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Automatic and Strategic Volitional Saccadic Eye Movements in Psychotic Patients

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Summary. Drug-free schizophrenics were compared with depressive psychotics and normal controls on two saccade initiation tasks which differed with respect to the type of stimulus that initiated a saccadic response. Strategic initiation (SIS) appears to use a route different from that in automatic initiation (AIS). The SIS task revealed slowed responding in psychiatrically ill patients if their cognition was impaired, but all groups responded similarly on the AIS task. Schizophrenics could be separated from depressed psychotics by their inability to utilize temporal redundancy to speed up saccade initiation on the SIS task. Neurophysiological evidence implicates specific impairments in the frontal eye field (FEF) and/or basal ganglia.

Key words: Schizophrenia – Saccades – Automatic and strategic processing

Introduction

It is commonly believed that there is little wrong with the saccadic eye movements of schizophrenic patients (Levin et al. 1981). In other words the initiation time, duration, peak velocity and overall quality of saccades are within normal range, although multistep saccades and overshoot have been reported to occur more frequently in schizophrenics (Mather and Putchat 1983). This contrasts markedly with research into smooth pursuit eye movements from which there is considerable evidence that this kind of eye movement is quite severely altered in schizophrenics. However, Done and Frith (1984) reported two different methods of eliciting saccadic eye movements which they classified as (i) automatic initiation of saccades (AIS) and (ii) strategic initiation of saccades (SIS). This classification is based on Schneider and Shiffrin (1977), who used the terms "automatic" and "controlled". Different terminology describing the same distinction has been used, e.g. action controlled by an external stimulus or an internal intention (Frith and Done 1986), "event triggered" and "information-guided" (Breitmeyer 1986).

Passingham (1987) and Goldberg (1985) have presented evidence for two brain systems, one involving the supplementary motor area which initiates internally driven responses, the other involving the arcuate pre-motor area which mediates stimulus-driven responses.

This dichotomy of action will probably turn out to be a continuum with highly automated responses at one extreme for which little information processing is involved in stimulus perception and response allocation and at the other extreme much information processing. It is probably sensible to consider that all of these actions are volitional in the sense that one is not obliged to make the response in the presence of a stimulus as in a reflexive response, since cortical control is possible. In the case of automatic responses this cortical involvement is simply inhibitory, i.e. preventing the most compatible response, if that response is undesirable. But if the most compatible response is desirable then inhibition is released.

In the case of strategic responses the stimulus to be responded to has no redundancy; it needs to be selected from the background and assessed for meaning, which demands considerable cortical analysis. After, or perhaps during, stimulus processing, response selection must be pursued since there is no S-R redundancy (in behaviourist terminology the prob-

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ability of the response is low). Compare making a saccadic eye movement to a spot of light in the peripheral visual field. It would appear that a refixation, so as to foveate this novel stimulus demands little cortical involvement, since in Huntington's disease these saccades are initiated when subjects are required *not* to make them (Leigh et al. 1983). Leigh et al.'s explanation for this is that in Huntington's disease cells of the pars reticulata of the substantia nigra cannot inhibit the superior colliculus. Thus the novel stimulus elicits a saccade towards the stimulus, which if the cortex or basal ganglia were able to suppress, they would do so if the experimental instructions so required. Contrast this with a different hypothetical experimental paradigm in which subjects are shown words in the centre of the screen and whenever they see the word "red" they make a leftwards horizontal saccade and refixate on a point some 10° left of centre, but when they see the word "dog" a rightwards saccade is demanded. Clearly here there is no stimulus or response redundancy and the probability of the response, given the stimulus, is very low.

In summary, then, saccadic responses can be separated into:

1. Reflexive saccades, e.g. the fast phase of vestibulo-occular nystagmus and optokinetic nystagmus for which direct pathways from the sensory systems to the pontine reticular formation exist (Raphan and Cohen 1978) and on which decortication in monkeys, cats and rabbits has no effect (Ter Braak 1936).

