# Antisaccades and Task Switching

## Studies of Control Processes in Saccadic Function in Normal Subjects and Schizophrenic Patients

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**ABSTRACT: Executive functions allow us to respond flexibly rather than stereotypically to the environment. We examined two such functions, task switching and inhibition in the antisaccade paradigm, in two studies. One study involved 18 normal subjects; the other, 21 schizophrenic patients and 16 age-matched controls. Subjects performed blocks of randomly mixed prosaccades and antisaccades. Repeated trials were preceded by the same type of trial (i.e., an antisaccade following an antisaccade), and switched trials were preceded by a trial of the opposite type. We measured accuracy rate and latency as indices of processing costs. Whereas schizophrenic patients had a threefold increase in error rate for antisaccades compared to normals, the effect of task switching on their accuracy did not differ from that in normal subjects. Moreover, the accuracy rate of trials combining antisaccade and task switching was equivalent to a multiplication of the accuracy rates from trials in which each was done alone. Schizophrenic latencies were disproportionately increased for antisaccades, but again they were no different from normal subjects in the effect of task switching. In both groups the effect of task switching on antisaccades was a paradoxical latency reduction. We conclude that the executive dysfunction in schizophrenia is not generalized but selective, sparing task switching from exogenous cues, in which the switch is limited to a stimulus-response remapping. The accuracy data in both groups support independence of antisaccade and task-switching functions. The paradoxical task-switching benefit in antisaccadic latency effects challenges current models of task switching. It suggests either carryover inhibition by antisaccadic performance in the prior trial or facilitation of antisaccades by simultaneous performance of other cognitive operations.**

**KEYWORDS: antisaccades; task switching; executive; attention; latency; schizophrenia**

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## INTRODUCTION

Antisaccades[1] are an example of controlled processing in which a habitual act (looking towards a suddenly appearing target with a prosaccade) must be overruled by a highly novel response (looking away from the target). The antisaccade/prosaccade relation is an example of a response pair with a dominance asymmetry. Dominance arises when one response gains an advantage over the other through prior experience, intraexperimental practice, or stimulus-response compatibility.[2] Thus, in the Stroop test,[3] reading the name of a color is dominant over stating the color of the ink when the two conflict. Dominance asymmetries vary along a spectrum.[4] The antisaccade/prosaccade pairing lies on the extreme of this continuum.

Dominance asymmetries have gained interest in studies of task switching. Task switching, another example of controlled processing, usually incurs added costs in prolonged latencies and increased error compared to task repetition (e.g., Refs. 5–7). It is not clear what generates these costs. Some studies report that the increase in latency induced by switching from a dominant to a nondominant task is less than the increase generated by switching in the reverse direction.[5] This has been interpreted as evidence of either negative stimulus-response priming[8] or carryover of inhibition of the current response from the prior trial, when it was an inappropriate response.[5] Thus, a nondominant response requires strong inhibition of the dominant response, which must be overcome to switch back to a dominant response in the next trial. In the reverse direction, a dominant trial does not need much inhibition of the nondominant alternative, and hence little inhibition carries over in the switch to a nondominant trial. The resulting asymmetry of switch costs with dominance asymmetry is hard to account for with other explanations of task-switching costs.[7]

Others note, however, that not all response pairings with dominance asymmetries engender asymmetric switch costs.[2] Monsell *et al.*[2] hypothesized that asymmetric switch costs may only occur with pairings that are highly asymmetric in dominance. Other factors may play a role also. Task switching can be a complex of many changing cognitive processes. Switches might require a shift in the stimulus dimension attended (word versus ink color in the Stroop test), the stimulus location attended, the classification of the stimulus needed, the response mode to use (verbal versus manual), and the stimulus-response mappings made, among others. The contributions of each of these factors to switch costs are relatively unknown.

The antisaccade/prosaccade pairing has some advantageous features for exploring these issues in task switching. First, the dominance asymmetry is extreme: most naive subjects have never performed an antisaccade, even though they perform prosaccades many times a minute while awake, every day of their lives. Second, the switch between prosaccades and antisaccades minimizes the number of changing task features. The stimulus for both prosaccades and antisaccades is a small peripheral light, with the same locations, the same relevant attribute (spatial location), and the same classification (right or left). Both tasks require the same response mode (saccade) with only two possible values (right or left). The key difference remaining is the stimulus-response mapping, which is reversed for antisaccades. Hence, if asymmetries between switching to antisaccades and switching to prosaccades are found, this would be strong evidence that the carryover of inhibitory influences from the prior trial are generated at the level of stimulus-response mapping.

