Biol. Cybern. 72, 185–196 (1995)

Biological Cybernetics

The three-loop model: a neural network for the generation of saccadic reaction times

Burkhart Fischer, Stefan Gezeck, Wolfgang Huber

Brain Research Unit, Institute of Biophysics, University of Freiburg, D-79104 Hansastr. 9, Freiburg, Germany

Received: 29 November 1993/Accepted in revised form: 8 August 1994

Abstract. This paper presents a computer simulation of the three-loop model for the temporal aspects of the generation of visually guided saccadic eye movements. The intention is to reproduce complex experimental reaction time distributions by a simple neural network. The operating elements are artificial but realistic neurones. Four modules are constructed, each consisting of 16 neural elements. Within each module, the elements are connected in an all-to-all manner. The modules are working parallel and serial according to the anatomically and physiologically identified visuomotor pathways including the superior colliculus, the frontal eye fields, and the parietal cortex. Two transient-sustained input lines drive the network: one represents the visual activity produced by the onset of the saccade target, the other represents a central activity controlling the preparation of saccades, e.g. the end of active fixation. The model works completely deterministically; its stochastic output is a consequence of the stochastic properties of the input only. Simulations show how multimodal distributions of saccadic reaction times are produced as a natural consequence of the model structure. The gap effect on saccadic reaction times is correctly produced by the model: depending only on the gap duration (all model parameters unchanged) express, fast-regular, and slow-regular saccades are obtained in different numbers. In agreement with the experiments, bi- or trimodal distributions are produced only for medium gap durations (around 200 ms), while for shorter or longer gaps the express mode disappears and the distributions turn bi- or even unimodal. The effect of varying the strength of the transient-sustained components and the ongoing activity driving the hierarchically highest module are considered to account for the interindividual variability of the latency distributions obtained from different subjects, effects of different instructions to the same subject, and the observation of express makers (subjects who produce exclusively express saccades). How the model can be extended to describe the spatial aspects of the saccade system will be discussed as well as the effects of training and/or rapid adaptation to experimental conditions.

Correspondence to: B. Fischer

1 Introduction

The generation of a visually guided saccadic eye movement is a very complex operation of the brain. It includes neural activity in the afferent visual system, the central visuomotor structures, and the efferent oculomotor system (retina, lateral geniculate nucleus, striate cortex, superior colliculus, parietal cortex, frontal eye fields, supplementary eye fields, prefrontal cortex, basal ganglia and brain stem). The optomotor system, the serial and parallel activation and the coordination of many structures resulting in a saccade that brings the fovea from one point of interest to the next can be described by a mathematical model or by artificial neural networks and the corresponding computer simulations. The fact that the natural system works with large numbers of elements (nerve cells) each receiving and producing large numbers of impulses with random intervals at various mean densities makes it difficult to understand how so many structures may cooperate in the generation of a saccade. Another difficulty arises from the fact that the saccade system is to some extent under voluntary control, but to some extent it may also work automatically. Psychological factors like expectation and prediction, like attention and motivation, like age and experience in the performance of laboratory saccade tasks, all influence the generation of saccades.

The parameters characterizing a visually guided saccade are its size, velocity and reaction time (SRT). During the last ten years a controversial observation was the occurrence of saccades after extremely short reaction times, called express saccades (abbreviation ES) in monkeys (Fischer and Boch 1983) and in human subjects (Fischer and Ramsperger 1984). These saccades often occur as a separate early peak in a bimodal or even trimodal reaction time distribution. While it has long been known that at least two systems exist, the frontal and the tectal system, for saccade generation (Schiller

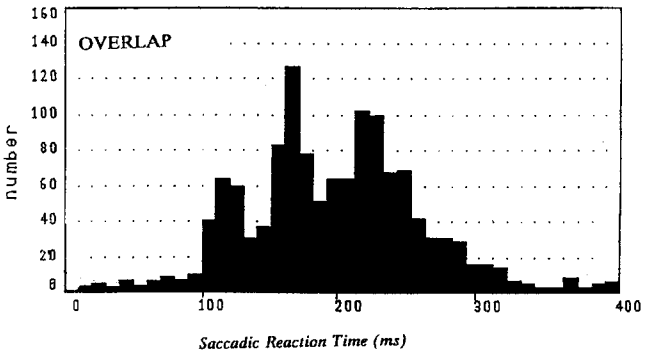

Fig. 1. The saccadic reaction times (SRTs) pooled from seven teenagers are displayed in a frequency histogram (bin width 10 ms). The distribution consists of three main peaks, some anticipations and some late reactions (above 350 ms). The data were collected under conditions in which the fixation point remained visible while a visual saccade target was presented randomly at 4 deg to the right or left (overlap task)

et al. 1980), SRT distributions often contain at least three modes (Fig. 1): ES, fast regular saccades and slow regular saccades (Fischer 1987, Fischer et al. 1993). ES are favoured in a gap task, where the fixation point is turned off some time before the target is presented; slow regular saccades are prominent in the overlap task, where the fixation point remains on until the end of a trial. The questions are: why are some SRTs long and others short, even when the same task is used? How is the multimodality generated? Why are not all saccades of the same short latency? The controversy not only concerns the existence of ES but also the multimodality of the distribution: some subjects produce ES in bi- or trimodal distributions as naive subjects, others need practice. The multimodality may disappear in the same subject depending on the physical and psychological conditions. While no doubts were raised with respect to data from monkeys (Rohrer and Sparks 1993), the existence of human ES was even denied (Kingstone and Klein 1993). A discussion of this problem is given by Fischer and Weber (1993).

The observation of the three modes in the SRT distributions (besides anticipatory responses and misses or late responses) has led to the idea that three main processes involved in the generation of visually guided saccades exist. Using the basic anatomical and physiological facts of the primate optomotor system, the three-loop model first proposed by Fischer (1987) takes up this idea and, in fact, describes correctly a number of observations on SRT in man and monkey. A review of the corresponding experimental facts is given elsewhere (Fischer and Weber 1993).

In this paper, we consider an artificial neural network. Its structure follows in a simplified way the known anatomical and physiological facts about the visuomotor system of the monkey. Instead of using the averaged impulse frequency as the signal (Dominey and Arbib 1992), we use the sequences of randomly distributed impulses as such. Only for certain purposes will we look at the average frequencies calculated from such impulse trains. The model elements are neural elements that mimic the synaptic transmission and impulse generation of real neurons. The purpose is to describe experimentally observed SRT data, for example, multimodality – its appearance and disappearance without changing the model parameters – on the one hand and to explain the occurrence of experimental phenomena that have not been used in the construction of the model, for example up and down modulations and displacements of the several peaks in the distributions or the occurrence of anticipations under certain conditions or the existence of express makers (subjects who almost exclusively produce ES irrespective of the task). In its present form, the model operates in the time domain only. In terms of neuroanatomy and neurophysiology this means that we consider only a restricted volume of the corresponding neural structures, many of which are retinotopically organized. The possible extension of the model into the space domain will be discussed.

