PERGAMON

Neuropsychologia 39 (2001) 771–786

NEUROPSYCHOLOGIA

www.elsevier.com/locate/neuropsychologia

Stroop performance in focal lesion patients: dissociation of processes and frontal lobe lesion location

D.T. Stuss [a,b,*], D. Floden [a,b], M.P. Alexander [a,c,d], B. Levine [a,b], D. Katz [d]

[a] *Rotman Research Institute, Baycrest Centre for Geriatric Care, University of Toronto, 3560 Bathurst Street, Ontario, Canada M6A 2E1*
[b] *Departments of Psychology and/or Medicine, University of Toronto, Ontario, Canada*
[c] *Department of Neurology, Harvard University, Beth Israel Deaconess Medical Center, Boston, MA, USA*
[d] *Boston University School of Medicine Healthnet, Braintree Hospital, Braintree, MA, USA*

Received 19 June 2000; received in revised form 3 January 2001; accepted 10 January 2001

Abstract

There were three primary objectives: to examine the usefulness of the Stroop interference effect as a measure of frontal lobe function; to investigate the possibility of distinct lesion effects for word reading or color naming; and to specifically determine the brain regions necessary for the performance of the incongruent condition. Fifty-one patients with single focal brain lesions in frontal and non-frontal regions and 26 normal control subjects (CTL) were administered the word reading, color naming and incongruent conditions of the Stroop task. Only frontal lesions produced significant impairment. Patients with posterior lesions were not significantly deficient in any condition. Damage to the left dorsolateral frontal lobe resulted in increased errors and slowness in response speed for color naming. Contrary to Perret (Neuropsychology, 1974; 12: 323–330), lesions of the left frontal lobe did not result in a selective interference deficit on the Stroop incongruent condition. Rather, bilateral superior medial frontal damage was associated with increased errors *and* slowness in response time for the incongruent condition. This result is interpreted as failure of maintenance of consistent activation of the intended response in the incongruent Stroop condition. The results and conclusion are compatible with the prevalent theories of both the Stroop effect and the role of the superior medial frontal regions. The role of the anterior cingulate cortex on performance of the Stroop task is likely related to task and patient context.

Keywords: Stroop; Focal lesion; Frontal lobes; Brain-behavior relations

1. Introduction

The Stroop test [65] is one of the most widely used paradigms in experimental psychology and clinical neuropsychology [40,44,56], yet the neural basis of performance on the Stroop test is incompletely understood. In recent years, functional imaging methods have been utilized to identify brain regions active during the component processes involved in the performance of the task. The functional imaging studies have illuminated several brain regions activated by the task, but lesion studies identify the brain areas essential for successful performance. Lesion studies have been relatively uncommon. Those available frequently have had inadequate number of patients or lesion documentation too imprecise to assess the importance of distinct brain regions. The goal of this study was to examine the effect of precisely specified brain lesions, particularly within the frontal lobes, on the different processes involved in Stroop performance.

Multiple versions of the Stroop test exist. The classic version [13,28,59] consists of three conditions: reading color words printed in black; naming color patches using the colors identified by their written names in the first condition; naming the color of ink in which a color name is printed when the color is incongruent with the name ('red' printed in the color green, and the subject names the color instead of reading the word). The third condition normally elicits what is called the 'Stroop' effect — a significant slowing of performance. The number of different words used varies among the test

* Corresponding author. Tel.: +1-416-7852500, ext. 2522; fax: +1-416-7852474.
E-mail address: dstuss@rotman-baycrest.on.ca (D.T. Stuss).

0028-3932/01/$ - see front matter
PII: S0028-3932(01)00013-6

versions, but is usually three or four. The colors are usually highly differentiable prototypical colors presented as either color patches [44] or colored letters (XXXX). The number of stimuli in each condition is usually 100. We used the Comalli [13] version as described in the methods.

There are many methodologies for analysis of test time [23,29,36,41,44,64,77,78]; however, error analysis of the Stroop task has not been common. Stroop [65] used an arbitrary procedure of adding two times the average response time per item for each error, a technique copied by Gardner and colleagues [28]. Smith [63] argued that no correction for errors was necessary because they are too infrequent in adult subjects. Total time measures have not, however, been adequate to demonstrate developmental differences in children, and error analysis has shown age group differences [59,76]. Brain disease may impair word or color processing or cause distractibility, bradykinesia, impulsivity, perseveration, or indifference, all qualities that should influence susceptibility to error [68,73]. Thus, error analysis may be particularly important in investigations of brain injured populations.