Another issue of note in the interaction of task dominance and task switching is that of independence. Models of task switching tend to treat the settings from current and prior trials as independent effects.[6] If these processes are indeed independent, the accuracy cost of a response that requires both functions (the switched antisaccade) should equal the product of the accuracy rates of each function in isolation (a switched prosaccade and a repeated antisaccade).[9] The possibility that these are not independent is raised by the fact that damage to similar prefrontal areas impairs both task switching[10] and antisaccade performance.[11]

We explored these issues in both a normal population and a patient group to determine if pathologic effects on antisaccade and task-switching costs were correlated or independent. We chose to study patients with schizophrenia. These patients consistently show deficits on nondominant tasks such as the Stroop test and the antisaccade task[12] and have dysfunctional task switching based on instruments such as the Wisconsin card sorting test.[13,14]

## METHODS

### *Participants*

*Normal Study.* Eighteen subjects (6 male) participated, with ages ranging from 13 to 54 years (mean 30.8 years, SD = 9.5). None had previously performed an antisaccadic task.

*Schizophrenic Study.* This included 21 outpatients maintained on stable doses of antipsychotic drugs for at least 6 weeks. Diagnoses were confirmed with the Structured Clinical Interviews for DSM-IV.[15] Clinical status was characterized with the Positive and Negative Syndrome Scale (PANSS)[16] and the Brief Psychiatric Rating Scale (BPRS).[17] Sixteen normal subjects matched for age, sex, handedness, and parental socioeconomic status[18] served as the controls.

Participants also completed two manual tests of sustained attention on a computer, the Vigil Continuous Performance Test (The Psychological Corporation, Harcourt Brace & Company, 1998) and an abbreviated version of the California Computerized Assessment Package (CalCAP).[19] Twenty of the 21 schizophrenic patients also completed a computerized version of the Wisconsin Card Sorting test (WCST, CyberMetrics Testing Services) with these results classified by published age and education-matched normative data.[20]

### *Apparatus and Eye Movement Protocol*

We recorded eye movements with a magnetic search coil technique (Crist Instruments, Bethesda, MD). Displays were generated by a Power Macintosh 9600/233, using programs written in C++ on the Vision Shell programming platform (www.kagi.com/visionshell) and back-projected with an Eiki LC-7000U projector. Eye position was digitized at 500 samples/s and a five-point central difference algorithm[21] derived velocity from eye position.

The initial display had a dark background with a white, 1-degree fixation ring at the center (FIG. 1). The fixation ring was flanked by two 0.7-degree white dots at right and left 20 degrees. Trials started when the subject's eye was within 3 degrees

