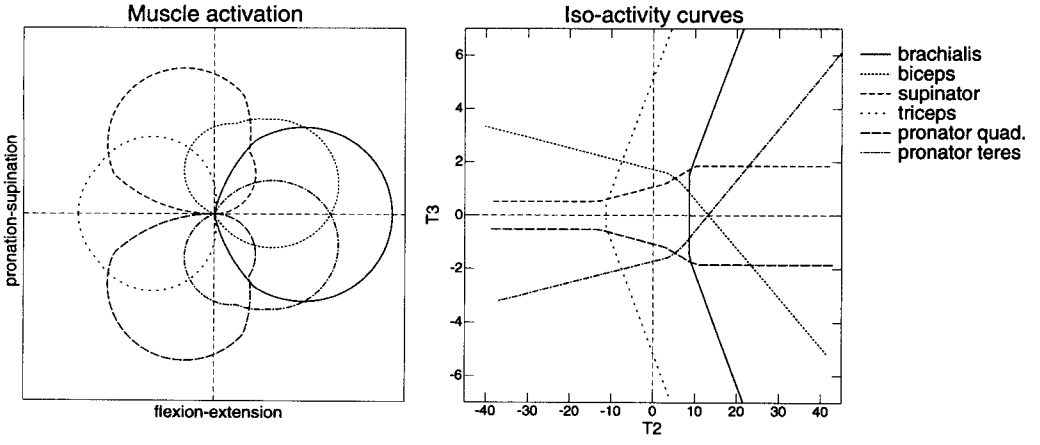

Fig. 4. The coordination of the muscles in our model of the elbow system. In the *right part* (iso-activity curves), we have connected for each muscle all combinations of torques which correspond with the same level of activation M_i of that muscle. The (positive) torques along the axes correspond to: elbow flexion (T_2) and forearm supination (T_3). These curves correspond to experimentally measured recruitment curves or iso-EMG curves. In the *left part*, a polar plot is shown of the muscle activations M_i that are needed for a movement (constant acceleration) as a function of the movement direction. These curves are comparable to EMG measurements as a function of (movement) direction

fair agreement with experiments (van Zuylen et al. 1988; Jongen 1989a; Miller et al. 1992; Goossens et al. 1993), both for the planar arm (Fig. 3) and for the elbow system (Fig. 4). Both our model and the experimental data only describe the final activity M_i of motoneurones. Motoneurone activity is not the same as the central input to the motoneurones: also afferent signals make an important contribution to this activity. A further discussion on this point can be found in papers discussing the equilibrium point hypothesis (Feldman 1986; Bizzi et al. 1992).

We want to represent the whole range of movements of the arm by a limited set of model movements. In this paper, we use movements of only one amplitude and one average speed, in directions spread uniformly across (two-dimensional) space.

2.5 Development of the connections

We assume that the enhancement of the connections between the afferents and the motoneurone pools depends only on their simultaneous activity during voluntary movements. In the model the two phases of movement, acceleration and deceleration, are treated separately. For each phase we used average values of spindle output, motoneurone pool activity and velocities. We calculated the changes in the connections by making movements in N different directions ϕ_n, uniformly spread across space. The strength of the connection J_{ij} between afferent j and motoneurone pool i was updated from the presynaptic and postsynaptic activities during these movements, averaged over all N directions ϕ_n, according to:

$$\Delta J_{ij} = \frac{1}{N} \sum_{n=1}^{N} ((M_i S_j)_{\phi_n}^{\text{acc.}} + (M_i S_j)_{\phi_n}^{\text{dec.}}) - \varepsilon_1 J_{ij} - \varepsilon_2 \sum_{j'=1}^{K} J_{ij'} \tag{7}$$