2. Automatic saccades (Posner 1978; Done and Frith 1984) which are "event triggered" via a route from either visual cortex or retina to the superficial layers of the superior colliculus (Breitmeyer 1986). The selective enhancement of the collicular cells can be inhibited by the frontal eye fields (FEF) – basal ganglia – superior colliculus route, thereby allowing limited volitonal control over the acceptability of a highly compatible response when the event, or stimulus, occurs.

3. Strategic saccades, which can be elicited in the absence of any stimulus or when the S-R compatibility is so low that one could just as well make a hand movement or vocal response. Without FEF or FEF-basal ganglia input this kind of saccade cannot be elicited, as is the case in Huntington's chorea (Leigh et al. 1983).

The literature on volitional saccadic eye movements in schizophrenia demonstrates a consistent use of methods that elicit automatic saccades. Thus Iacono et al. (1981), Levin et al. (1981) and Mather and Putchat (1983) used a spot stimulus that moved abruptly to the left or right of fixation. However, Done and Frith (1984) demonstrated that although schizophrenics produced saccades with normal reaction time with this paradigm, a more strategic saccadic task induced slower reaction times in schizophrenics.

In the experiment to be described here we have used the same two experimental paradigms reported in Done and Frith (1984).

Subjects and Methods

Seventeen cases with a Present State Examination (PSE) classification (Wing et al. 1974) of S+ (a core group of schizophrenics) were selected because of their lack of recent exposure to medication. Eleven cases appeared to have never had neuroleptic treatment before, and of these 11 none had received benzodiazepines within 3 days prior to testing; 1 case had in the past received lithium treatment but had not taken lithium medication during the previous month; and one case had received procyclidine medication. Of the other 5 cases, 2 had not received neuroleptics for at least 1 month prior to testing, 1 was a first admission whose drug history was uncertain but who was thought to have not received any medication recently, 1 had received 40 mg Depixol (flupenthiseol decanoate, Lundbeck Ltd., UK) injections 1 week prior to testing but no other medication, and the fifth case was not on any regular medication but had received 100 mg chlorpromazine on each of the 2 preceding days for agitation. Data for 1 case failed to be recorded for the SIS task and hence there were only 16 cases performing this task.

Two control groups were also used. One group ($n = 10$ for SIS and $n = 8$ for AIS)[1] was made up of nurses, CRC employees and occupational therapists; all reportedly were mentally healthy and unmedicated and of comparable age to the schizophrenics. The other group comprised 5 drug-free PSE D+ patients (D+ is a group termed "depressive psychosis", in whom the major psychopathology is depression accompanied by psychotic symptoms, usually delusions, which are not first-rank delusions) and 2 non-psychotic, drug-free, retarded depressives. This made a total of 7 in the depressive control group, although data for only 6 were available for the AIS task.

The selection of drug-free patients was made possible by an ongoing medication trial within the Division which has been described elsewhere (Johnstone et al. in press).

Procedure. The two tasks both demanded of the subjects a saccadic eye movement 10° to the left, or right, of fixation. One version was considered as demanding strategic initiation of saccades (SIS taks). The other version produced automatic initiation of saccades (AIS taks). In both versions a subject sat with his eyes approximately 40 cm away from a PET (CBM) VDU. On a given trial the subjects observed a rectangular box in the centre of the screen. After a time interval (approximately 2.0 s) a spot appeared in the centre of the box either alone (50% trials) or together with a warning signal (50% trials) which consisted of a letter "L" or "R" immediately above the spot. At the same time two +'s appeared 10° to left and right

[1] The difference in numbers of subjects is due to low frequency counts (i.e. < 3/6 records) in one of the eight conditions arising from anticipatory or misdirected or uninitiated saccades, non-central fixation or other subject errors.

of the fixation spot. Subjects were instructed that a letter above the spot acted as a warning signal so they could prepare themselves prior to receiving the imperative stimulus. "L" indicated a leftward movement, "R" a rightward movement. The time interval (SOA) between the appearance of the spot (the "Ready" signal) and the imperative stimulus was either 0.25 s (50% trials) or 2.0 s (50% trials). These conditions prevailed in both versions. However, with respect to the SIS task the imperative signal was a presentation of the letter "L" or "R" *beneath* the spot after a 0.25 or 2.0 s SOA. Approximately 0.5 s after the imperative signal one of the crosses at the periphery of the screen changed to the letter "O" and the other cross changed to the digit "Ø". The subjects were instructed that as soon as they saw the imperative signal they had to move eyes as quickly as possible in the appropriate direction and report whether the letter O had a line passing through it, or not. The subject was also instructed to observe the fixation spot when it appeared.