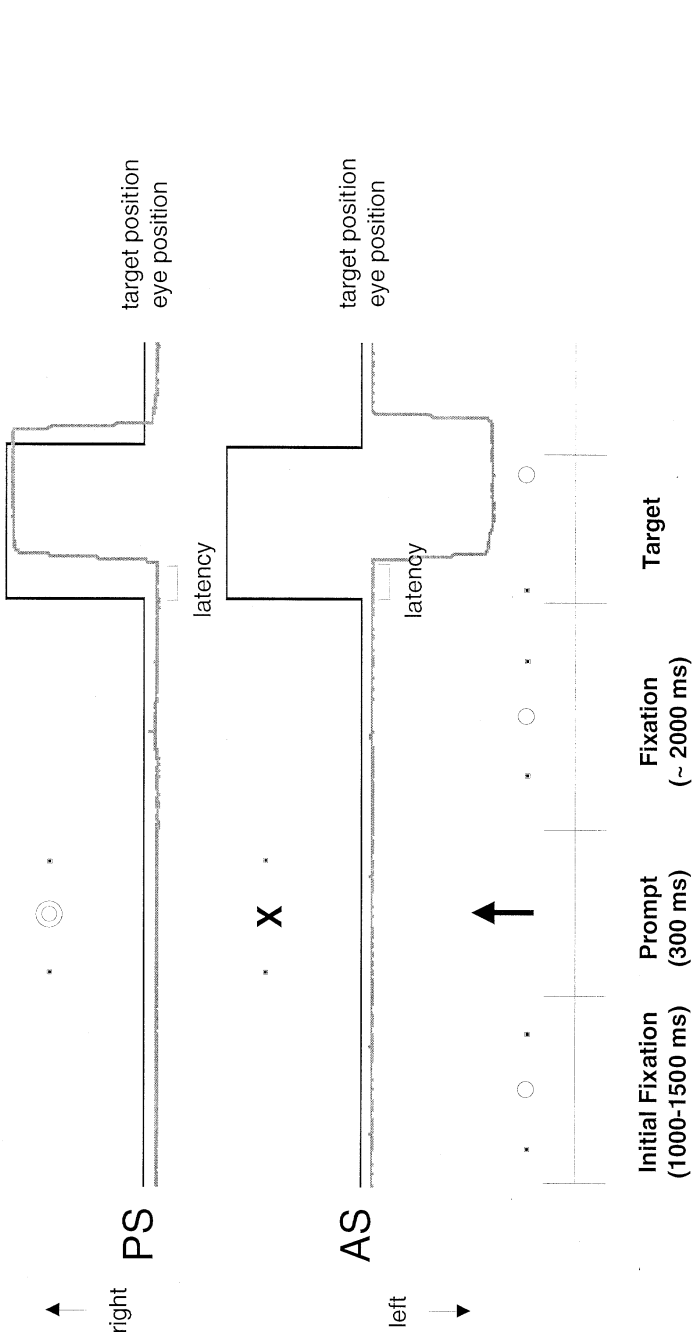


**FIGURE 1.** Trial illustration. Progress over time is from *left to right*. *Top lines* show horizontal position traces of targets (*black smooth lines*) and eyes (*grey irregular lines*) for a correct prosaccade (PS, *top*) and antisaccade (AS, *below*). Rightward motion is shown as up, by convention. *Bottom diagrams* show what the screen shows at each interval. The trial begins with a fixation period, with the eyes and target (ring) at zero position, or mid-screen. Two small dots mark the possible right and left locations of the target at all times. At the prompt, different screens are shown for prosaccades and antisaccades. The former are cued by a yellow double ring, the latter by a blue cross. The fixation screen then returns, followed by the appearance of the target, which triggers an eye movement response, either toward (prosaccade) or away from (antisaccade) the target. The trial is terminated when the eye enters a zone surrounding the desired eye location.

**TABLE 1. Definition of relative latency effects**

| | Mixed-task | | | Residual switch costs |
|---|---|---|---|---|
| | Repeat | Switch | | |
| Prosaccade (PS) | A | B | for PS: | B – A |
| Antisaccade (AS) | C | D | for AS: | D – C |
| Antisaccade (AS) costs: | C – A for repeat | D – B for switch | | |

of center. After 1–1.5 seconds, the fixation point was replaced by one of two prompts, a yellow "O" 4.5 degrees in diameter for prosaccade trials or a blue "X" spanning 4.5 degrees for antisaccade trials. Prompts were replaced after 300 ms by the white fixation ring. After a mean interval of 2 seconds the ring target shifted to one of the two peripheral dots.

Single-task blocks had 26 trials, either all prosaccades or all antisaccades. Mixed-task blocks had 52 trials, a random mix of prosaccades and antisaccades. Each block was repeated four times, generating 104 trials of each type. In the mixed-task blocks, about half required similar (repeated) and half required different (switched) responses from the previous trial. Blocks were given in a counterbalanced order to militate against the effects of learning and fatigue. In total there were 12 blocks between which short rests were provided. All subjects performed a practice session of 20 trials of each of the three different blocks.

### *Analyses*

Trials from mixed-task blocks could be either "repeated trials," preceded by a trial requesting the same response (e.g., antisaccade preceded by an antisaccade), or "switched trials," preceded by a trial requesting a different response (e.g., an antisaccade preceded by a prosaccade). Consequently, there were three *conditions* — blocked (from single-task blocks), repeated, and switched — for both saccadic *tasks*, prosaccades and antisaccades, yielding six different saccade groups.

The first trial of each block was eliminated from analysis. Accuracy was calculated for each subject on each of the six saccade groups. Means and standard deviations for latencies of correct trials were calculated for each subject.

This analysis focuses on the comparison of switched and repeated responses, to identify the "residual switch costs" for both prosaccades and antisaccades ("residual" because this reflects the cost that cannot be eliminated by advance preparation during the 2-second period between the prompt and the stimulus[7]). Cost is identified as the subtraction of repeated from switched trial results. A similar subtraction of prosaccade latencies from antisaccade latencies within each condition yields the estimate of antisaccade latency costs (TABLE 1). *A priori* paired *t* tests were used for specifically identified costs in the normal study. In the schizophrenia study, ANOVA was used to compare effects between the two subject groups.

## RESULTS

### *Normal Study*

Task switching lowered the accuracy of prosaccades from 98.7 to 91.9% (paired *t* test, $p < 0.002$). Switching reduced antisaccade accuracy from 90.2 to 84.3% (paired *t* test, $p < 0.01$) (FIG. 2A).