The three terms of this equation describe a hebbian learning process. Although the weakening and strengthening of connections are equally important for their strength, Hebb (1949) did not postulate under what conditions connections weaken. The first term is the postulate of Hebb: a connection becomes stronger (synapses grow or more synapses are created) when the presynaptic neurone and the postsynaptic neurone are simultaneously active. For each direction ϕ_n the activities of afferents and motoneurone pools are calculated separately for the acceleration and deceleration phases. The other two terms in (7) describe two mechanisms for the decay of the synapses. The first mechanism (ε_1) is a decay proportional to the strength of the connection. The second decay mechanism (ε_2) can be seen as the result of a competitive process between afferents on the surface of a motoneurone; this decay is thus proportional to the total strength of projections onto the motoneurone pool i. Evidence for such competitive processes has recently been reported (Nelson et al. 1990; Lo and Poo 1991). We use only excitatory connections: $J_{ij} > 0$. If J_{ij} as calculated by (7) becomes negative, J_{ij} is set to zero. Typical values that we used for the decay parameters were: $\varepsilon_1 = 0.2$, $\varepsilon_2 = 0.06$. Movements were made in $N = 360$ equally spaced directions.

In the simulations, the synapses developed during movements until they reached stable values: (7) is evaluated until $\Delta J_{ij} = 0$.

2.6 Testing of the model

To check the quality of the model, we have to compare the calculated values of J_{ij} with experimental data. There are no direct measurements of the strength of the connections between Ia-afferents and motoneurones in humans. Short latency stretch reflexes are the most direct manifestation of these connections.

We will consider the question of which muscles are activated to counteract a position perturbation. In general, activating only the muscles that are stretched will not result in a force or movement exactly opposite to the perturbation. This is due to the non-orthonormal attachment of the muscles (if, for instance, the supinating muscles m. biceps and m. supinator are stretched by a pronating perturbation, the reaction will be not only supination, but also flexion of the elbow) and/or to the mechanics of the system (in general, to counteract a movement about one joint in a two-jointed limb requires torques about both joints). With regard to the elbow system, it was shown that only the muscles that were stretched showed reflex activity at monosynaptic latency (Gielen et al. 1988), whereas with regard to the planar arm, a monoarticular elbow flexor was activated at short latency in reaction to a rotation about the shoulder (Smeets and Erkelens 1991).

To compare the model behaviour with these experimental results, we elicit short latency reflexes in the model by displacements of the model arm (equal velocity for all directions), in the absence of any background motoneurone pool activity. The amplitude A_i of the monosynaptic reflex in muscle i due to a displacement of the arm is calculated according to:

$$A_i = \sum_{j=1}^{K} J_{ij}(S_j - S_j^0) \tag{8}$$

where S_j is the output of afferent j during the displacement, calculated according to (6), with $M_j = 0$ for all muscles j. S_j^0 is the output of afferent j when the arm is not moving [$S_j^0 = -v_0$ according to (6)]. The reflex activations A_i lead to torques around the joints [(1) in which the activation M_i of the motoneurone pool is equal to A_i], resulting in a movement.

The stretch reflex is generally assumed to counteract displacements of the hand. If the connections are ideal, then for a direction of displacement ϕ_n^d, the torques caused by the reflex activation of the muscles will result in a movement in the direction $\phi_n = \phi_n^d - \pi$. To make statements about the quality of the reflex action, we have to define a measure of the quality. For this we used E, the root of the squared direction error, averaged over N equally spaced directions ϕ_n^d:

$$E^2 = \frac{1}{N} \sum_{n=1}^{N} (\phi_n^d - \pi - \phi_n)^2 \tag{9}$$

in which $\phi_n^d - \phi_n$ is restricted to the interval $(0, 2\pi)$. The maximal value of E is π, in which case the reflex movement is for all perturbations in exactly the same direction as the perturbation. The minimum value of E is zero: the reflex movement is then for all perturbations exactly opposite to the perturbation.