2 Outline of the model

The existence of different characteristic modes in the distributions of SRTs is the basic observation under consideration. An example of a multimodal SRT distribution is shown in Fig. 1 (Fischer et al. 1993). The first group in the distribution of the SRTs consists of very short reaction times (below 90 ms), corresponding to anticipatory reactions (Wenban-Smith and Findlay 1991). The next group forms a clear peak just above 100 ms and consists of ES, which are usually rare or completely absent in the distributions of naive adult observers performing the overlap task (Mayfrank et al. 1986). The peak in the range of 160 ms consists of fast regular saccades, and the third peak above 200 ms forms the group of slow regular saccades. There are, in addition, some saccades of very long reaction times scattering between 270 ms and 400 ms or more. Such saccades are occasionally observed when the subject for some reason fails to make a saccade in direct response to the onset of the target.

Figure 2 shows the principle of the three-loop model as proposed by Fischer (1987). Besides an afferent visual module 'VIS' and an efferent motor module 'MOT', there are three central modules 'COM', 'DEC' and 'ATT'. We

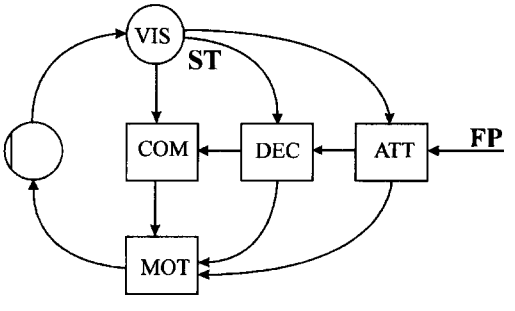

Fig. 2. Schematic diagram showing the five modules organised in three loops. The 'VIS' module feeds into the centre modules 'ATT', 'DEC' and 'COM' that in turn feed into the 'MOT' module. FP and ST denote the activity induced by fixation point offset and target onset, respectively

use these labels because of a possible functional interpretation of the nature of the modules, including their possible structural cortical or subcortical correlate, i.e. computation of saccade metrics, decision to make a saccade and attentional disengagement. FP and ST denote the fixation point and the stimulus, respectively.

Each of the central modules is a member of a loop from the VIS module to the MOT module. The central modules are connected with each other in the order shown (Fig. 2). This kind of connectivity makes it possible for activity to occur in a serial manner (VIS-ATT-DEC-COM-MOT) or in a parallel manner (VIS-COM-MOT; VIS-DEC-MOT; VIS-ATT-MOT). Still other combinations are possible, like VIS-DEC-COM-MOT or VIS-ATT-DEC-MOT. The different combinations will have different probabilities of occurrence depending on the exact nature of the connections, the delay times between the modules, and the driving input activity into the model resulting from retinal and/or extra-retinal events.

The modules consist of a number of interconnected neural elements projecting to another number of neural elements in the next module (see Fig. 3). The neural elements are artificial neurons receiving and sending impulses according to the summation of their synaptic potentials.

The impulse trains feeding into the modules mimic the electrophysiological recordings from retinal and/or cortical cells whose receptive fields were stimulated by more or less adequate visual stimuli. The onset of the target will produce impulse trains in the afferent visual system: one of them is shown in the top of Fig. 3. These will feed into all three central modules. The module 'ATT' also receives an extra input (besides that through the connection with ST) of the same stochastic nature but of different origin: the origin may be a retinal (for example, the offset of the fixation point, FP) or any other sensory event, or even an internal event which activates the input lines to 'ATT'. After convergence at the MOT module, the output activity will trigger a saccade. Within each of the modules, there will elapse a certain time to raise the activity in response to the input, and each connection between the modules will introduce a certain delay time.

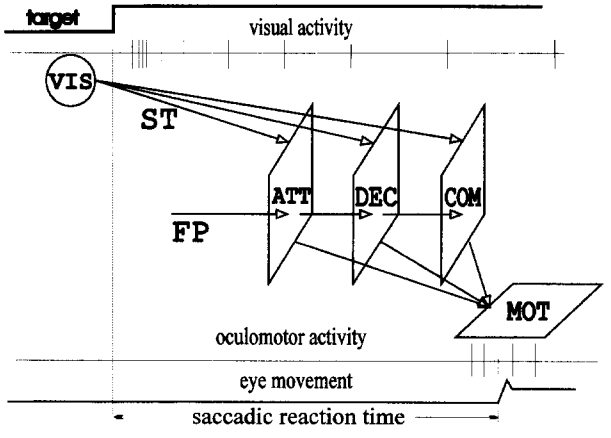

Fig. 3. The timing scheme of the model. The target onset stimulus through the 'VIS' module excites the centre modules 'ATT', 'DEC' and 'COM'. The time course of the excitation is governed by the delay times and strengths of the couplings. All centre modules feed into the 'MOT' module that eventually triggers the saccade

The stochastic properties of the input impulse activity lead to stochastic variations of the time between the beginning of the input activity and the beginning of the output activity of the last module, even though the network itself is completely deterministic. The other alternative of using stochastic variations of the connections within a block of trials does not make much physiological sense, because neither the synapses and their coupling strength nor the conduction times will change by any appreciable amount within seconds (the duration of a trial) or within 10 to 20 min (the duration of a block of trials). Systematic changes of these parameters should be taken into consideration when the effects of daily practice and the adaptation to the experimental conditions are studied.

3 Structure of the neural elements and the modules

Each module M is built of a number N_M of elements. Each element is a model neuron. Within each module every element is connected to all others. Furthermore, each element of a module projects to each element of those modules to which it is connected according to the three-loop idea (Fig. 3). For example, each of the ATT module's neurons projects to the other neurons within the ATT module as well as to the neurons in the DEC and the MOT modules. A neural element sends an impulse whenever its internal potential exceeds a threshold value; the dynamics of the internal potential is controlled by a linear differential relaxation equation and driven by the incoming impulses. This behaviour of the artificial neurons has been used earlier (Buhmann and Schulten 1986).

The state of the neural element with label i is described by its internal potential $U_i(t)$. Time t is discrete, with a time step Δt of 1 ms. $U_i(t)$ specifies the momentary sensitivity of the neuron and its ability to fire an action potential in the next time step. It is controlled by a first-order differential equation:

$$\frac{U_i(t+\Delta t) - U_i(t)}{\Delta t} = -\frac{U_i(t)}{T_R} + \alpha A_i(t) \quad \text{if } U_i(t) < U_{thr}$$

$$U_i(t+\Delta t) = U_F \quad \text{if } U_i(t) \geqslant U_{thr}$$

When the potential $U_i(t)$ exceeds the threshold value U_{thr}, it is reset to the refractory value U_F, and an impulse is generated according to:

$$o_i(t+\Delta t) = \begin{cases} 1 & \text{if } U_i(t) \geqslant U_{thr} \\ 0 & \text{else} \end{cases}$$

That is, $o_i(t)$ describes the output impulse. $A_i(t)$ is the summed neural activity function of impulses converging to cell i.

$$A_i(t) = \sum_{j=1}^{4N_M} w_{ji}\, o_j(t - d_{ji}) + \sum_{k=1}^{2N_1} w_{ki}^{(a)}\, o_k^{(a)}(t - d^{(a)})$$

In the first sum, the impulses coming from the other neural elements (N_m denotes the number of elements in a module) are weighed against the corresponding synaptic strength w_{ji}, which represents the strength of the effect of an impulse sent by element j to element i. w_{ji} of non-existing connections (e.g. recurrent pathways) are set to zero. d_{ji} is the delay time between these two elements. w_{ji} are static (time-independent) parameters of the network. The second sum represents the input that is fed into the network (N_I denotes the number of input lines for each of the two inputs, ST and FP). The other constants are:

T_R: the relaxation time of the internal potential, 20 ms
U_F: the refractory potential that the element takes after firing, -15 mV
U_{thr}: the threshold potential, $+20$ mV
α: global scaling factor for the coupling strength

The factor α could, of course, be included in the coupling constants w_{ji} and $w_{ki}^{(a)}$, but it is convenient to have a global scaling factor.