A correct response on a switched antisaccade (ASs) trial requires both a correctly performed task switch and a correctly performed antisaccade. If these two functions are independent, the proportion of correct ASs responses should be equivalent to the proportion correct for antisaccades without switching (repeated antisaccades, ASr) multiplied by the proportion correct for task switching without antisaccades (switched prosaccades, PSs).[9] Thus:

$$\text{ASs} = \text{ASr} \bullet \text{PSs} \quad \textbf{(1)}$$

A paired *t* test comparing ASs to ASr•PSs showed no significant difference ($p = 0.76$). A more stringent test across individual subjects used an error rate linear regression of ASs versus ASr • PSs. This yielded a significant correlation ($r = 0.62$) with a slope of 0.65 and an intercept of 6.2 (FIG. 2B), not differing significantly from a slope of 1 and an intercept of zero ($p = 0.14$). These results are consistent with the hypothesis that these are independent effects.

Task switching increased the latency of prosaccades by 14 ms (SD = 22, $p < 0.02$). However, antisaccades showed the reverse relation: switching reduced latencies by 16 ms (SD = 20, $p < 0.004$). Thus, rather than a switch cost, there was a switch benefit for antisaccades. This paradoxical reduction occurred in 14 of 18 subjects (FIG. 3B). The result was to reduce the antisaccade cost from 64 ms (SD = 32) in repeated trials to only 34 ms (SD = 35) in switched trials (FIG. 3A).

How could such an unexpected reduction arise? We first considered whether it reflected a speed/accuracy trade-off. Certain participants may have been primed to make more rapid responses to the antisaccade prompt when it followed prosaccade trials than when it followed antisaccade trials. If so, latency and accuracy switch effects for antisaccades should be positively correlated; this was not found ($r = 0.14$). Another possibility is that rather than a switch cost from the prior trial, there was an "antisaccade cost," that is, that an antisaccade in the prior trial increased the latency of the next response, whether prosaccade or antisaccade. If so, the switch effect on antisaccades should be negatively correlated with the switch effect on prosaccades; again, this was not found ($r = 0.27$) (FIG. 3B). Rather, the only correlations noted were those from comparisons with the manual reaction time measures of attention from the VIGIL and CalCAP tests. The paradoxical switch effect for antisaccades (but not prosaccades) significantly correlated with three separate measures on these tests; the shorter the reaction times (i.e., the more attentive the subject), the smaller the paradoxical task-switch effect on antisaccadic latency .

### *Schizophrenic Study*

Antisaccades were significantly less accurate than prosaccades (task main effect: $F(1,35) = 58.49$, $p < 0.001$). There was a significant group-by-task interaction ($F(1,35) = 11.06$, $p = 0.002$), with schizophrenic subjects similar to normal subjects