Another way to test the resulting mechanical behaviour is to measure the stiffness field of the arm (Hogan 1985). The stiffness tensor **R** resulting from the connections was calculated by a two-dimensional linear least squares fit of the resulting torques $\vec{T}$ to the displacements $\Delta\vec{\theta} = \dot{\vec{\theta}}\Delta t$

$$\vec{T} = \mathbf{R}\Delta\vec{\theta} \tag{10}$$

3 Results

Figure 5 shows an example of the connections J_{ij} for both systems. The equilibrium values of J_{ij} were independent of their values at the start of the simulation. Independent of the choice of parameters in (7), the autogenic connections for all muscles are stronger than connections with other muscles. The values of the decay parameters determine the strength of the connections with other muscles. The values of the decay parameters determine the strength of the connections with other muscles. Large values of ε_2 result in only autogenic connections. The decay parameters were chosen so that about half of the connections equal zero.

When the arm is displaced, the muscles are activated in response, according to (8). Polar plots of the predicted reflex-amplitude for some muscles are shown in Figs. 6 and 7. For the other muscles of our model, the reflex amplitudes follow from the symmetry in the model. In general, a muscle shows reflex activity in response to displacements that stretch the muscle. Due to the asymmetrical connection-matrix, however, the directions of

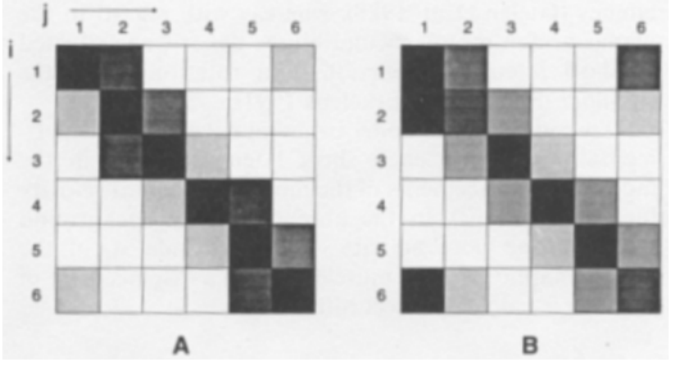

Fig. 5a, b. The connection matrices calculated with the values: $\varepsilon_1 = 0.2, \varepsilon_2 = 0.06$. A *square* in row i and column j indicates the strength J_{ij} between motoneurone i and afferent j. *Dark squares* denote strong connections, *light squares* weak connections and a *white square* $J_{ij} = 0$. Numerical values of the connections are given in Table 2. **a** The connection matrix for the muscles of the planar arm. The symmetry in the model is reflected by a translational symmetry in this plot. **b** The connection matrix for the muscles of the elbow system. The symmetry in the model is reflected by a point symmetry relative to the triceps in this plot