The overall network of neurons is structured into the four modules 'ATT', 'DEC', 'COM' and 'MOT' by means of assigning equal strengths to couplings between elements from the same modules. The fifth module 'VIS' in the three-loop model is represented by the input that is fed into the network.
The coupling is organized as follows:

1) $w_{ji} = W_{XY}$, where X is the module containing element j and Y, the module containing element i. Since there are four modules, the w_{XY} form a 4×4 matrix with zero-values for non-existing connections. All w_{ji} are fixed parameters.
2) d_{ji} are random numbers drawn from the uniform distribution with mean value m_{XY} and width σ. The m_{XY} also form a 4×4 matrix, whereas σ is a real number. Once selected for a given block of simulated trials, the values remain constant. When a new block is started, the values will be selected again, but – by definition – they will always have the same mean value and the same scatter.
3) $w_{ki}^{(a)} = w_{ZY}^{(a)}$, where Z is the input module containing $o_k^{(a)}$ and Y the module containing element i. Since there are two input modules, FP and ST, the $w_{ZY}^{(a)}$ form a 2×4 matrix.

The stochastic 'smearing out' of the delay times d_{ji} is physiologically motivated, because it is unrealistic to assume that all conduction times and all synaptic delays are exactly the same. For the model it is necessary to use a randomly selected set of delay times to avoid synchronicity of the impulse activity of the elements of one module. The values of d_{ji} are stochastically initialized once for a given simulation run, as described, and remain constant. This means that the network itself is operating completely deterministically.

It should be mentioned that the model at this stage does not use any inhibitory connections. No feedback is used between the modules.

4 Characteristics of the input

As indicated in Fig. 3, there are two input lines, FP and ST, which feed impulses into the ATT, DEC and COM modules (the abbreviations stand for 'fixation point' and 'stimulus', respectively). The onset of the activity ST is considered the consequence of an external event, i.e. the onset of a saccade target, and drives all three central modules. FP may be activated by an external visual event, like the offset of the fixation point, but it may also be interpreted as being activated by stimuli of another modality or by internal events. It is this input line which represents the influence of the 'rest of the brain' on the network. In the easiest case, we consider the FP activity as being solely produced by the offset of a fixation point. Later, however, we will also see the effect of adding spontaneous activity to this input, which does not necessarily have to do with fixation but, for example, with attentional states.

The impulse trains generated for the input FP and ST are given by $o_k^{(a)}(t)$ $(k = 1, \ldots, N_1)$ and by $o_k^{(a)}(t)$ $(k = N_1 + 1, \ldots, 2N_1)$, respectively. The $o_k^{(a)}(t)$ are binary 0/1-valued functions, similar to the element outputs $o_i(t)$. They are the $2N_I$ outputs of the 'VIS' module and are generated according to the probability functions $p_{ST}(t)$ and $p_{FP}(t)$. This is the main point where randomness enters the model. All dynamics other than the production of the input impulses $o_k^{(a)}(k)$ from the functions $p_{ST}(t)$ and $p_{FP}(t)$ are deterministic.

$$\langle o_k^{(r)}(t) \rangle = P(o_k^{(r)}(t) = 1) = \begin{cases} P_{FP}(t) & \text{for } 1 \leqslant k \leqslant N_I \\ P_{ST}(t) & \text{for } N_I + 1 \leqslant k \leqslant 2N_I \end{cases}$$

During a time period in which the p-functions are constant, the intervals between impulses are thus exponentially distributed. The probabilities $p_{FP}(t)$ and $p_{ST}(t)$ as functions of t are given by:

$$p_Z(t) = \begin{cases} A_Z^{spon} & \text{for } t < T_Z \\ A_Z^{spon} + F_Z(A_Z^{tr}(t) + A^{sus}) & \text{for } t \geqslant T_Z \end{cases}$$

where Z = FP or ST F_Z is the factor for the transient and the sustained component. While A^{sus} remains constant as well as A_{ST}^{spon} and A_{FP}^{spon}, A_{ST}^{tr} and A_{FP}^{tr} decay exponentially:

$$A_Z^{tr}(t) = A_0^{tr}\, e^{(T_Z - t)/T_{ton}} \quad \text{for } t \geqslant T_Z$$

After stimulus onset at time T_{ST}, or fixation point offset at time T_{FP}, the transient component rises instantaneously to its maximum and decays exponentially with a decay time T_{ton}. This is a typical time course of the responses of visual neurons in the retina, the lateral geniculate nucleus or the visual cortex. $p_Z(t)$ is a direct measure of the average impulse frequency in the input lines. We will consider below the effect of adding spontaneous activity to FP without changing the absolute peak activity and without changing any other parameter of the model.

5 Selection of constants and parameters

An actual simulation of the model needs the specification of 4 values to describe the characteristics of a single neural element (micro-internal constants), of 19 values to define the modules and the input lines (macro-internal constants) and 2 external values, namely T_{FP} and T_{ST}, to characterize the physical conditions of the experiment to be simulated. However, only 2 of them are considered free parameters that can be fitted to reproduce certain experimental results. The other values are considered constants specified by physiological data other than the special reaction times. We will specify the values step-by-step proceeding from the internal values (describing a neural element, a module, the connectivity between the modules, the dynamic properties of the inputs) to the external values specifying strength of the stimuli and the experimental conditions to be simulated.

5.1 Micro-internal constants

A single neural element is completely characterized by four values:

(i) The relaxation time of the internal potential, $T_R = 20$ ms. This value closely corresponds to the decay time of excitatory postsynaptic potentials.
(ii) The refractory potential after the element has fired an impulse, $U_F = -15$ mV.
(iii) The threshold potential, $U_{thr} = +20$ mV.
(iv) The global scaling parameter for the coupling strength, $\alpha = 2.8$

5.2 Macro-internal constants

A single module is characterized by another four values.

(i) The number N_M of neural elements within a module. If one uses a too small number, the variability in the impulse activity will be too large to obtain a satisfying, reliable functioning of the module. We calculated the reliability of the impulse activity in an all-to-all connected network for an increasing number N_M of neural elements driven by a stationary input. The reliability R is defined by the normalized variance V of the mean number of impulses at any time step (1 ms) averaged over the time of the stimulus duration of 1000 ms: $R = 1 - V$. It was found that R increases until $N = 8$ or 10 and decreases again for N larger than 24. The reason is that for large N, the number of randomized parameters in the network (time delays and coupling constants) increase, counterbalancing the reduction of the variance due to more elements. In the range between $N = 10$ and $N = 24$, the reliability R stays about constant at a value of 0.7 (E. Hausenblas, unpublished). We used $N = 16$ neural elements in each module.