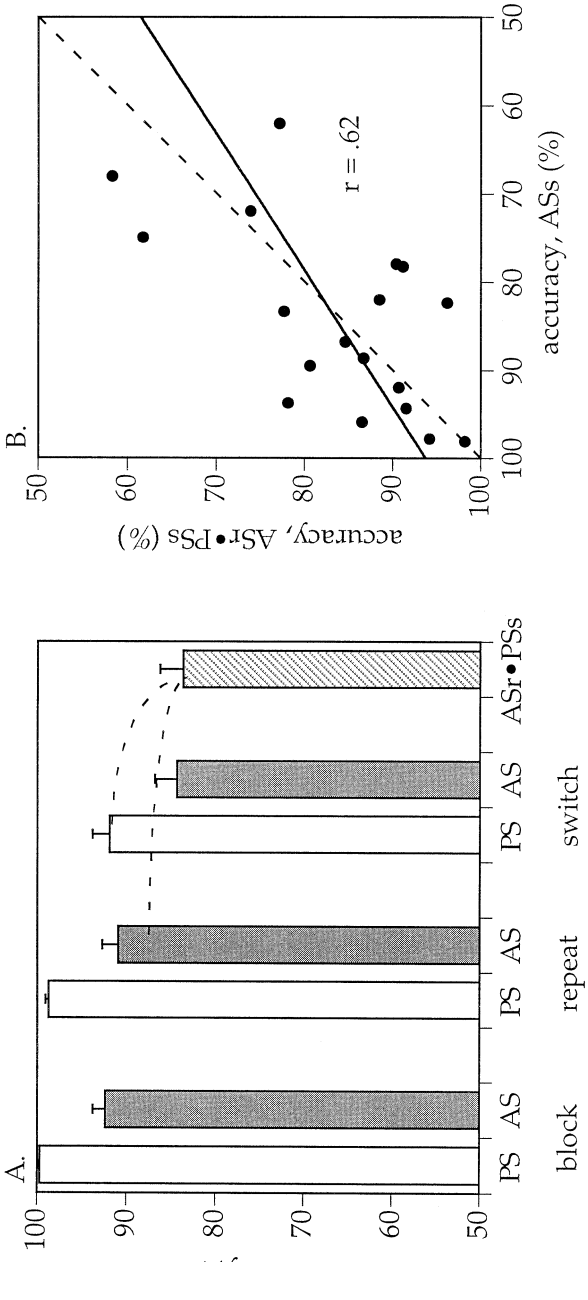


**FIGURE 2.** Error costs, normal study. (**A**) Mean accuracy for prosaccades (PS) and antisaccades (AS) under the three different conditions. "Block" data are from single-task blocks, "repeat" and "switched" data from mixed-task blocks; repeated trials are those with the same type of response requested in the prior trial (e.g., antisaccades preceded by an antisaccade), and switched trials are those with the other response requested in the prior trial. The "ASr • PSs" column is the mean of the product of the accuracy rates of switched PS and repeated AS. If task switching and antisaccades are independent, this should equal the switched AS cost in the adjacent column. Error bars are 1 standard error. (**B**) Linear regression of the switched AS cost (ASs) with the product ASr • PSs across all subjects, indicated as a *solid line*. Independence predicts a line with a slope of 1 and an intercept of zero (*dashed line*).

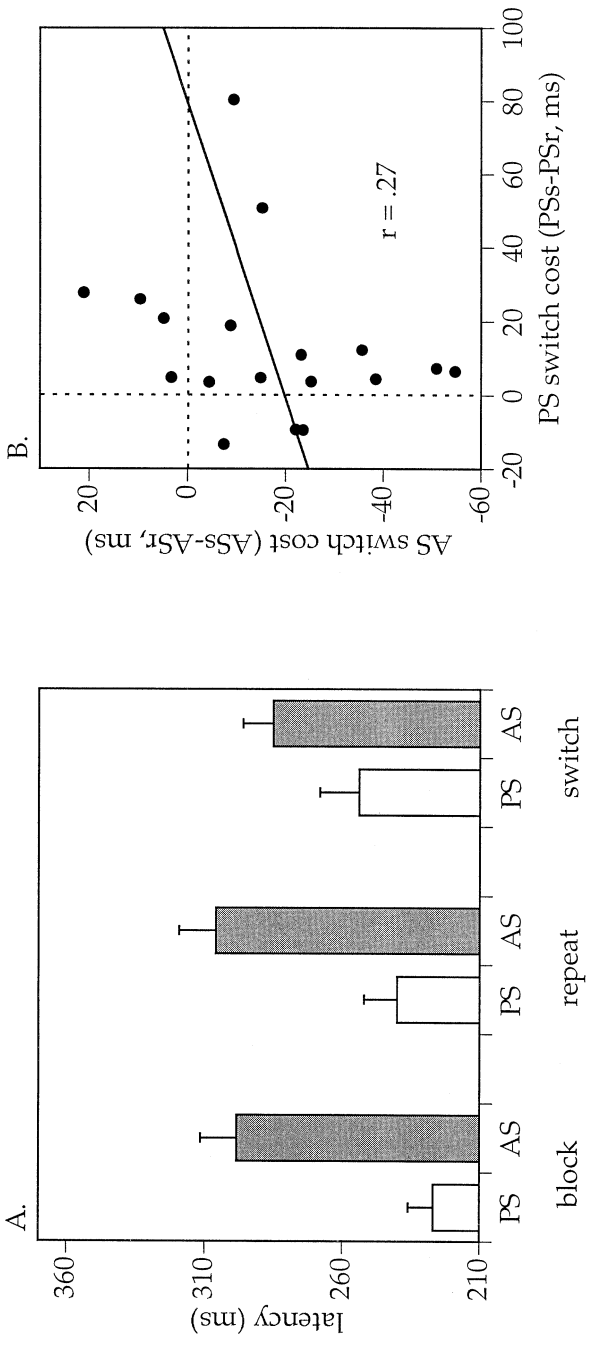


**FIGURE 3.** Latency costs, normal study. (**A**) Mean latencies for prosaccades (PS) and antisaccades (AS) under the three different conditions. Error bars indicate 1 standard error. Note that the mean latency of switched antisaccades is paradoxically shorter than that of repeated antisaccades. (**B**) Correlation of the switch cost for task switching (mean difference of switched minus repeated trials) for prosaccades versus antisaccades. No significant relation is demonstrated.