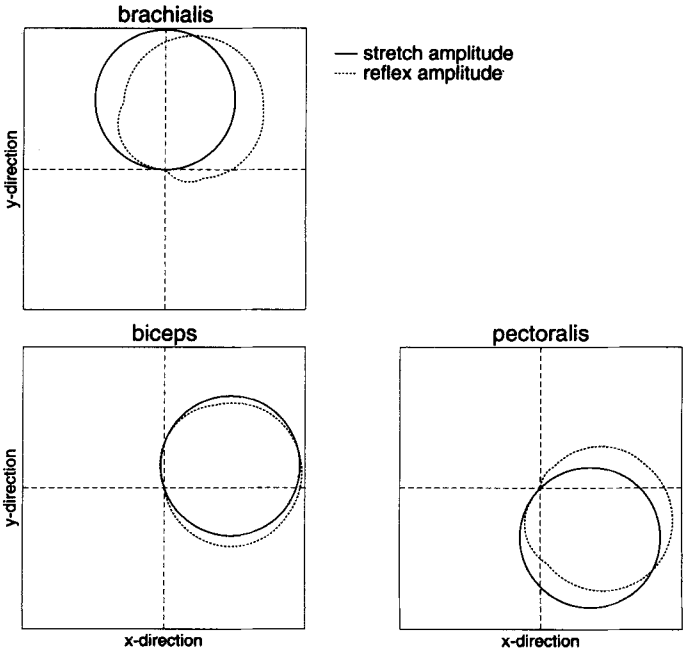

Fig. 6. Polar plot of the relative amplitudes of the stretch reflex for the muscles of the planar arm as a function of the direction of the displacement. For comparison, the relative amplitudes of muscle stretch during the same displacements are plotted in the same figures. The positive x-axis denotes a displacement in the flexion direction, and the positive y-axis a displacement in the supination direction. For the muscles which are not shown in this figure, the amplitudes of the stretch reflex follow from the symmetry in the system. For calculation of the reflex amplitude, the connections of Fig. 5a are used

Table 2. The numerical values of the connections J_{ij} between afferents and motoneurones as shown in Fig. 5

Planar arm							Elbow						
Afferent Motoneurone	1	2	3	4	5	6	Afferent Motoneurone	1	2	3	4	5	6
1. brachialis	0.9	0.7	0	0	0	0.2	1. brachialis	1.6	0.6	0	0	0	0.6
2. biceps	0.3	2.2	0.6	0	0	0	2. biceps	0.9	0.8	0.4	0	0	0.2
3. pectoralis	0	0.7	1.0	0.3	0	0	3. supinator	0	0.4	1.2	0.2	0	0
4. triceps s.h.	0	0	0.2	0.9	0.7	0	4. triceps	0	0	0.4	1.2	0.4	0
5. triceps l.h.	0	0	0	0.3	2.2	0.6	5. pronator quad.	0	0	0	0.2	1.2	0.4
6. infraspinatus	0.3	0	0	0	0.7	1.0	6. pronator teres	0.9	0.2	0	0	0.4	0.8

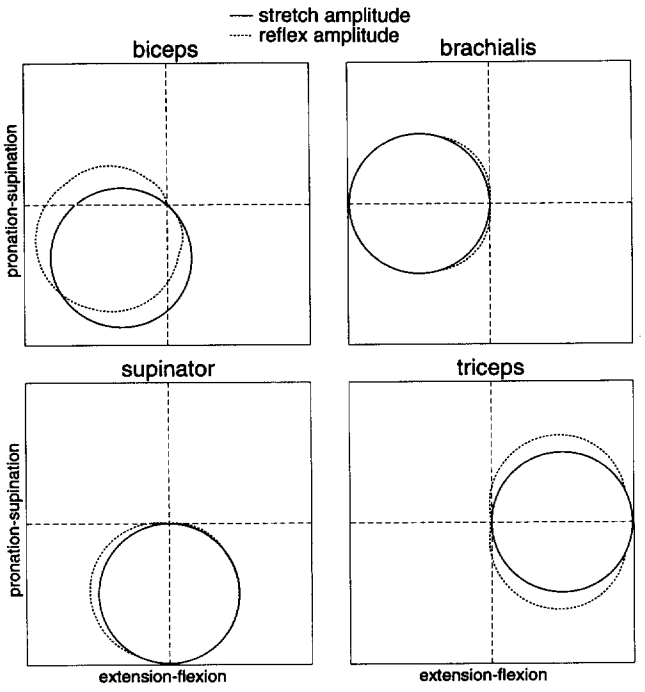

Fig. 7. Polar plot of the relative amplitudes of the stretch reflex for the muscles of the elbow system as a function of the direction of the displacement. For comparison, the relative amplitudes of muscle stretch during the same displacements are plotted in the same figures. The positive x-axis denotes a displacement in the flexion direction, and the positive y-axis a displacement in the supination direction. For the muscles which are not shown in this figure, the amplitudes of the stretch reflex follow from the symmetry in the system. For calculation of the reflex amplitude, the connections of Fig. 5b are used

maximum reflex amplitude do not coincide with the directions of maximum muscle stretch (deviations of up to 20°), and muscles show reflexes in directions in which they are not stretched.