In the AIS task the imperative signal consisted of the fixation spot moving rapidly from the centre of the screen to the peripheral point marked with the "+". On arrival the "+" transformed into either the letter "O" or the digit "Ø". Subjects were required again to report whether the character had a line through it or not. In both versions there were 48 trials. This allowed for six replications of each combination of the three conditions (direction of movement, duration of SOA, presence/absence of warning). All eight combinations ($2 \times 2 \times 2$) were presented in a random order before repetition.

Eye movements were recorded with Beckman skin electrodes attached to the outer canthus of each eye with a reference electrode on the back of the right hand. Eye movements were amplified and recorded on an Akai 4000 DS MIL-II tape recorder. The tape recorder was subsequently analysed using ELSA (a package designed by C.D. Frith for the analysis of psychophysiological data) on a PDP 11/10. Eye movements were recorded over a 1.08 s epoch commencing at the onset of the imperative signal. Sampling occurred every 5 ms. A visual analogue of the eye movement was displayed on VDU along with an analogue of the stimulus channel. This allowed for visual examination of eye movements pre- and post-stimulus onset such that non-central fixations, anticipations, blinks and other erroneous eye movements could be eliminated before the marking routine analysed the record. An automatic marking routine was used to standardize the markings for onset and termination of saccades, thereby avoiding subjective biases. Output from the routine provided the RT as well as amplitude and duration of the saccade.

Results

Strategic Saccades

Log transformations were performed on the data to eliminate the positive skew of reaction time data, before calculating repeated measures analysis of variance (ANOVA) using BMDP2V. Two separate analyses were performed in comparing schizophrenics with each to the two control groups. This was done instead of a single ANOVA, since data presentation is clearer. In the ANOVA for schizophrenics compared with normals there were the following significant main effects and interactions: (i) group difference – $F(1,24) = 4.34$, $P < 0.05$; (ii) effect of warning cue – $F(1,24) = 89.0$, $P < 0.0001$; (iii) effect of SOA – $F(1,24) = 28.2$, $P < 0.0001$; (iv) a group × SOA interaction – $F(1,24) = 7.36$, $P < 0.01$; (v) a warning × SOA interaction – $F(1,24) = 8.77$, $p < 0.01$. Geometric means are used in the presentation of data in Fig. 1a.

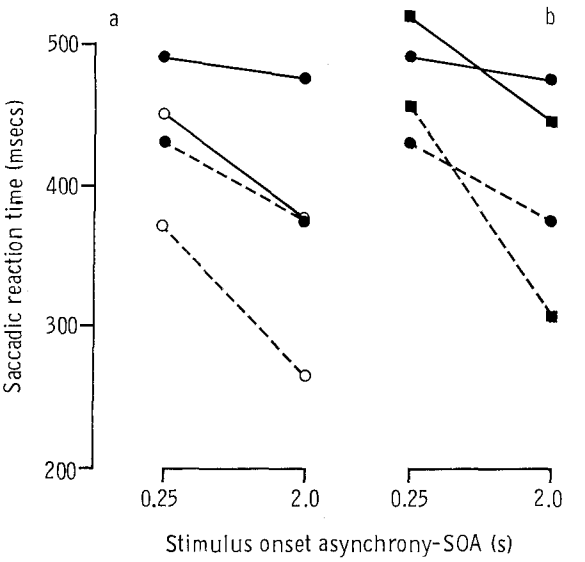

Fig. 1a, b. Effect of warning cue and SOA on strategic saccadic reaction times. (----) Warning cue given; (——) No warning cue given; (●) Schiz ($N = 16$); (■) Depr ($N = 7$); (○) Norm ($N = 10$)

With regard to the main effects it is clear that the schizophrenic group was slower overall than the controls. The other main effects are to be expected. With regard to the interactions it is interesting to note that the schizophrenic group was less facilitated by the 2 s SOA, although the highly facilitating effect of the warning cue was similar in both groups.