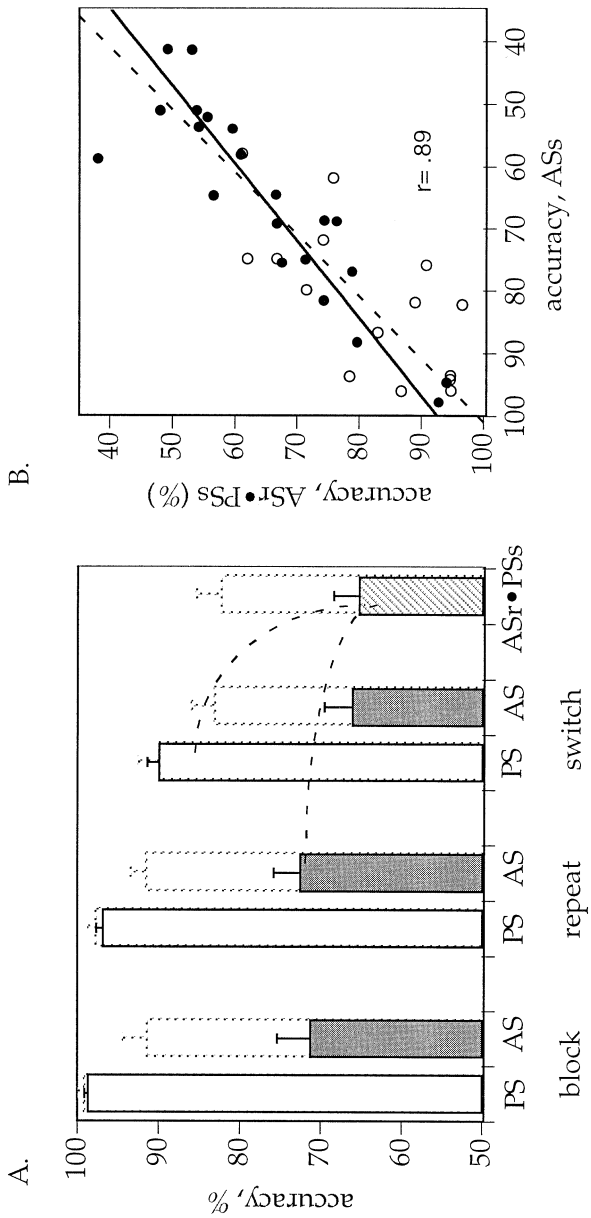


**FIGURE 4.** Error costs, schizophrenic patients. (**A**) Mean accuracy for prosaccades (PS) and antisaccades (AS) under the three different conditions. Conventions as in FIGURE 2. Dotted bars show data for normal age-matched controls. Error bars are 1 standard error. There is a significant reduction of accuracy for antisaccades, but the effect of switching on either prosaccades or antisaccades is no different from that in normal subjects (FIG. 2). (**B**) Linear regression of the switched AS cost (ASs) with the product ASr • PSs across all subjects (schizophrenia, *black circles*; control, *white circles*), indicated as a *solid line*. Independence predicts a line with slope of one and intercept of zero (*dashed line*). The schizophrenic data conform closely to this relationship.