The predicted reflex amplitude can be compared with the data of experiments done on the stretch reflex in human arm muscles. Smeets and Erkelens (1991) showed that for an arm in the same position as our planar arm model, an extension of the shoulder produces a short latency reflex in m. pectoralis major, m. biceps and m. brachialis. For a similar displacement the model of the planar arm gives reflexes in the equivalent muscles. Gielen et al. (1988) showed that a rotation of the elbow in the pronation direction results in a short latency reflex in m. biceps and m. supinator. In general, no short latency reflex was seen in the other muscles, although a few subjects sometimes showed a short latency reflex in m. brachialis or m. triceps as well. For the same perturbation, our model gives large reflexes in the supinator and biceps, and very weak reflexes in the brachialis and triceps. These features of our model were robust with respect to variation of $\varepsilon_{1,2}$ or v_0.

To give an indication of the influence that non-autogenic connections have on the quality of the reflex, we compare the reflex due to the simulated connections with the quality of reflexes that are due to autogenic ($J_{ij} = \delta_{ij}$) connections only. The autogenic reflexes give $E = 0.24$ for the planar arm, and $E = 0.22$ for the elbow system. When the connections presented in Fig. 5 are used, the movement is generally better directed against the displacement: $E = 0.19$ for the planar arm and $E = 0.12$ for the elbow system.

Changes of 50% in either values of $\varepsilon_{1,2}$ or v_0 in (6) and (7) have only a slight ($< 5°$) effect on the direction of the maximal reflex response of a muscle. The resulting values of E were between 0.18 and 0.24 for the planar arm, and between 0.10 and 0.15 for the elbow system.

Changes in the anatomical parameters have a clear effect on the behaviour of the system. Changing these parameters changes the combinations of motoneurone pool activities used for voluntary movements. This has a considerable effect on the connections J_{ij} and therefore on the direction of the reflex responses. For both systems 50% changes in one of the anatomical parameters yields a variation in E of between 0.12 and 0.26. The quality of the autogenic reflexes also changes by a change in anatomical parameters; in all cases considered, the quality of the reflexes based on the connections calculated by (7) is better than the quality of the autogenic reflexes.

From the stretch reflexes from our model, we calculated a stiffness tensor $\mathbf{R}$ according to (10). Variations of parameters in our model have a considerable effect on the values of $\mathbf{R}_{ij}$. The relative magnitude of these values was more or less constant: $\mathbf{R}_{11} > \mathbf{R}_{21} > \mathbf{R}_{12} > \mathbf{R}_{22}$. The mechanical behaviour of a real limb to position perturbations is not only governed by the stretch reflex. The mechanics of the muscles plays an important role, too. It is therefore not possible to compare the stiffness predictions with experimental data, such as those of Flash and Mussa-Ivaldi (1990). However, one aspect of measured stiffness has to be due to effects of the stretch reflex. If the projections between the afferents and the motoneurones of two muscles are not symmetrical, this will in general lead to an asymmetric stiffness tensor and thus a rotational component in the stiffness field (Hogan 1985). The asymmetric connections from our model give rise to an asymmetric stiffness tensor, with $\mathbf{R}_{21} > \mathbf{R}_{12}$. Experimental data (Table 1 in Flash and Mussa-Ivaldi 1990) show small asymmetries in the measured stiffness. They did not test the configuration we used in the model; for most arm configurations, however, the asymmetry is in the same direction as in our model. So, also in this respect, the connections resulting from our simulations are not contradicted by experimental data.

4 Discussion

We have shown that in a simple model system of the human arm, hebbian mechanisms may give rise to a rather realistic reflex organisation. It might seem counterintuitive that a hebbian mechanism can be the basis of this development because muscle spindles fire when a muscle is stretched, and the firing of motoneurones causes muscle contraction. However, the organisation resulting from our model is for an important part the result of the stretch of the antagonists when they are activated to brake the movement.

