Investigating Graphesthesia Task Performance in the Biological Relatives of Schizophrenia Patients

by Bernard P. Chang and Mark F. Lenzenweger

Abstract

This study compared the performance of 39 biological relatives of persons with schizophrenia to that of 30 normal adult controls on graphesthesia processing, a complex somatosensory processing task. The relatives performed significantly worse on the graphesthesia task compared to the healthy controls. The relatives and control subjects, however, did not differ on two neurocognitive control tasks. These data are interpreted within the context of a somatosensory deficit linked to schizophrenia liability.

Keywords: Schizotypy, schizophrenia, graphesthesia, somatosensory system, liability indicator.


A wealth of clinical and empirical observations have suggested the possible involvement of a somatosensory deficit in schizophrenia and schizotypic psychopathology (e.g., individuals believed to carry the liability for schizophrenia). Both Kraepelin (1919) and Bleuler (1911) made clinical observations regarding the presence of disturbances in body perception by schizophrenia patients. Bleuler (1911) noted “that even in well-oriented patients one may observe the presence of complete analgesia which includes deeper parts of the body as well as the skin” (p. 57). Rado (1960) and Meehl (1962, 1990) emphasized the presence of somatosensory-related impairments in schizophrenia in their respective models of schizotypic psychopathology and schizophrenia, with each describing such deficits within a model that posited a heritable factor common to both schizophrenia patients and schizotypes. The presence of various somatosensory deficits in schizophrenia and schizotypy has also been supported by numerous empirical studies (for a review, see Lenzenweger 2000). In overview, past work has found schizophrenia patients to show impairments in weight discrimination (Ritzler and Rosenbaum 1974) and pain processing (see Dworkin 1994 for a review) as well as elevated two-point discrimination thresholds (Broekema and Rosenbaum 1975). Extending the exteroceptive somatosensory findings from schizophrenia, Lenzenweger (2000) reported diminished exteroceptive sensitivity in relation to psychometrically assessed schizotypic features; specifically, elevated two-point discrimination thresholds were associated with increased levels of schizotypy-related psychometric deviance (e.g., schizophrenia-related Minnesota Multiphasic Personality Inventory deviance, magical ideation, perceptual aberrations, referential thinking). Chang and Lenzenweger (2001) furthered this work by finding reduced sensitivity (indexed by the signal detection parameter d') in a sample of biological relatives of persons with schizophrenia on a two-point discrimination measure. In this context, it is worth noting that somatosensory functioning encompasses a range of functions that tap into different perception processes, including fine touch, temperature, and pain (nociception), as well as movement, tension, and vibration (Kolb and Whishaw 1996); somatosensation does not reflect the workings of a single unitary system or process. Research in somatosensation in schizophrenia has addressed many of these varying components.

Investigation of somatosensation in schizophrenia and schizotypic psychopathology has not been restricted to the study of basic perception processes. The cognitive processes associated with somatosensory perception have also been examined. For example, psychophysiological work by Josiassen et al. (1985) found abnormal somatosensory evoked potentials to be associated with deviations in attention-related evoked potentials (e.g., P400) in a group of psychometrically identified schizotypes. Boning et al. (1989) found that somatosensory stimulation in a group of persons with schizophrenia was associated with decreased activity in brain regions partici-
pating in an attentional network. Martin et al. (1995) discovered that persons affected with schizophrenia performed worse on a graphesthesia task, which is a complex somatosensory task in which subjects are asked to identify numbers "written" on the palm of their hands with a stylus. The graphesthesia task has a distinct "cognitive" component in that an individual must process and integrate rich information (e.g., identification of number stimuli) received through a basic somatosensory input or route. Martin et al. (1995) interpreted the graphesthesia impairments to be possibly indicative of some form of frontal/parietal abnormality within the schizophrenia patients. However, because schizophrenia patients were examined in this study, these investigators (Martin et al. 1995) were not able to address the extent to which the graphesthesia performance deficits derived from a core schizophrenia liability; reflected a state disturbance (due to psychosis); or were the sequelae of medication, deterioration, and/or institutionalization effects in these patients.