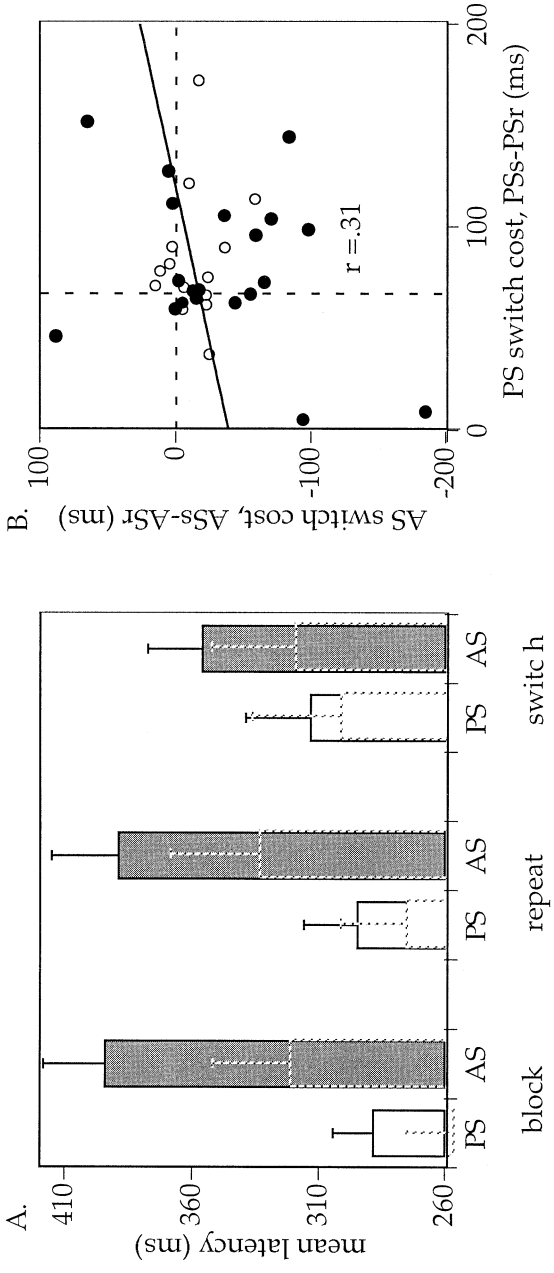


**FIGURE 5.** Latency costs, schizophrenic patients. (**A**) Mean latencies for prosaccades (PS) and antisaccades (AS) under the three different conditions. Dotted bars show data for normal age-matched controls. Error bars indicate 1 standard error. Compared to those in normal subjects, the antisaccade costs are increased, but the task-switching costs are not. Again, the mean latency of switched antisaccades is paradoxically shorter than that of repeated antisaccades. (**B**) Correlation of the switch cost for task switching (mean difference of switched minus repeated trials) for prosaccades versus antisaccades. Schizophrenia, *black circles*; control, *white circles*. No significant relation is demonstrated.

in prosaccade accuracy ($t(35) = 0.59$, $p = 0.56$), but less accurate on antisaccades ($t(35) = 4.57$, $p \leq 0.0001$). Switched trials were less accurate than repeated trials (switch main effect: $F(1,35) = 34.81$, $p < 0.0001$), but there were no significant interactions with group or task (FIG. 4A). Whereas in the normal group the effects on accuracy of antisaccades and task switching were approximately equal ($t(15) = 0.47$, $p = 0.65$), the accuracy costs for schizophrenia were much greater for antisaccades than for task switching ($t(20) = 4.88$, $p < 0.0001$).

The tests for independence of switching and antisaccade function (ASs = ASr • PSs) again showed that ASs did not differ from ASr • PSs ($t(20) = 0.79$, $p = 0.44$) and that there was a strong correlation of ASs with ASr • PSs, with $r$ of 0.89, ($F(1,19) = 33.04$, $p < 0.0001$), a slope of 0.81, and an intercept of 7.4, again not significantly different from a slope of 1 and an intercept of zero (FIG. 4B).

The latency data (FIG. 5) showed that antisaccades were more delayed than prosaccades (task main effect: $F(1,35) = 170.53$, $p < 0.0001$). There was a group-by-task interaction ($F(1,35) = 9.25$, $p = 0.002$); although schizophrenic patients had longer latencies than normal subjects on both tasks (prosaccade $t(35) = 3.33$, $p = 0.0009$; antisaccade $t(35) = 7.09$, $p \ll .0001$), they were much slower to initiate antisaccades. Switching affected the latency of prosaccades and antisaccades differently (switch-by-task interaction: $F(1,35) = 22.08$, $p < 0.0001$). Switching prolonged prosaccade latency ($t(35) = 3.52$, $p = 0.0004$) but reduced antisaccade latency ($t(35) = 3.15$, $p = 0.002$). Group did not interact with switch ($F(1,35) = 1.28$, $p = 0.26$) or with switch by task ($F(1,35) = 1.58$, $p = 0.21$). Thus, schizophrenic patients had similar task-switching effects to those of normal subjects, and the paradoxical task-switching reduction for antisaccade latency was reproduced, being present in 15 of 21 patients. Again, an explanation based on a speed–accuracy trade-off was not supported by correlation analyses of accuracy and latency switch effects in either group (normal: $r = -0.22$, $p = 0.41$; schizophrenia: $r = 0.05$, $p = 0.82$).

Of great interest was the comparison in our schizophrenic patients of task-switching costs with performance on the WCST, a standard clinical instrument purported to measure switching behavior. The group means were in the mildly impaired range for total errors ($\mu = 82 \pm 16$) and perseverative errors ($\mu = 83 \pm 18$). However, there was no correlation between either total or perseverative WCST errors and our task-switching costs in accuracy or latency. Even schizophrenic patients with abnormal WCST performance could have normal task-switching costs.