The model yields at least qualitatively reasonable connections between afferents and motoneurone pools. The monosynaptic reflex resulting from these connections is in general better directed than a pure autogenic reflex. The rotational component of the stiffness field resulting from our simulations is in agreement with the measured value. The results are not very sensitive to the exact choice of the parameter values. Moreover, the exact formulation of the learning rule (7) is not crucial for these results: in another study (Smeets 1991), a slightly different learning rule yielded comparable results.

No information about the quality of reflex activation is used for the development of the monosynaptic connections; the development is based only on local information concerning the simultaneous activity of afferents and efferents during voluntary movement. The results of the simulations show how a simple model of the motor system can develop realistic patterns of co-ordination. Since the real nervous system has more types of neurones and a much more complex organisation (e.g. McCrea 1986), a full description needs more mechanisms and parameters. Unfortunately, these mechanisms and parameters are generally not known and therefore hard to implement.

The simplicity of a model contributes to the understanding of its behaviour. However, some of the simplifications we made deserve more discussion, because they could have major effects on the results. One of the major simplifications in our model is the treatment of time. In our model we assumed that all signals could be averaged separately over the two phases of movement. In performing this averaging, we assumed that the delays were negligible. Two-sources of delay are important in the human arm. The first one is the conduction time for signals to travel from the spinal cord to the muscles and back (about 20 ms). The second is the delay between the arrival of the motoneurone signal on a muscle and the resulting contraction (about 70 ms). Having neglected these delays, we thus assumed that both the acceleration and deceleration lasted much longer than 100 ms. On the other hand, we justified neglecting centripetal and Coriolis forces by assuming that the movements were over a short distance. These two assumptions are somewhat contradictory, unless the movements are very slow.

For faster movements, we can estimate the effect of taking real delays into account. One effect of the delays is that the agonist hardly shortens during its activation, and that the antagonist is activated approximately at maximum lengthening velocity. This means that the correlation between the afferent activity and the motoneurone pool activity will be better than in our model. On the other hand, the delays introduced by conduction times might cause a slight decrease of this correlation. The overall effect would be an increase of the correlation. Since the performance of the model depends on this correlation, taking delays into account will improve the behaviour of the model.

Another important simplification in our model is the synchronous activation of intrafusal and extrafusal muscle fibres. For the model, it is crucial that the activation of these two types of fibres is positively correlated. If they are not simultaneously active, there will be no correlation between the motoneurone pool activity and the spindle activity, which results in weak autogenic connections. Correlation, however, does not require 100% synchronous activation of intrafusal and extrafusal muscle fibres. So some modulation of the α-γ-coactivation will not hamper the behaviour of the model. If we want to extend the model to study the control properties of the reflex, loosening of the α-γ-coactivation will be needed. Otherwise, the positive feedback present in the model can lead to instabilities.

One of the most important features of this model is that it learns without testing its performance. It improved the stretch reflex without testing this reflex, thus without any knowledge of the errors in the system. The performance of the system could further be improved by extending the model with some form of learning from errors. Wolpaw and Carp (1990) showed in monkeys that the connections responsible for the short latency stretch reflex can indeed change by learning from errors. A possibility for doing this in our model is for instance to change the value of the decay parameters according to the quality of the performance.

In this paper we have presented a model for the development of neural connections for motor control. The basis of this development is the correlation in the activity of motoneurones and afferents. Neurones, muscles and dynamics of movements are modelled as simply as possible. Apart from the anatomical parameters (listed in Table 1), only three parameters (v_0, ε_1 and ε_2) can be chosen freely in this model. One set of parameter values leads to a behaviour of the model which conforms reasonably to experimental results obtained for the stretch reflex in two systems with two degrees of freedom. Furthermore, this study demonstrates that complex, well-tuned structures can be the result of quite simple learning processes.

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