The present study, therefore, extends the past work on graphesthesia into the realm of unexpressed schizophrenia liability and the study of the schizotype. Would similar impairments in graphesthesia performance as those reported by Martin et al. (1995) be observed in a group of individuals at a heightened risk for carrying schizophrenia liability (i.e., biological relatives of persons with schizophrenia)? An investigation of such individuals might help us to better understand the pattern of somatosensory impairments in schizophrenia through an examination of subjects who are most likely to be carriers of schizophrenia liability but whose neuropsychological functioning is not complicated by psychosis or other well-known third-variable confounds. To accomplish the goal of this study, we sought to examine whether performance on a graphesthesia task would be impaired in a group of biological first degree relatives of persons with schizophrenia (i.e., so-called familial schizotypes; see Lenzenweger 1998) compared to a group of normal control subjects. In choosing to study the biological relatives of schizophrenia patients, we were guided by past work that found similar basic somatosensory disturbances (e.g., poorer two-point discrimination performance, elevated pain thresholds) in relation to schizotypy in individuals who had no prior history of psychosis (e.g., Lenzenweger 2000). The study of the first degree biological relatives of schizophrenia patients represents one of three well-known strategies for the study of schizotypic psychopathology, the others being the laboratory approach (e.g., psychometric schizotypy methods) and the clinical/phenomenological approach (e.g., DSM-IV schizotypal personality disorder [SPD]) (see Lenzenweger 1998).

In summary, the primary goal of this study was to determine whether a group of biological/familial schizotypes with no past history of psychosis would display graphesthesia task performance impairments that, ideally, would not be explained as a function of more global neurocognitive impairments. The latter hypothesis regarding possible global neurocognitive impairment could be addressed directly by reference to other data that had been collected previously (Chang and Lenzenweger 2001) on the same subjects.

Methods

Subjects. The study compared a sample of 39 first degree biological relatives of persons with schizophrenia to a group of 30 healthy controls with no family history of schizophrenia. The sample of relatives was collected primarily from three hospitals in the Boston/Cambridge metropolitan region (Massachusetts General Hospital, Cambridge Hospital, and the West Roxbury Veterans Administration). Control subjects were recruited from the same region. To ensure that the relatives had a valid family history of schizophrenia, we required that three criteria be met before a relative could be used in the study: (1) the identified patient relative had to have received the formal diagnosis of schizophrenia from either a psychiatrist or a clinical psychologist, (2) the affected relative had to have received psychopharmacological treatment explicitly for schizophrenia, and (3) the affected relative had to have been hospitalized specifically for schizophrenia on an inpatient service at least once. While the actual relatives that were diagnosed with schizophrenia were not personally interviewed to confirm their diagnoses, we established a high threshold for what would be accepted as evidence of schizophrenia in the affected relatives (e.g., treatment explicitly for schizophrenia in addition to the diagnosis), allowing us to exclude false-positive "schizophrenia patient" cases at the expense of false negatives. No case was accepted as an instance of schizophrenia merely on the basis of psychotic phenomenology; the diagnosis of schizophrenia had to have been given and the treatment administered had to have focused explicitly on schizophrenia as the working diagnosis. The normal control subjects, who were recruited from the same metropolitan area, were screened using the Diagnostic Interview Schedule Screening Inventory (DISSI) to ensure that they had no history of schizophrenia, bipolar illness, or unipolar depression. Control subjects were also screened to ensure they had no prior history of psychosis among their first degree biological relatives. It is noted that any false positives among the schizophrenia relatives or false negatives among the control subjects would only serve to minimize observed group differences on the study measures. All study subjects gave written informed consent to participate in the study after the research procedures had been fully explained to them, and all were monetarily
compensated for their participation in the study. The proportion of male and female subjects was approximately equal in both subject groups (53% female in controls, 56% female in relatives; \( \chi^2(1, n = 69) = 0.07, p > 0.80 \)). The racial/ethnic composition of the overall combined sample was 65 percent Caucasian-American, 10 percent African-American, 18 percent Asian-American, and 7 percent other. The two subject groups did not differ significantly on age (\( t(67) = 0.685, p = 0.50 \)). Moreover, the two subject groups did not differ on educational level achieved (\( t(67) = 1.31, p > 0.20 \)), on paternal educational level (\( t(67) = 0.35, p > 0.73 \)), or on maternal educational level (\( t(67) = 0.87, p > 0.39 \)). The majority of the subjects (36 out of 39) in the relatives group shared no relationship with each other. Moreover, poor performance on the graphesthesia task was not concentrated in relatives from the same families. The subjects in the present study were also used in a separate methodological (i.e., signal detection) study of exteroceptive somatosensation reported previously (Chang and Lenzenweger 2001).