## DISCUSSION

Our estimates of the antisaccade effects in normal subjects accord with prior results, particularly those of the largest study to date (168 subjects), which documented latency costs of 50–80 ms and accuracy rates of 90%.[22] The finding of much reduced antisaccade accuracy and increased latency costs in schizophrenia is also consistent with prior reports[23–25] (although some studies did not find increased latency costs [26,27]).

We found that the error rates of task switching to prosaccades and of antisaccade performance without task switching (i.e., repeated antisaccades) in normal subjects were similar, about 9%. This was not true in schizophrenic patients, where the anti-

saccade error rate tripled that of normal subjects but task switching to prosaccades was as accurate as in controls. Furthermore, the fact that the interaction of accuracy costs of task switching and of antisaccade performance fit a multiplicative interaction (equation 1) is consistent with independence of current-trial dominance effects from prior-trial–switching effects.[9]

The latency data showed that antisaccade costs were nearly four times the costs of task switching in normal subjects. Again, whereas antisaccade latency costs were elevated by schizophrenia, task-switching costs were not. Schizophrenic patients showed the same pattern of effects of task switching on prosaccades and antisaccades that was seen in controls. Thus, both the accuracy and latency data support the hypotheses that task switching and antisaccade performance are independent and selectively vulnerable to pathology. This has obvious implications for the debate about whether all executive control processes that govern volitional behavior are mediated by a single attentional system or are distributed among distinct prefrontal networks.[28,29]

The fact that our measures of saccadic task switching did not correlate with the WCST results deserves comment. Although instruments such as the WCST are thought to measure task switching, they are multidimensional, requiring several cognitive processes for successful performance. Poor performance on the WCST, for example, can reflect problems in sustained attention, concept formation, or working memory as well as task switching.[30,31] Our results suggest caution in drawing conclusions from these multidimensional tests. On the other hand, it must be stressed that our saccadic task switch involves a fairly pure stimulus-response remapping. Paradigms with additional switches between stimulus dimension, location, response mode, value, or even other factors such as sequence predictability may reveal differences attributable to schizophrenia. At the least, our results place some boundary limits on where any hypothetical task-switching deficit must lie in this condition.

The latency results confirm that asymmetric switch costs are indeed found with a highly asymmetric dominance task pair like the antisaccade/prosaccade relation. This is true even though the switch between these tasks is limited to stimulus-response remapping. However, not only is the cost reduced for our (nondominant) antisaccade task, but also it is reversed, to give a switch *benefit* to antisaccades. Whereas an asymmetry might be construed as consistent with task-set inhibition [5] or stimulus-cued negative priming[8] hypotheses of task switching, no current model of task-switching processes can account for the paradoxical reduction of antisaccade (nondominant) latencies by task switching, which we believe to be a novel finding. Although small, the paradoxical reduction was consistent across subjects and was found in both the normal and the schizophrenic studies. This reduction is not due to a speed/accuracy trade-off, but it is less in subjects who are more attentive, with shorter manual reaction times on tests of vigilance.

How could a paradoxical reduction in latency arise? There are at least two possible explanations. First is that rather than a task-switching cost, there may be a "nondominant stimulus-response mapping cost" carried over from the prior trial, affecting both prosaccades and antisaccades. Thus, an antisaccade stimulus-response mapping in the prior trial may inhibit the saccade system in general in the current trial. Although we could not demonstrate a correlation between task switch costs of prosaccades and switch costs for antisaccades, this does not entirely exclude this possibility, given the magnitude of the within-subject variance in saccadic latencies.

Second, rather than a general detrimental antisaccade effect carrying over from the prior trial, it may be that the operation of a second cognitive function, such as task switching, facilitates the execution of nondominant responses such as antisaccades specifically and yet delays habitual responses such as prosaccades. Some support for this can been found in a recent study of antisaccades performed simultaneously with an attentionally demanding perceptual discrimination task.[32] These investigators found that simultaneous performance of other attentional tasks may interfere with the programming of reflexive responses, both delaying them and also facilitating nondominant responses. In our study, the possibility of an attentional basis to this facilitatory effect on the nondominant antisaccade response is indicated by significant correlations with manual reaction time measures of vigilance. These showed that the paradoxical effect is smallest in those subjects who are most attentive. Thus, those subjects who are most adept at deploying attention may actually need to devote less resources to the secondary cognitive operation of task switching, resulting in less facilitation of the primary operation of antisaccade generation. Which of these two fairly different accounts is responsible for this interesting effect of task switching on antisaccade latency requires further investigation.

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