(ii) Within each module all elements are connected to each other (all-to-all) with identical coupling constants $w_{ji} = 0.78$. This value was empirically found through the condition that the modules output impulse frequency lie in a realistic range given a realistic input frequency between zero and 250 imp/s.

(iii) The mean delay time between the elements within a module was set to 10 ms. This time includes a synaptic delay, typically 2–3 ms, and a conduction time accounting both for axonal and dendritic transport within the module.

(iv) The scatter σ of the delay times was set to 30%. (This does not mean that the delay time is not a constant: for each block of simulated trials, a set of d_{ij} values was selected randomly and then held constant. If one repeats the simulation by initializing another block of trials, the d_{ij} values are selected again but with the same mean value and the same scatter σ.)

The network of modules that forms the three-loop model needs the specification of the delay times (5 values) and the coupling constants between the modules (5 values). So far, we have not used SRT data to specify constants or parameters of the model. Now, we consider the position of the three peaks in the SRT distribution (see Fig. 1).

Delay times. The mean delay times $m_{ATT,DEC}$ between the ATT and DEC modules and $m_{DEC,COM}$ between the DEC and COM modules are specified by the difference in position of the main peaks in the experimental reaction time distributions: slow regular − fast regular = 50 ms = $m_{ATT,DEC}$; fast regular − express = 50 ms = $m_{DEC,COM}$. The mean delay times between the three central modules and the MOT module ($m_{ATT,MOT} = m_{DEC,MOT} = m_{COM,MOT}$) were set to 30 ms each in order to centre the earliest peak at about 100 ms. [To be as realistic as possible, we introduced also a constant delay of 30 ms for the visual afference (response latency of neurons in the visual cortex) and another 20 ms for the oculomotor efference (latency of saccades elicited by electrical stimulation of the brainstem); these two constants have only the effect of adding 50 ms to the reaction times.] The shortest time between a visually driven input impulse and the corresponding output impulse of the MOT module therefore is 80 ms. The actual delay times d_{ij} were selected in the same way and had the same scatter as those for the internal delays. The scatter was again 30% as for the internal delay times.

Coupling constants. The five coupling constants w_{xy} have no direct physiological correlate that could be accessed experimentally. They were found by trial-and-error until and fastest reaction time mode was never obtained when only ST was activated (overlap trials, see below). On the other hand, it was required that the fastest mode dominate the distribution, when FP was activated 150 ms before ST became active (gap trials). In any case, the set of coupling constants have to guarantee that the network generates – at least in the majority of trials – a saccade; thus, the couplings must not be too weak. On the other hand, the couplings must be weak enough to let the network become inactive when the input is turned off. We set the coupling constant between one module and the following module in the succession ATT-DEC-COM-MOT to 0.28. This concerns the three coupling constants $w_{ATT-DEC}$, $w_{DEC-COM}$ and $w_{COM-MOT}$. To guarantee that a saccade can only be generated when the activity in the ATT and the DEC modules is high, the coupling between the ATT and DEC modules to the

MOT module was set to $w_{ATT,MOT} = w_{DEC,MOT} = 0.08$. Figure 4 lists all these parameters in a semidiagrammatic way.

The two input lines driving the network require specification of their transient (amplitude and decay time) and their sustained response components (three values for each line). According to the realistic response properties of neurons in the retina, lateral geniculate nucleus, or visual cortex, we set the maximal transient amplitude $A_0^{tr} = 3$, the decay time T_R of the transient amplitude to 40 ms and the sustained component to $A^{sus} = 1$. We set $A_{ST}^{spon} = 0$ and $F_{ST} = 16$ Hz. The value of the spontaneous activity A_{FP}^{spon} and the factor F_{FP} are considered the only really free parameters of the present model.

Furthermore, we have to specify the parameters for the coupling between the inputs and the modules. We chose the same value for both inputs to the ATT module because the onset of each input has to produce the same effect in the ATT module. Because the DEC module as well as the COM modules receive impulses from both the preceding module and the input ST, we set the coupling constant relating ST to these modules to the same value. Therefore, two coupling constants remain to be specified: one for the inputs ST and FP to the ATT module and another for the input ST to the DEC and COM module. The values of these coupling constants are important because they determine the relative contribution of each of the modules and therefore the size and the number of the peaks in the output distribution. These parameters were again selected to correctly reproduce the effect of increasing the gap duration from zero (no gap) to values above 300 ms. We selected $w_{ST,ATT}^{(a)} = w_{FP,ATT}^{(a)} = 0.80$ for the coupling between the inputs and the ATT module and $w_{ST,DEC}^{(a)} = w_{ST,COM}^{(a)} = 0.35$ for the coupling between ST and the DEC and the COM modules, respectively.

A saccade is triggered if one of the N_m artificial neurons of the MOT module generates an output.

5.3 External values

Finally, we have to specify the experimental condition to be simulated. Since we are considering only temporal characteristics, we just have to specify the time of target onset T_{ST} relative to the time of fixation point offset T_{FP}. Note that these two numbers do not contribute to the nature or operation of the model.

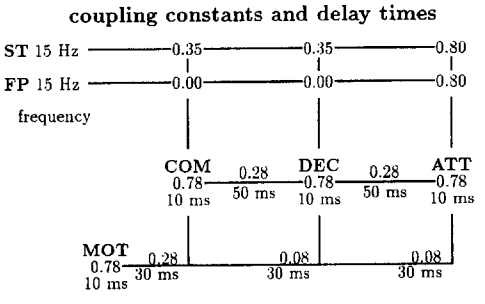

Fig. 4. Parameter set used for simulations unless stated otherwise. The delay times are given in milliseconds as indicated. The coupling constants are dimensionless numbers

Figure 4 shows schematically the parameter set used for saccade reaction time simulations. The dimensionless numbers are the coupling constants, and the delay times are given in milliseconds as indicated in the scheme. These parameters are held constant for all simulations presented in this paper.

6 Results of simulations

Before we present the results of simulations for specified experimental conditions, we consider the general question of how multimodal distributions of reaction times are generated by the model.

6.1 Multimodality

The generation of the different modes of SRT times are shown in Fig. 5. We simulated a condition in which the fixation point is turned off 250 ms before the target is presented (gap 250 task). Depending on the dynamic and stochastic activity in the modules, saccades are triggered at different times depending on whether or not the activity in the modules survive for a long time: there is no special mechanism for generating these modes, they occur as a consequence of the stochastics in the input activity, of the connectivity of the network, and of the temporal conditions of the experiment. Figure 5 shows three trials from a block of simulations. In the upper part the ATT module becomes active as a consequence of the offset of the FP, and it stays active with some variations over at least 300 ms. After a delay, the DEC module is activated by the ATT output and also stays active. Finally, the COM module is activated by both the onset of ST and the output activity of the DEC module. All three together activate the MOT module which finally triggers a saccade after another (efferent) delay. The result is a reaction time of 98 ms, which contributes to the express peak. In the middle, another trial is depicted from the same block. Here the ATT module loses some of its activity after 100 ms, and therefore the DEC module receives a weaker input and does not stay active. ST onset reactivates the DEC module, and after a delay the COM module is activated. Therefore, in this trial the result is a longer reaction time of 144 ms, which contributes to the fast regular peak. In the trial shown at the bottom, neither ATT nor DEC remain active, and it takes ST to activate all three modules in a serial way. The result is a still longer reaction time of 203 ms, which contributes to the slow regular peak.