Measures and Procedures

**Graphesthesia task.** The graphesthesia task was based on the procedure outlined in the Halstead-Reitan Neuropsychological Test Battery (Reitan and Wolfson 1993). Graphesthesia is a common task used by neurologists and neuropsychologists in the evaluation of complex somatosensory processing. The task is “cognitive” in the sense that somatosensory input carries a rich body of information (e.g., shape, form) that is perceived, integrated, and processed with respect to number stimuli. With use of a stylus device that had a vinyl-coated pointed brass tip, a series of numbers was “written” on the palmar surface of the dominant hand of subjects (a unilateral assessment is consistent with the past research and clinical literature on graphesthesia administrations). Numbers were drawn from a distal to proximal position (e.g., phalange to carpal) using steady pressure. A correct response was defined as the accurate identification of the number drawn on the palm of the subject. The task involved 15 such trials of number identification. Total scores were generated by assigning 1 point if the subject correctly identified the number and a 0 if the subject did not correctly identify the number; the scores for individual trials were then summed. Subjects were administered practice trials on the graphesthesia task prior to the experimental trials, as described below.

**Control neurocognitive tasks.** To ensure that the relatives of the schizophrenia patients were not displaying evidence of a global neurocognitive deficit, we used data that had been collected on these same subjects for another investigation (Chang and Lenzenweger 2001). The two tasks for which additional data were available were as follows:

1. **Miller-Selfridge Task** (MST; Miller and Selfridge 1950). The MST is a test of verbal recall as well as the ability to use redundancies that have been introduced according to a standard protocol to increase recall as outlined by Miller and Selfridge (1950).

2. **Rey-Osterrieth Complex Figure Test** (RCFT; Osterrieth 1944; Meyers and Meyers 1995). The RCFT is a nonverbal memory task that has been associated with frontal cortex processing (Waber and Bernstein 1995; Savage et al. 2000) as well as parietal lobe functioning (Webster et al. 1994). Subjects are asked to copy and then recall line figures from memory following a 15-minute delay.

**Clinical measures**

1. **Schizotypal Personality Questionnaire** (SPQ; Raine 1991). The SPQ is a 74-item true/false self-report questionnaire that assesses cognitive, perceptual, affective, and interpersonal features based on symptom criteria used in the description of SPD as defined by DSM-III-R (APA 1987). The SPQ generates nine separate dimensional scores, one for each of the nine DSM-III-R SPD criteria.

2. **Psychosis screening** (DISSI; Robins et al. 1981). All subjects completed the self-administered computerized screening version of the Diagnostic Interview Schedule to assess lifetime presence of schizophrenia or a schizophreniform psychosis. No subject in either group met criteria for suspected prior or present schizophrenia-schizotypal psychosis. One member of the biological relative group met screening criteria for bipolar affective disorder and was excluded from the study. No members of the control group met criteria for bipolar affective disorder. Three of the biological relatives met criteria for suspected past major depressive episode, while one of the nonrelative controls met criteria for suspected past major depressive episode.

**Procedures.** All study procedures were administered by either a doctoral student in experimental psychopathology (B.P.C.) or an advanced undergraduate research assistant, both of whom received extensive prior training from the second author (M.F.L.). Subjects who had signed up for the study were contacted by telephone and invited to participate in a study of “perception and cognition,” for which they would be paid an honorarium of $50. Subjects who agreed to participate were individually tested in the second author’s laboratory at Harvard University. After having all aspects of the study explained to them, subjects completed informed consent forms and a demographics
form. Throughout the experiment, because of design of the recruitment and scheduling protocol, all experimenters were blind to the group membership status (i.e., relative vs. control) of all subjects.

Following completion of a demographic information form, the graphesthesia task was administered to the subjects. For the graphesthesia task, each subject was told that the task would be to indicate the number being drawn on the surface of his or her palm. Before beginning the assessment, all subjects received a series of test (practice) stimulations and sample numbers written on their palm to ensure that they could unequivocally, although comfortably, feel the instrument touching their skin. Subjects were unable to view their hand during the task administration, as it was occluded by a screen. The sequence of administration of the number stimuli "written" on the subjects' palms was randomized across subjects. As noted above, the total number of stimuli presented to each subject was 15 and the number of stimuli correctly identified served as the task performance index.