In the ANOVA of the schizophrenics compared with depressed control group the following notable results could be observed: (i) no significant group difference – $F(1,21) < 1$; (ii) a large warning cue effect – $F(1,21) = 56.6$, $P = 0.000$; (iii) a large SOA effect – $F(1,21) = 17.0$, $P = 0.000$; (iv) a group × SOA interaction – $F(1,21) = 4.5$, $P < 0.05$; (v) a warning cue × SOA effect – $F(1,21) = 8.0$, $P < 0.01$.

Geometric means are used in the presentation of data for the schizophrenic and depressed subjects in Fig. 1b.

Clearly the tardiness of saccadic initiation by schizophrenic patients is not diagnosis specific but related to some general property of psychosis which will be explored later.

Automatic Saccades

Data for this version of the task were transformed and dealt with in a fashion similar to the strategic (SIS) version.

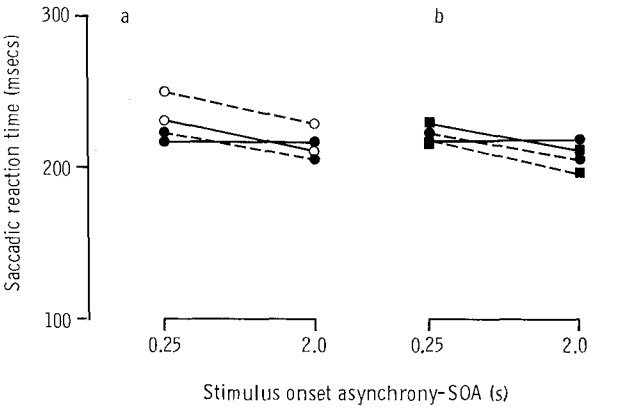

Fig. 2a, b. Effect of warning cue and SOA on automatic saccadic reaction times. (----) Warning cue given; (——) No warning cue given; (●) Schiz (N = 17); (■) Depr (N = 6); (○) Norm (N = 8)

In the ANOVA comparing schizophrenics with normal controls the following results were noted: (i) no overall group difference, F (1,22) <1; (ii) no effect of warning cue F (1,22) = 1.53, $P=0.23$; (iii) a significant effect of SOA, F (1,22) = 14.35, $P<0.001$; (iv) no significant interactions.

Figure 2 presents the geometric means for these two groups on this task.

In the ANOVA for the contrast of schizophrenics and depressed patients there were only two significant main effects, namely warning cue, F (1,20) = 6.3, $P<0.002$ and SOA F (1,20) = 8.2, $P<0.01$, but no significant interactions. Interestingly, in a single ANOVA utilizing the data for all three groups a significant warning cue × group interaction was observed, F (2,27) = 4.45, $P<0.02$. This interaction is due to a slight enhancement of reaction time in the depressed patients after a warning cue, but a slowing down after the warning cue in the normal controls.

Summary of Results

The contrast in the results from these two saccade initiation tasks confirms the supposition that one is strategic and the other is automatic for the following reasons.

1. In the AIS task conscious preparation via a warning cue could not be used to enhance reaction time in the normal control subjects; indeed, there was a tendency for the warning cue to slow down reaction time.

2. The standard deviation of scores within normal subjects was small in the AIS taks. The mean of the standard deviation for each subject taking conditions separately was 28.8. This compares with a value of 83.8 for the SIS task. This suggests a limited opportunity for varying reaction speed in the AIS task, which is probably due to the use of a relatively fixed route from stimulus analysis through to response production.