6.2 The gap effect

The characteristics of the model imply qualitatively that when time elapses between the offset of the fixation point and the onset of the target (gap task), activity builds up in the network such that when the target appears, the activity related to target onset can lead to a saccade earlier than is the case when the fixation point remains on (overlap task). The difference in the mean reaction times

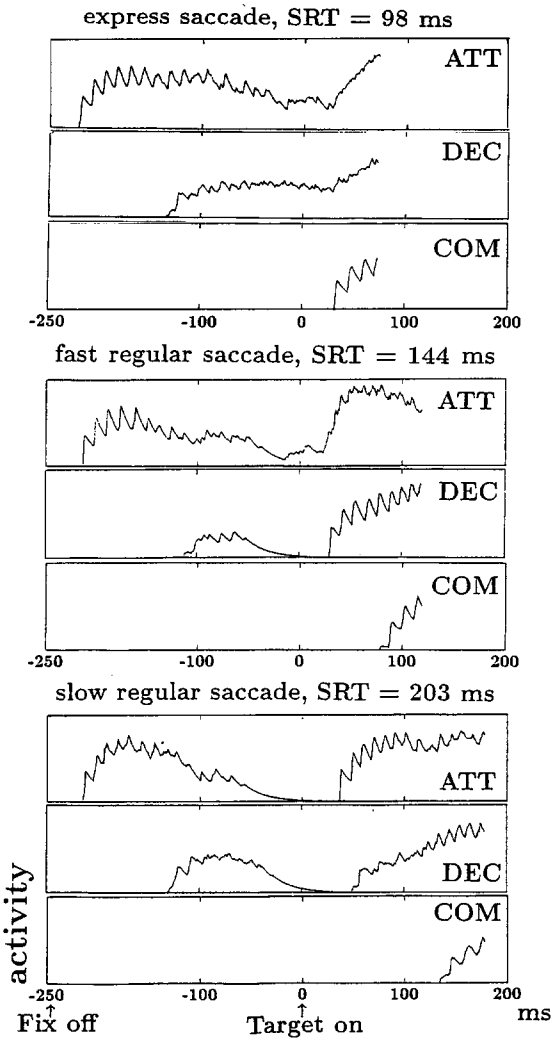

Fig. 5. The average impulse frequency activity of the neural elements within each of the three modules is displayed as a function of time in a gap 250 ms task. The traces end at the time of saccade trigger. Three different trials with the same model parameters are shown. They result in an express, a fast regular and a slow regular saccade, respectively. Depending on the activity of the modules at the time of target onset, saccades are trigged at different times

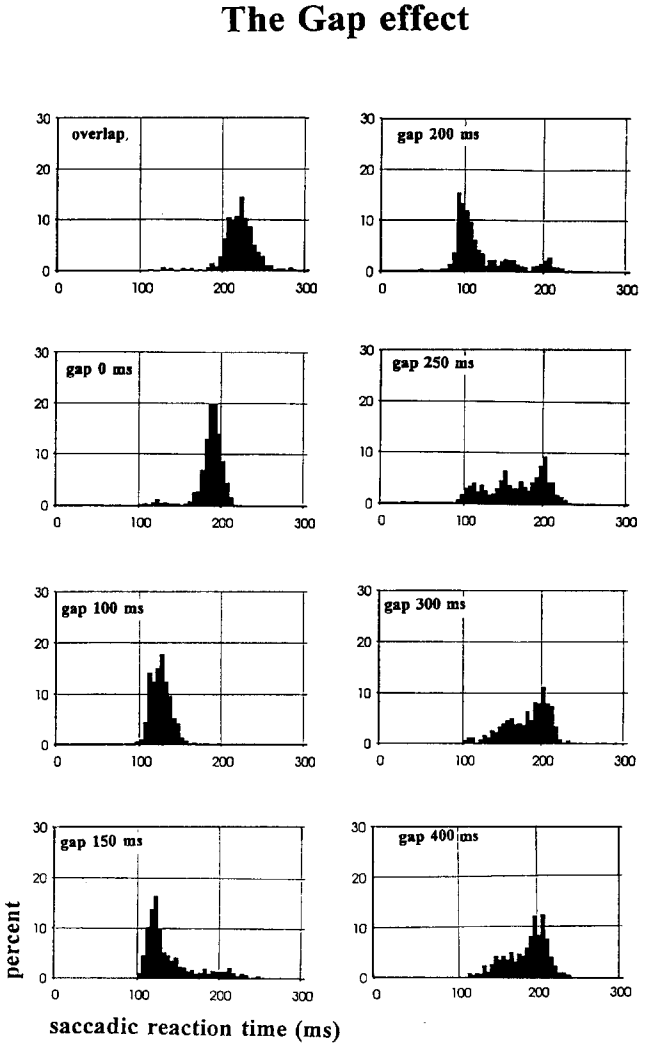

Fig. 6. SRT distributions obtained by varying the time of target onset and holding all other parameters constant. Each histogram (binwidth 5 ms) contains 500 values. Increasing the interval between fixation point offset and target onset, starting with the overlap condition, the distribution narrows and shifts towards shorter times. For gap values above 150 ms, the distribution becomes multimodal, and the fraction of ES decreases in favour of fast regular saccades

between the overlap and the gap task is a well-known experimental finding (Saslow 1967) and has been called the gap effect.

A series of simulations for different gap durations from negative (overlap) to positive values is shown in Fig. 6. The top distribution at the left side was obtained when the fixation point remained on for 1000 ms, certainly longer than the longest reaction time (overlap paradigm). The 'classical' condition, when the fixation point is turned off at the same time as the target is presented, is depicted just below (gap 0 ms or single-step paradigm). Increasing the gap duration in steps of 50 ms changes the SRT distribution in a rather characteristic way. Short gaps of about 150 ms lead to a decrease of the reaction time as compared with overlap trials. Multimodality occurs clearly for gaps between 200 and 400 ms.

Several aspects of this series of simulations are not reproductions of what has been used to construct the model, but rather appears as correct byproducts of the model's way of operation:

(i) The early peak of ES is reduced and eventually disappears altogether for gap durations above a certain optimal value (ca. 200 ms).
(ii) Bi- or trimodal distributions are obtained only for certain gap durations.
(iii) The different peaks do not occupy a given fixed position along the reaction time scale.

The way the model works does not always permit us to separate different peaks under all conditions: a given saccade cannot be classified as being of one or the other type on the basis of its reaction time only. This is particularly true for a gap duration of 100 ms, where only one

peak can be seen, which is neither of the pure express nor of the pure fast regular type. Yet all distributions are obtained using the same set of parameters with only the physical condition (the duration of the gap) varying.