After finishing the graphesthesia task, subjects were also given the SPQ, then debriefed and paid. Total experimental time for the graphesthesia task as well as completion of the demographic form, DISSI, and the SPQ was approximately 1 hour per subject.

**Statistical Analyses.** The two subject groups were contrasted for differences in the mean level of performance on the graphesthesia task using the $t$-test (2-tailed). Cohen's $d$ (1988) was used to measure the size of the observed effect; we also report the effect size $r$ (Rosenthal and Rosnow 1991). Based on prior research (see above), we had a clear-cut a priori hypothesis, namely that the biological first degree relatives of schizophrenia patients would perform more poorly on the graphesthesia task. Associations were examined using the Pearson product-moment correlation coefficient (2-tailed test of significance).

**Results**

The primary focus of this investigation was to determine whether the biological relatives of individuals diagnosed with schizophrenia, who on average have a higher statistical likelihood of carrying schizophrenia liability, would show impaired performance on the graphesthesia task. The mean of the 30 control subjects on the graphesthesia task was 10.17 (SD = 1.60), whereas the mean for the 39 biological relatives was 9.26 (SD = 1.79). The biological relatives of persons with schizophrenia performed significantly worse compared to the normal controls on the graphesthesia task, representing a substantial "medium-sized" effect (as per Cohen 1988; $d = 0.54$, effect size $r = 0.26$). In this context we note that the box plot analysis of the graphesthesia score did not reveal either outlier or extreme value cases in the relative or control groups, suggesting that the results were not being driven by a small number of especially good (or poor) performers in either subject group. The difference between the two subject groups for graphesthesia performance remained significant even when the effects of age and education were accounted for through an analysis of covariance ($F(1,65) = 4.783, p < 0.05$).

A substantive concern was whether any differences seen in graphesthesia task performance could be attributed to a global neurocognitive deficit. Such a consideration is reasonable given, for example, Meehl's (1962) model of schizotaxia, which posits the existence of a general, ubiquitous neurointegrative deficit known as hypokrisia in the central nervous systems of those carrying the schizophrenia liability. As noted above, the subjects in this study had also been evaluated on two neurocognitive tasks that served as control measures, and the subjects' performance on these tasks can be referred to in order to address this issue. As reported in Chang and Lenzenweger (2001), the schizophrenia relative and control groups did not differ significantly on words recalled on any of the four conditions of the MST, the verbal recall (cognitive/memory) task, or on the RCFT (Osterrieth 1944; Meyers and Meyers 1995), a nonverbal memory task (see Chang and Lenzenweger 2001 for extensive detail). Furthermore, correlational analyses done within the schizophrenia relative group alone and for the combined sample of subjects failed to reveal any significant correlations between graphesthesia performance and performance on the RCFT (copy, recall) or MST (four conditions). This pattern of data, for the relatives versus controls, from the MST and RCFT as well as the correlational analyses argues against the suggestion that the relatives' poorer performance on the graphesthesia task was simply the reflection of a global neurocognitive impairment among the relatives. Moreover, the absence of educational level differences is also consistent with the notion that the two subject groups did not differ on global intellectual level, assuming educational level as an approximate proxy for intellectual level.

We also administered the SPQ to examine whether any of the phenomenological features of schizotypal psychopathology were related to performance on the graphesthesia task. For purposes of this exploratory analysis, the Pearson product-moment correlation coefficient was used to assess associations between the SPQ dimensions and the graphesthesia task performance. Table 1 contains data from analyses conducted for the relatives group that provides a full picture of the relations between individual differences on the two measures (i.e., SPQ and graphesthesia performance). As can be seen from table 1, within the rel-
Table 1. Correlations of graphesthesia performance with schizotypal personality disorder features in 39 biological relatives of schizophrenia patients and 30 normal controls

