COMPUTER SIMULATION OF OCULOMOTOR CONTOL IN EXTRAPYRAMIDAL DISORDERS

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ABSTRACT

Computer simulation with a model of the saccadic burst generator, proposed by Scudder (1988), demonstrated that metrics of saccades as well as their velocity characteristics can be altered without changing the characteristics of the burst generator itself. The model could be used to explain impairment of saccadic eye movements in for instance Parkinson's disease (PD), since in this disorder at brainstem level pontine structures, where the burst generator is located, are unaffected. Comparison of saccades in PD patients with computer simulations showed that the model provided a reasonable explanation for the observed reduction in maximum saccade velocity, but is not appropriate to explain the hypometria of saccades.

INTRODUCTION

The Robinson model (1981) is a commonly accepted and widely used neural model for the generation of saccadic eye movements. However, there is a growing experimental evidence for some shortcomings. Firstly, the input to the burst generator, called the motor error, is represented in head coordinates, although such neural commands never have been demonstrated and always seem to be presented in retinal coordinates. Secondly, long lead burst neurons (LLBN), found in the para pontine reticular formation (PPRF) do not play a role in this model. Thirdly, an internal local feedback loop has been demonstrated, which does not carry signals of the neural integrator. In the Robinson model motor error is computed as the difference between desired eye position and actual eye position. The latter position is derived from the neural integrator as an efferent copy.

With respect to these shortcomings Scudder (1988) recently has modified the Robinson model by the introduction of LLBN's. In his model LLBN's receive a topographically weighted excitatory projection from the superior colliculus (SC), the 'where' signal, and an ipsilateral inhibitatory projection from inhibitatory burst neurons (IBN). The LLBN's will integrate these two 'velocity' commands and subsequently will summate them. The result serves as an input to the excitatory burst neurons (EBN) and replaces the motor error signal of the Robinson model. Another difference in the Scudder model is that omni-pauze neurons (OPN) are not excited by a 'when' signal but receive an indirect weak excitatory 'trigger' from the SC. In this manner 'when' and 'where' signals for saccadic generation are not strictly separated anymore, as is the case in the Robinson model; the 'when' signal is now derived from the 'where' signal. Consequently, the Scudder model may generate saccades which velocity characteristics and metrics depend on the distribution of the input signal, which definitely is not possible with the Robinson model.

Scudder suggested that his model may provide an explanation for changes seen saccade metrics and saccade velocity, as may occur in for instance Parkinson's disease (Carl and Wurtz, 1985). To investigate this hypothesis, we measured in patients with extrapyramidal disorders the characteristics of their saccades in the horizontal as well in the vertical direction and compared the results with our own computer simulations of the Scudder model.

METHODS

Saccadic eye movements have been measured in a control group consisting of 14 healthy individuals, and a patient group consisting of 7 subjects with Parkinson's disease and 5 subjects with Parkinsonian symptoms. Eye position was recorded by means of the double magnetic induction method (Bour, 1984). The relation between saccade amplitude and maximum saccade velocity for visually elicited saccades has been determined in four different directions being rightward, leftward, downward and upward. Mean saccadic gain was measured for anticipatory saccades during a 0.55 Hz square-wave tracking paradigm with an amplitude of 20 degrees.

The Scudder model was implemented on a Commodore Amiga computer. The weightings of all projections, the conduction delays as well as the bias of each neuron could interactively be adjusted. Also the waveshape of the Gaussian collicular discharge could be changed by adjustment of the peak-value and the standard deviation. Eye position has been computed by a convolution of the plant characteristics with the calculated innervation of the oculomotoneurons. Eye velocity was derived from eye position. After simulation the firing rates of

each individual neuron, the resulting eye movement and its velocity could immediately and graphically be displayed.

RESULTS

It was found in the group of seven PD patients that for anticipatory saccades six had abnormal reduction of vertical gain and five had abnormal reduction of horizontal gain. In the group of five patients with Parkinsonian symptoms these values were three and four respectively. So hypometria was found most frequently in the vertical direction. In none of the patients with extrapyramidal disorders (PD patients and patients with Parkinsonian symptoms together) a reduction of maximum saccade velocity in the horizontal direction was found. However, in comparison to the horizontal direction or the other vertical direction in seven out of the twelve patients of the total group an abnormal reduction (more than 30%) of maximum saccade velocity was found either in upward or in downward direction or both. This reduction was most pronounced for saccades larger than 10 degrees. There was no clear correlation between the reduction of saccadic gain and the reduction of maximum saccade velocity.

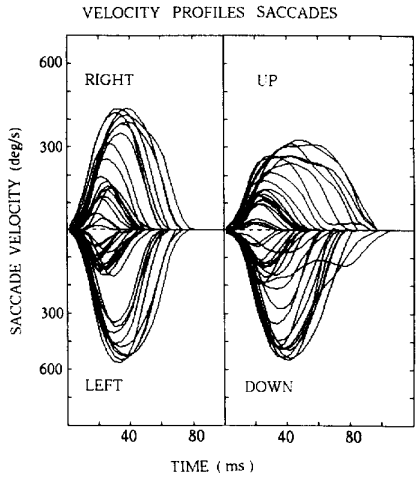

Figure 1

Figure 1 shows for a PD patient the velocity profiles of visually elicited saccades with different sizes in all four directions. The reduction of the maximum saccade velocity for larger saccades in the upward direction (about 150 degrees/second) clearly can be observed.

Model simulations were performed while the standard deviation of the Gaussian waveshape was increased simultaneously with a decrease of the peak-value such that the integral of the distribution remained the same. This resulted in two saccades with about the same size, however their velocities were different. Figure 2 shows how the maximum saccade velocity decreases for saccades larger than about 8 degrees. The time plotted at the end of each curve divided by the square root of 2 refers to the standard deviation of the Gaussian distributed collicular discharge. In order to obtain multistep saccades this time value had to be larger than 60 ms. Initially, a broadening of the waveshape decreases the maximum saccade velocity of the larger saccades. Finally, a further broadening will lead to hypometria especially for the larger saccades.

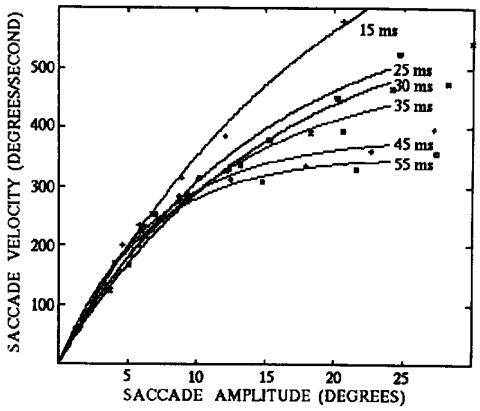

Figure 2

DISCUSSION

One could speculate that in the patient group investigated the collicular discharge would be broadened by a disturbed desinhibition of the SC by the substantia nigra. According to the Scudder model, initially this would lead to a reduced maximum saccade velocity and with a further broadening to hypometria. In all patients except one hypometric saccades were found. However, seven out of twelve patients had reduced maximum velocity of saccades, but only in the vertical direction. So we must conclude that the Scudder model provides a reasonable explanation only for the reduced maximum saccade velocity in either upward or downward or both directions with respect to the horizontal direction, but seems to be not appropriate to explain the hypometria of saccades. It is important to note that the maximum saccade velocity is only reduced with respect to the other horizontal direction but in itself is not abnormal. This is in accordance with the Scudder model.

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