It should be noted already at this point that small changes of the numerical value of the amount of activity driving the ATT module and the strength of its internal connections can lead to drastic changes in the distributions. In real experiments the variation from subject to subject may be considerable. Yet, according to the model, the differences in the saccadic system of the subjects may be only very small. We will come back to this point when discussing Figs. 7 and 8.

6.3 The latency of express saccades

It has been a problem in the past to identify the population of ES in a single experimental distribution (Kingstone and Klein 1993). If only one peak could be identified (unimodal distribution), one was left with the criterion of the mean reaction time only. If two peaks were obtained, the question still was whether the first should be considered as containing express or fast regular saccades. Implicit in these considerations is the idea that express and fast regular saccades have a constant latency. The present simulations show – in agreement with the experimental results – that both the size and the position of a given peak in the distribution can change to some extent without changing any network parameters. The most straightforward change is in the intensity of the stimulus (or target) driven input activation. In a gap 200 ms situation, weak input activity just above threshold leads to rather long latencies ($>$ 200 ms). An increase of the impulse frequency yields a small express peak (20% of all saccades) centered at 113 ms. Further increase gives rise to a single express peak with a mean latency of 90 ms. It is the whole set of distributions which allows us to identify the express peak and to see its small but significant displacement along the reaction time scale. In a single distribution it is therefore sometimes difficult or even impossible to identify express or fast regular saccades by their reaction times only.

Similarly, we can consider the latency of ES obtained in the overlap task (see below). Under otherwise identical conditions the ES under overlap conditions occurs about 15 ms later than the express peak obtained under gap 200 ms conditions, even though in this case both distributions contain only ES. The reason for these small changes in the latency of the ES is that under gap conditions the target-induced activity ST meets a preactivation in the network and leads without any appreciable delay to the generation of a saccade by activating the MOT module neurons. Under other circumstances the network is not or only weakly preactivated, and the activation due to the appearance of the target has still to contribute to the activation of the MOT module via the COM module.

What applies to the position of the express peak also applies to the position of the fast regular and slow regular peaks: they do not have exactly fixed mean latencies, rather their position depends on the experimental conditions, even when identical sets of parameters for the model are used. The same is true for the experimental data. It is often necessary to include other parameters of the saccades (e.g. their amplitudes or velocities) not considered in the present model, in order to solve this problem.

7 Modulations of the ATT-module input

Some subjects produce almost exclusively ES in gap as well as in overlap tasks, others produce variable sizes of express peaks, and still others do not form ES at all (Fischer et al. 1993). The last group of subjects may, however, learn to make different numbers of ES by daily practice. Still other subjects may exhibit a large express peak for saccades to one side but only a small express peak for saccades to the opposite side. Furthermore, a subject who produces many ES to a given target may produce only a few ES to the same target if the corresponding location is attended at the time the target is presented. Instructions of fixation also modulate SRTs. Finally, the distribution of SRTs may drastically change in relation to the context of the experiment: if a given type of trial (for example, a fixed gap duration of 200 ms) is a member of some sort of other trials (for example, two other gap durations, 300 and 400 ms) being randomly intermixed, the reaction times may change if the same type of trial (gap 100 ms) is mixed with other sorts of trials (gaps 100 and 400 ms) or is not mixed with any other trials. Corresponding experimental results have been obtained by Weber et al. (personal communication).

The question arises of how these modulations of the saccade-generating system can be described within the framework of the three-loop model. The requirement is, of course, that the network parameters themselves be kept constant. Since only the activity of the ATT module reflects the modulatory effects of other brain structures, we investigated the effect of adding different amounts of activity to FP driving the ATT module without changing any other parameters of the network.

7.1 Modulation of the transient input

The factor F_{FP} influences both the transient and the sustained component of FP, but as long as the transient component is well above the sustained component, the transient part dominates the effects. Note that this factor does not contribute to the overlap data, because FP is never activated in this condition.

Figure 7 shows the result of the simulations for three different values of F_{FP}(F_{FP} = 13, 15, 17 Hz) and four different gap durations. We set the maximal value of $p_{FP}(t)$; this is $p_{FP}(T_{FP}) = F^{*}_{FP}(A^{tr}_{FP}(T_{FP}) + A_{SUS}) = 52, 60, 68$ Hz for F_{FP} = 13, 15, 17 Hz in the upper right corner of each histogram in Fig. 7. First, note that the changes in the peak frequency considered here are only small: 8 and 16 Hz, respectively. Second, the results indicate that for short gaps the effects of increasing the activity of FP line are only small (gap 0 ms, left side of Fig. 7) or consist of an apparent shift of the SRTs to shorter values (gap 100 ms).

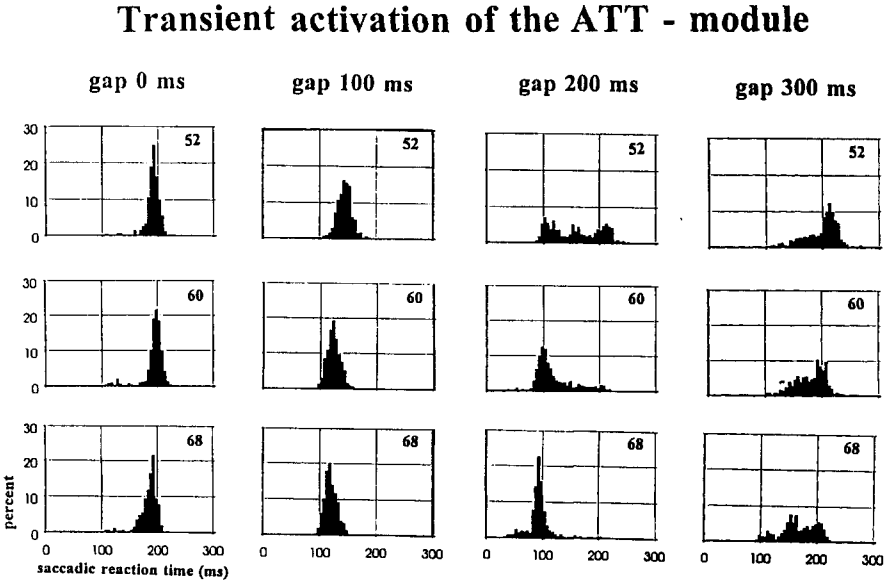

Fig. 7. Distributions for different values of F_{FP} as indicated by the numbers (binwidth 5 ms). The peak activity of FP is displayed in the *upper right corner* of each histogram. Small changes in the activation of FP line lead to strong changes for certain gap durations, but leave the overlap data unchanged

For medium gap durations (200 ms) drastic changes occur: whereas trimodal distributions are obtained for the lower value, a single express peak dominates the distributions for the higher value, and a few anticipations occur. For even longer gaps (300 ms, right side) the effects become weaker again: a few ES are favoured for the high value, but the anticipations seen for gap 200 ms disappear.

Any change in the experimental conditions, physical or psychological, that lead to a change in the strength of the activity driving the ATT module has an effect on the SRT distribution. The effect strongly depends on the duration of the gap and is absent in the overlap condition. Therefore, if a subject, for example, produces different activities in the right as compared with the left hemisphere in response to the offset of the fixation point, his/her SRT distributions may be the same for right and left directed saccades when the overlap task is used, but different when the gap task with certain gap durations is used. If now this subject's attention is manipulated asymmetrically (allocation of attention to one side by means of an attentional cue), the distributions may eventually become the same for saccades to either side.