<table>
<thead>
<tr>
<th>Schizotypic feature</th>
<th>Schizophrenia relatives (n = 39)</th>
<th>Normal controls (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ideas of reference</td>
<td>0.09</td>
<td>0.05</td>
</tr>
<tr>
<td>Excessive social anxiety</td>
<td>-0.03</td>
<td>0.09</td>
</tr>
<tr>
<td>Odd beliefs/magical thinking</td>
<td>-0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>Unusual perceptual experiences</td>
<td>-0.36*</td>
<td>0.20</td>
</tr>
<tr>
<td>Odd/eccentric behavior</td>
<td>-0.11</td>
<td>0.12</td>
</tr>
<tr>
<td>No close friends</td>
<td>0.01</td>
<td>-0.34</td>
</tr>
<tr>
<td>Odd speech</td>
<td>0.20</td>
<td>0.05</td>
</tr>
<tr>
<td>Constricted affect</td>
<td>0.24</td>
<td>0.34</td>
</tr>
<tr>
<td>Suspiciousness</td>
<td>-0.02</td>
<td>-0.22</td>
</tr>
</tbody>
</table>

Note.—Values are Pearson product-moment correlation coefficients.
* p < 0.05 (2-tailed)

Discussion

Biological relatives of persons diagnosed with schizophrenia performed significantly worse on a graphesthesia task compared to controls. This effect could not be attributed to differences in age, educational level (as a proxy for general intellectual level), or a global neurocognitive impairment. The results of this study are consistent with the clinical observations of such individuals as Bleuler (1911) and Meehl (1962), who noted the presence of somatosensory dysfunctions in schizophrenia and schizotypic psychopathology. The poor performance on the graphesthesia task in a sample of first degree biological relatives of persons affected with schizophrenia suggests that graphesthesia impairments are not limited to patients with diagnosed schizophrenia and may be seen even in nonafflicted but at-risk biological relatives.

These findings may be interpreted within the context of a neuropsychological disturbance common to both schizophrenia and schizotypic psychopathology that possibly implicates parietal cortex involvement. Much clinical and empirical data support the role of the parietal cortex in a wide number of somatosensory processing tasks (see Kolb and Whishaw 1996 for review). Past work has found the parietal cortex, notably the primary somatosensory cortex (anterior parietal cortex), to be associated with haptic (tactile, fine touch) processing (Pruett et al. 2000), pain processing (Clark et al. 2001), and temperature processing (Zhang et al. 2001) as well as other somatosensory functions such as proprioception (Rabin et al. 1999). From the clinical perspective (i.e., brain damage), impaired performance on the two-point discrimination task (fine touch/exteroceptive processing) has been classically associated with parietally based damage (Semmes et al. 1960; Corkin et al. 1970), with increasing impairments in the tasks most closely associated with primary somatosensory cortex damage (Pause et al. 1989; Kolb and Whishaw 1996; Grusser et al. 2001). Importantly, as discussed in Lenzenweger (2000) and Chang and Lenzenweger (2001), the performance of psychometric and biologically identified schizotypes (as well as schizophrenia patients) on the two-point discrimination task revealed patterns similar to those of patients with parietally based cortical damage (i.e., impaired two-point discrimination performance), which leads one reasonably to consider the possibility that some parietally associated deficit occurs in this population of relatives.

The suggestion of a parietally influenced exteroceptive impairment may provide researchers with a preliminary framework for making sense of the observations of impaired graphesthesia performance. Clearly, the graphesthesia task involves both an exteroceptive somatosensory perception component, but, importantly, the task also involves the integration and processing of number stimuli (a cognitive component). The primary somatosensory cortex is innervated with a rich number of connections to many cortical regions in the brain associated with various
cognitive processes. For example, recent work has suggested that cognitive and perceptual differences in somatosensation may involve patterns of activity between the primary somatosensory cortex and the anterior cingulate (Wei and Zhou 1999; Aziz et al. 2000). Other research has discussed the rich connection between the prefrontal cortex and the primary somatosensory cortex in relation to pain processing and the subjective estimation of pain thresholds (Treede et al. 1999; Olausson et al. 2001). Clearly, the graphesthesia task involves both a cognitive demand (e.g., identification of numbers) and a somatosensory input (e.g., haptic sensation felt on palm). Performance on the graphesthesia task likely involves the need for some “cross talk” between cortical regions associated with cognitive processing and cortical regions associated with somatosensory processing. Therefore, the poor graphesthesia performance observed in the schizophrenia relatives may very well be not the result of either frontal or parietal dysfunction alone, but rather some attempt to integrate the somatosensory information in a cognitively efficient/accurate manner. In our prior study (Chang and Lenzenweger 2001), we collected two-point discrimination data on the same population of relatives studied here and found no significant correlation between graphesthesia task performance and two-point discrimination task performance within the relatives (r = 0.07), clearly suggesting that the two somatosensory processes associated with the different tasks may be relatively separate and, perhaps, unique in their respective neural circuitry.