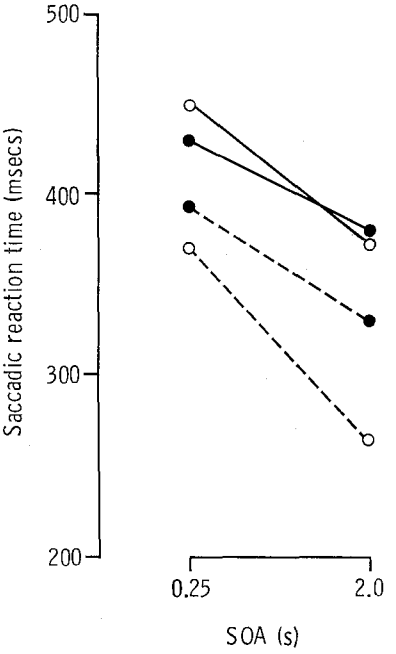

Fig. 3. Effect of warning cue and SOA in the SIS task for a select group ($N=6$) of schizophrenics (see text for basis of selection). (----) Warning cue given; (——) No warning cue given; (●) Schiz (N = 17); (○) Norm (N = 8)

In respect of group differences the schizophrenics have a slow reaction time on the SIS task compared with the normal controls (F (1,24) = 4.34, $P<0.05$ but are comparable on the AIS taks (F 1,22 <1). However this finding is not peculiar to the schizophrenics. When they are compared with a depressed control group their reaction times are comparable on both SIS and AIS tasks. It was also noticeable to us that the normal control – schizophrenics SIS difference was much smaller than we had previously found (Done and Frith 1984). This was initially somewhat worrying until we realized that after the first study we became more interested in testing drug-free patients not only on saccadic initiation tasks but also on a variety of finger reaction time tasks, since we wanted to compare performance of saccade initiation and finger-lifting responses. This requirement will have biased our subject selection in favour of more able subjects. Indeeed, when those schizophrenic patients who completed the saccade initiation tasks as well as the finger reaction time tests are extracted and analysed separately, the difference between schizophrenic and normal controls on the SIS task disappears, F (1,14) <1, although the main effects of warning cue [F (1,14)

= 52.51, $P = 0.000$], SOA [F (1,14) = 31,02, $P = 0.000$] remain and the group × SOA interaction also remains [F (1,14) = 2.7, $P = 0.12$]. The geometric means are ploted in Fig. 3.

These results suggest that performance by schizophrenics on the SIS task varies. Indeed, our suspicion that saccadic initiation time in psychiatrically ill patients is not specific is bolstered by a correlation of −0.39 (df 11, $P < 0.10$) between mean SIS reaction time and the score on the WAIS digit symbol substitution test on those patients, both schizophrenic and depressed, for whom both sets of data were recorded while the patients were still drug free. Thus SIS reaction time varies according to the degree of generalized slowing of cognition. What is more interesting is the robustness of the group × SOA interaction. It appears that schizophrenics in particular are less able to enhance saccadic initiation times when the SOA is increased from 0.25 to 2.0 s. This interaction was found in the earlier study (Done and Frith 1984), F (1,22) = 6.94, $P < 0.01$, in the current analysis including all schizophrenics versus normal controls F (1,24) = 7.36, $P < 0.01$ as well as all schizophrenics versus depressive controls, F (1,21) = 4.5, $P < 0.05$ and a trend even in a very select group of schizophrenics ($n = 6$) F 1,14 = 2.7, $P = 0.12$ for whom overall reaction time was comparable with normal controls. It cannot therefore be explained away as an artefact of test difficulty for two reasons. Firstly, the test is as difficult for the depressed patients who show a normal effect of SOA. Secondly, the SOA × group effect persists even in the small group of schizophrenics whose overall SIS reaction time was quite normal. It also cannot be explained away as an artefact of lack of motivation or distraction (e.g. as a result of hearing voices), since maintenance of focused attention when given a warning cue is quite normal even after 2 s.

Discussion

The results presented here demonstrate that one cannot assume that reaction time for the initiation of saccades is normal in schizophrenic patients without specifying the type of saccade that the subject is required to make. We suggest that there is a gradation, which may or may not be a continuum, from reflexive saccades through automatic saccades to strategic saccades. Schizophrenics and depressed subjects, even when they have motor or cognitive retardation, can elicit reflexive and automatic saccades with normal reaction times, but both groups have slow reaction times when required to perform strategic saccades. The reason for this at first sight need not be attributed to anything other than a slowing of information processing during stimulus analysis and response selection. However, this interpretation cannot explain the slow reaction time in both groups even after a 2 s SOA together with a warning cue. In this condition stimulus analysis and response selection should be unnecessary (see Frith and Done 1986) and thereby allow any change in the fixation field to elicit an appropriate saccade. One might conjecture that this condition is akin to an automatic saccade. However, the reaction time in this condition for normals ($\bar{x}$ = 266 ms) is still noticeably longer than the reaction time for even unwarned stimuli for normal subjects in the AIS task ($\bar{x}$ = 211 ms). Thus two different routes to action must be utilized by the two different methods.