Another example involves the intertrial effects observed when trials with different gap durations are randomly mixed in a single experimental session. When the results for a given gap duration from this 'random experiment' are compared with those obtained with the same gap duration collected during a 'non-random experiment' (i.e. when only this gap duration is used throughout the session), the subject seems to have adapted to the longest gap used in the random experiment, thereby decreasing the chances of making ES during the trials with shorter gap durations (Weber et al., unpublished observation). In the light of the three-loop model, this effect is attributed to a modulation of the FP-induced activation being lower when long gaps are intermixed.

7.2 Modulation of the spontaneous activity

Here we consider the effect of adding spontaneous activity to the ATT input line FP (without changing the absolute height of the transient activity induced by the offset of the fixation point in the case of gap trials). The strength of the spontaneous activity influences mainly the operation of the model during overlap trials, because the gap trials are dominated by the much stronger activity induced by fixation point offset. Figure 8 depicts several simulated distributions for overlap and two gap (100 and 200 ms) conditions. From top to bottom the spontaneous means firing rate is given by the numbers in impulses per second. The left column (overlap) shows that increasing the input activity changes the distributions from a broadly tuned one without any ES into bimodal distributions which exhibit fast regular and ES. Further increase (not shown) leads to single express peaks in the overlap condition.

Subjects who produce many ES in the overlap task have been found by Fischer and Weber (1990) among a group of dyslexic subjects. According to later studies (Biscaldi et al. 1994) normally reading subjects can also be found who produce exclusively ES in the overlap task. Note that according to the model the input activity ST leads to an express saccade in many cases, because the ATT and DEC modules are already activated. This implies that the subject has in many trials lost his/her voluntary control over the saccade system. We conclude from this notion that such a subject is unable to perform an anti-saccade task, where the saccades are to be made in the direction opposite to the target. Instead, one predicts that the subject makes many saccades involuntarily to the target. Indeed, this is what happens with these so-called express makers (Biscaldi et al. 1994).

Monkeys with their tectal fixation system chemically deactivated also produce many ES during overlap trials, and in addition, they make 'unwanted' saccades to suddenly appearing stimuli during periods in which they are

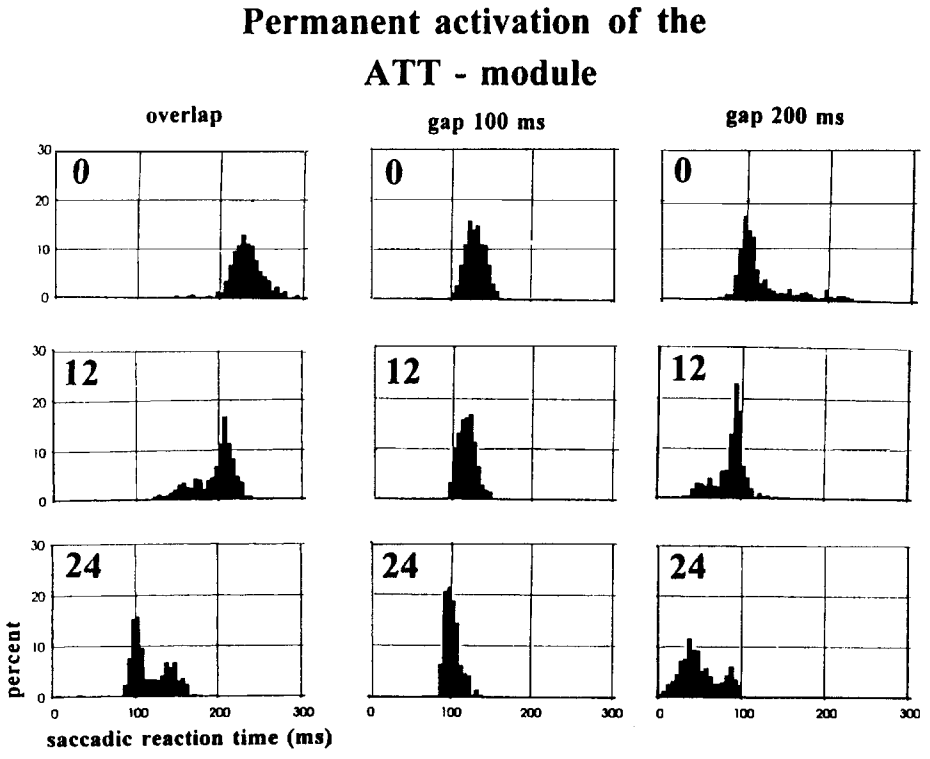

Fig. 8. Distributions for three different conditions and for three different values of the spontaneous activity of the 'ATT' module (binwidth 5 ms)

supposed to maintain fixation. These monkeys behave like the human express makers. Losing permanent activity in the fixation system means an increase of the permanent activity in the FP line.

Increasing the ATT activity in a gap 100 ms task does not have such a dramatic effect: a single peak at about 130 ms moves towards shorter values until it turns into a single peak at about 100 ms. The data obtained with gap 200 ms indicates a loss of ES in favour of responses before 80 ms. These are saccades triggered by the offset of the fixation point or – if one wishes – triggered by anticipation of the target.

Assuming that different subjects have different amounts of activity driving their ATT module, one understands why there are subjects who produce little or no ES, others who produce a clear bimodal distribution in the gap task but no ES in the overlap condition, and still others who make lots of ES in both tasks (Fisher et al. 1993). Interestingly, the express peak obtained in overlap trails occurs about 10 ms later than that in the gap 200 ms task (compare lower left with upper right diagram of Fig. 8). Even this detail is in surprisingly good agreement with the experimental results.

8 Discussion

The three-loop model was designed to simulate the temporal aspects of saccade generation: it is used to produce the SRT data obtained from monkeys and human subjects.In particular, the model provides a theoretical account for the gap effect and the multimodality observed in the distributions. A saccade can be generated by serial activation of the three central modules under certain conditions, for example under overlap conditions, when the subject makes slow regular saccades, but can be generated by parallel activation, for example under gap 100 ms condition, when an ES is produced.

Roughly, the interpretation of the model components (ATT, DEC, COM) in anatomical and functional terms could be: ATT – disengagement of fixation/attention-related activity in the parietal cortex (Mountcastle et al. 1987), the rostral part of the superior colliculus (Munoz and Wurtz 1992), the omnipauser neurons in the brainstem (Langer and Kaneko 1990) (note that an increase in the ATT activity is interpreted as a stronger disengagement which corresponds to a decrease of activity of fixation-related neurons); DEC – decision-related activity in the frontal cortex and/or the basal ganglia; COM – computation of metrics in the primary visual cortex and/or the tectal system. A more complete account for the neuroanatomical and neurophysiological facts related to the components of the model has been presented earlier (Fischer and Boch 1991; Fischer and Weber 1993).