Several caveats should be noted regarding our study. The first concerns subject recruitment. The biological relatives of individuals diagnosed with schizophrenia were recruited from hospital bulletin board signup sheets placed near clinical facilities in and around the Boston, MA, metropolitan area. The persons who signed up were, therefore, somewhat self-selected and quite aware that they had a positive family history for schizophrenia; however, this feature of subject ascertainment is not unique to this study of biological first degree relatives. In addition, several researchers have claimed that awareness of one’s family history for schizophrenia may explain, in part, the lower scores on self-report schizotypy measures (see Chapman et al. 1994, p. 181), which is reflected, in our minds, in the higher (but not grossly elevated) SPQ feature levels we observed in these relatives (see Chang and Lenzenweger 2001). Furthermore, although structured clinical interviews of the schizophrenia-affected relatives were not done in this study to confirm the diagnosis of schizophrenia, several methodological safeguards were put in place in our study (e.g., insistence on treatment specifically for schizophrenia) to ensure that the affected relatives were likely to have been suffering from schizophrenia. As noted above, any false-positive relatives (i.e., relatives with no true schizophrenia affected relative) would have introduced a level of noise within the relative group that would have diminished the effects we observed on the graphesthesia task between groups. Finally, use of the safeguards we have described has worked well in prior work in which the direct diagnosis of affected relatives could not be accomplished (e.g., Lenzenweger and Loranger 1989).

Another important caveat concerns the type of somatosensory processing that was being assessed with the graphesthesia task. Graphesthesia task performance makes use of haptic processing and perception. Haptic (tactile, fine touch) sensation falls under the larger class of somatosensory processing called exteroceptive, a term that encompasses a wide number of types of somatosensory processing, such as pain and temperature processing. However, in addition to exteroceptive processing, somatosensation covers a much broader range of sensory phenomena, such as proprioception (i.e., limb position and kinesthetics). Prior research has noted the presence of proprioception impairments using a series of weight discrimination (kinesthetic) tasks in persons with schizophrenia (Ritzler and Rosenbaum 1974; Ritzler 1977; Leventhal et al. 1982). It is unclear whether there is any specific relationship between such proprioceptive impairments and the haptic deficits reported in this study as well as Chang and Lenzenweger (2001). We did not administer any proprioceptive tasks along with the exteroceptive tasks in this study and, therefore, were unable to study directly any relevant relationships between the various forms of somatosensation. We are now conducting a study that attempts to improve the understanding of the possible relationship between the various forms of somatosensation processing by examining relative performance on experimental tasks that draw from a variety of forms of somatosensation.

In summary, we found that the first degree biological relatives of individuals diagnosed with schizophrenia showed impairments in a somatosensory task (i.e., graphesthesia) that involves not only haptic (exteroceptive) components but also the cognitive processing of the somatosensory information. The biological relatives did not differ from the control subjects in terms of performance on two control cognitive tasks or educational level (see Chang and Lenzenweger 2001). These data extend the work of Martin et al. (1995), who found graphesthesia impairments in persons with schizophrenia, as well as the work of Chang and Lenzenweger (2001; Lenzenweger 2000), which suggests the presence of a somatosensory disturbance in both psychometrically identified schizotypy and among first degree biological relatives of schizophrenia affected cases. Interestingly, poor performance on the graphesthesia task was essentially uncorrelated with...
performance on a more purely exteroceptive task (i.e., two-point discrimination) in these subjects, a pattern of results that supports the notion of more than one somatosensory deficit being associated with schizophrenia liability. Future investigation examining further the different types of somatosensory impairments in schizophrenia and schizotypic psychopathology may clarify both the presence and the nature of the somatosensory deficits that appear to be linked to schizophrenia liability.

References


Acknowledgments

We thank Robert H. Dworkin, Stephen M. Kosslyn, William Milberg, and Ken Nakayama for valuable input at various stages of this research. We also thank Jennifer Ballard, who assisted with some of the data collection reported here.

This research was supported in part by the Sackler Scholars Programme in Psychobiology at Harvard University, which is supported by the Mortimer and Theresa Sackler Foundation (B.P.C.). It represents part of a series of studies submitted in partial fulfillment of the degree of Doctor of Philosophy in the Department of Psychology at Harvard University, Cambridge, MA.

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