Bruce and Goldberg (1985) suggested that there are perhaps three pathways through which the FEF can influence saccade elicitation. One route is a direct projection to the intermediate layers of the superior colliculus; the second route is indirect from FEF via the caudate to substantia nigra, which then projects to the intermediate layers of the superior colliculus; and the third route avoids the superior colliculus by projecting from FEF to the medial dorsal nucleus of the thalamus, the mesencephalic reticular formation, and the pontine reticular formation. However, these pathways do not include inputs from the visual or parietal cortices. Breitmeyer (1986) describes an "early warning" system that alerts the visual system to objects suddenly arriving in or moving into the visual field. Detection of such events then triggers an appropriate saccade. This system depends upon fast-conducting transient fibres arriving at the superior colliculus from the visual cortex or directly from the retina. Since cells in the superior colliculus show selective enhancement for behaviourally significant stimuli prior to goal-directed saccades, this route could easily be involved in the AIS task. Since the superior colliculus is limited to coarse localization and detection of visual stimuli rather than identification of stimuli (Robinson and Goldberg 1978), it would not be susceptible to enhancement from the warning cue, although it could be enhanced, perhaps by reticular activation, during the SOA interval.

For the SIS task we would speculate that visual, parietal and frontal cortex will be involved and perhaps the frontal-basal ganglia route (Bruce and Goldberg 1985). Thus visual cortex is involved in the identification of the warning cue and imperative stimuli, and the parietal cortex will be involved in the shifting of visual attention in response to the warning cue. It is at present difficult to assess which regions are involved in saccade initiation in this taks. Cells responding only to stimuli if they are behaviourally significant have been identified in experimental para-

digms which present extrafoveal rather than central stimuli. Such specific enhancement can be found in cells of the parietal cortex (Breitmeyer 1986), FEF (Bruce and Goldberg 1985), superior colliculus, caudate (Rolls et al. 1983) and substantia nigra pars reticulata (Evarts et al. 1984). However, Bruce and Goldberg found that 20% of FEF cells would respond before purposive saccades made in the absence of a visual stimulus. They also found FEF cells firing prior to a "go" signal (the offset of the fixation light). This latter paradigm was also used by Hikosaka and Wurtz (1983), who found cells in the substantia nigra pars reticulata which lowered their firing rate prior to the "go" signal. This condition is perhaps similar to the warning cue condition of the SIS task which schizophrenics performed quite normally. We are therefore left with the speculation that the enhancing of cells in this system, perhaps by reticular activity, but certainly as a function of time interval between S_1 and S_2, is deficient in drug-free schizophrenics.

It is interesting to speculate that a similar deficit is responsible for the "crossover effect" (cf. Neuchterlein 1977). The basic paradigm, although there are a number of variants here, is that subjects must respond manually to stimuli presented at either regular or irregular intervals. Schizophrenics unlike normals fail to improve their RTs in the predictable regular series when the time interval exceeds 2 s (Huston et al. 1937), although this precise value varies between studies. So the facilitation of a 2 s over a 0.25 s SOA in normals, in a predictable series, would not occur in schizophrenics and so an interaction should be observed. One could criticize this argument on the grounds that the series used here is unpredictable, since it is a mixture of 2 s and 0.25 s intervals. However, once 0.25 s of SOA has elapsed without presentation of the imperative stimulus, the time delay is always 1.75 s, and as such must be highly predictable and should facilitate reaction time (Frith and Done 1986, experiment 1). Thus there are similarities between the observed group × SOA interaction reported here and the crossover effect. We believe that the effect in saccade initiation is more robust and more available to neurophysiological modelling.

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