Whereas there have been many attempts to model the process of saccade generation in terms of the transformation of the retinal error into the motor activity specifying size and velocity of the saccade (for a review, see Robinson 1981), little theoretical work is available to understand the reaction time data. A model of facilitation due to the disappearance of the fixation point has been presented by Reulen (1984). However, this model, while producing a clear gap effect, fails to produce multimodal

distributions. Reulen correctly noticed an early peak of SRT in data obtained in the overlap condition, and he concluded that the trigger for the facilitation does not necessarily come from the offset of the fixation point, but rather can be activated by internal events. Yet in this case the facilitation model would produce only two modes (not three), and in the gap task it would produce only one mode (not two or three). By contrast, the three-loop model accounts for all three peaks and in addition for anticipations and late responses occurring in most real experiments. Furthermore, the facilitation model predicts that the reaction times reach an asymptotic steady-state value with increasingly long gap durations. Instead, the experimental data show that SRTs shorten the most for some intermediate gap durations and increase again for longer gaps. Due to the dynamics the three-loop model can produce the effect of varying the gap duration.

Another aspect of the impulse version of the three-loop model is the production of reasonably different SRT distributions by only slight changes of the input activity driving the ATT module. This may serve as a basis to explain the large interindividual variability observed in naive subjects (Fischer et al. 1993).

While the three-loop model produces the correct distributions of reaction times, it makes – in its present state – no predictions at all in the spatial domain. What is therefore needed is an extension of the present model with the COM module computing the motor commands from the retinal error, possibly under control of the ATT module.

Effects of adaptation and learning may be incorporated into the model by slight changes in the FP activity to the ATT module. Alternatively or in addition, changes can be introduced by changing the coupling constants within the module or by modulating them according to their 'successful' use (hebbian synapses). It remains to be seen by concrete applications to what extent actual data like the effects of daily practice on SRTs in monkeys (Fischer et al. 1984) and in human subjects (Fischer and Ramsperger 1986) can be reproduced by the model. The simulated data shown in this paper do not exhibit such clear bimodal distributions as those obtained by Fischer and his collaborators. However, they used mainly highly trained subjects, whereas the present simulations should account for data from naive subjects. If, for example, some of the coupling constants are modified by 'training', it may well be that clear bimodality is obtained for certain gap durations.

Inhibition – in this stage – is not needed in the model, because the activity in each module declines after some time by itself. It must be noted, however, that in the present state the activity in the different modules is reset at the end of each trial. In reality, of course, such a resetting (if it occurs at all) must be mediated by an extra process. It will be very important to find out where and when the resetting occurs and whether or not it is complete. The existence of intertrial effects suggests that every trial carries part of the history of the preceding trials, thus giving rise to context-dependent effects in SRT distributions (Jüttner and Wolf 1992 and Weber et al. 1993, manuscript submitted).

Finally, one should be able to simulate the results of experiments where valid or invalid visual endogenous or exogenous cues are used at various time intervals before target presentation. The visual signals of these cues will feed into the central modules, giving rise to a higher preactivation, which then leads to shorter latencies or to anticipations depending on the lead time of valid cues. These simulations are to be compared with the corresponding experimental results. Eventual discrepancies between the model and the real data will show at which states other higher brain functions – like memory for the cue position – will come into play or to what extent the simple processes used in the three-loop model are sufficient to describe the data.

Acknowledgements. This work was supported by the Deutsche Forschungsgemeinschaft (DFG), Sonderforschungsbereich 325, Tp C5. The help of Erika Hausenblas from Salzburg in evaluating the reliability of single neural modules is greatfully acknowledged.

References

Biscaldi M, Weber H, Fischer B, Stuhr V (1994) Mechanism of fixation in man: evidence from saccadic reaction times. In: Findlay J (ed) Eye Movement research: mechanisms, processes and applications. Elsevier, North-Holland, Amsterdam (in print)

Buhmann J, Schulten K (1986) Associative recognition and storage in a model network of physiological neurons. Biol Cybern 54:319–335

Dominey PF, Arbib MA (1992) A cortico-subcortical model for generation of spatially accurate sequential saccades. Cereb Cortex 2:153–175

Fischer B (1987) The preparation of visually guided saccades. Rev Physiol Biochem Pharmacol 106:1–35

Fischer B, Boch R (1983) Saccadic eye movements after extremely short reaction times in the monkey. Brain Res 260:21–26

Fischer B, Boch R (1991) Cerebral cortex. In: Carpenter R (ed) Vision and visual dysfunction Vol 8. Eye movements. Macmillan, London, pp 277–296

Fischer B, Ramsperger E (1984) Human express saccades: extremely short reaction times of goal directed eye movements. Exp Brain Res 57:191–195

Fischer B, Ramsperger E (1986) Human express saccades: effects of randomization and daily practice. Exp Brain Res 64:569–578

Fischer B, Weber H (1990) Saccadic reaction times of dyslexic and age-matched normal subjects. Perception 19:805–818

Fischer B, Weber H (1993) Express saccades and visual attention. Behav Brain Sci 16:553–567

Fischer B, Boch R, Ramsperger E (1984) Express-saccades of the monkey: effect of daily training on probability of occurrence and reaction time. Exp Brain Res 55:232–242

Fischer B, Weber H, Biscaldi M, Aiple F, Otto P, Stuhr V (1993) Separate populations of visually guided saccades in humans: reaction times and amplitudes. Exp Brain Res 92:528–541

Jüttner M, Wolf W (1992) Occurrence of human express saccades depends on stimulus uncertainty and stimulus sequence. Exp Brain Res 89:678–681

Kingstone A, Klein RM (1993) What are human express saccades? Percep Psychophys 54:260–273

Langer TP, Kaneko CR (1990) Brainstem afferents to the oculomotor omnipause neurons in monkey. J Comp Neurol 295:413–427

Mayfrank L, Mobashery M, Kimmig H, Fischer B (1986) The role of fixation and visual attention in the occurrence of express saccades in man. Eur Arch Psychiatry Neurol Sci 235:269–275

Mountcastle VB, Motter BC, Steinmetz MA, Sestokas AK (1987) Common and differential effects of attentive fixation on the excitability of parietal and prestriate (V4) cortical visual neurons in the macaque monkey. J Neurosci 7:2239–2255

Munoz DP, Wurtz RH (1992) Role of the rostral superior colliculus in active visual fixation and execution of express saccades. J Neurophysiol 67:1000–1002

Reulen JP (1984) Latency of visually evoked saccadic eye movements. I. Saccadic latency and the facilitation model. Biol Cybern 50:251–262

Robinson DA (1981) Control of eye movements. In: Brookhart JM, Mountcastle VB, Brooks VB, Geiger SR (eds) Handbook of physiology – the nervous system II. American Physiological Society, Bethesda, pp 1277–1320

Rohrer WH, Sparks DL (1993) Express saccades: the effect of spatial and temporal uncertainty. Vision Res 33:2447–2460

Saslow MG (1967) Effects of components of displacement-step stimuli upon latency for saccadic eye movement. J Opt Soc Am 57:1024–1029

Schiller PH, True SD, Conway JL (1980) Deficits in eye movements following frontal eye-field and superior colliculus ablations. J Neurophysiol 44:1175–1189

Wenban-Smith MG, Findlay JM (1991) Express saccades: is there a separate population in humans? Exp Brain Res 87: 218–222