Whichever test form is used and however it is measured, results on the three conditions are quite constant among normal subjects. Word reading is performed the fastest, color naming is slower, and the incongruent condition takes the longest to complete. If the results are consistent, the interpretations of the results have not been. Many incorporate some features of modulation of controlled and automatic processing [10,44,77,80]. For instance, if word reading is automatic and color naming is not, or at least less so, words should be read faster than colors are named. The parallel processes may compete for a limited capacity response outlet. The relatively faster speed of processing of words could interfere with the slower color naming, resulting in slower responses or more errors. If word reading is automatic and color naming is not, it is also possible that color naming requires more attention, particularly in the incongruent condition. Some support for this position exists [24], particularly if a gradient of automaticity-control is employed as in the process-dissociation framework [41].

The task instructions hypothetically also invoke working memory, keeping stimulus properties and response options open [35]. The Stroop interference effect could then be the loss of activated response preference in working memory. Within working memory, response preferences must be embedded in a general goal program [11,77]. The on-going interference generated by the successive stimuli in the test, each sharing some perceptual properties with the target stimulus and the response (color name or word) could cause decay of goal maintenance. If not precisely defined, the psychological mechanisms for performance of the Stroop incongruent condition appears to require an ability to maintain a consistent response intention in mind, by some capacity to strengthen or activate one response characteristic [12].

The neural basis for performance of the different conditions of the Stroop is also incompletely understood. Damage to left occipital or temporal structures (whether one lesion or two separate ones) would affect word or color recognition. Damage to left temporoparietal structures would impair word production. Damage to the frontal lobes might result in a general slowing for all conditions. This could be secondary to damage in the medial frontal lobes, because of the importance of this region in initiation and sustaining any behavior [1,66,70]. An alternative hypothesis is that maximum general slowing would occur after left frontal damage in the predominantly right-handed subjects being tested, because of the linguistic-motor demands for all three conditions of the Stroop [4]. None of these observations is novel.

It is the disproportionately impaired performance on the incongruent condition that gives the Stroop test its power and interest. For the incongruent condition, damage to the prefrontal lobes should disrupt performance most. A major role of the frontal lobes is to control response options [27,57,72] through marshalling inhibitory processes, establishing response selection, or maintaining constant activation of the intended goal. It is not surprising that most research on localization of the brain structures required for the Stroop has focused on the frontal lobes.

There is evidence from disparate sources for a general importance of the frontal lobes for the Stroop. Evoked potential studies isolate an important role for anterior (frontal) attentional systems [78]. Among patients with epilepsy, those with either right or left frontal focal abnormalities on EEG are slower on the incongruent condition than patients with temporal lobe abnormalities [15]. Comparisons of epilepsy patients after either frontal or temporal lobectomies have demonstrated that frontal surgery produces more impairment. There may be greater slowness in all conditions, but disproportionately so for the incongruent condition without increasing error rates [60] or an increase in error rates with marginal slowing [15]. Depending upon instructions, there may be an important speed–accuracy trade-off, at least in patients with frontal lesions. No strong localization claims were made, but these patients had primarily superior medial excisions.

Many different studies have defined regions within the frontal lobes that may have more specific roles in processes necessary for the Stroop. There are complementary studies from functional imaging in normal individuals and neuropsychological assessment of lesion effects. From functional imaging different investigators

have proposed a variety of frontal sites as key: left inferior lateral [74], left superomedial [50], right frontal polar [3] and bilateral anterior cingulate, perhaps with right predominance [3,50]. In an fMRI study of a different task that incorporated some of the demands of the Stroop, both lateral frontal regions appeared recruited for monitoring and detecting errors and both cingulate regions appeared activated for sustaining attention during interference [8]. Lesion studies have also suggested different possible frontal regions: left lateral [15,30,53], right lateral [75] and superomedial [31]. Stuss et al. [69] demonstrated that the orbital frontal region is *not* essential for the Stroop.

The repeated demonstration of medial frontal participation in the incongruent Stroop, either directly in activation or indirectly by impairment in lesion studies, suggests a critical role for the ACG and/or the SMA. These experiments converge with abundant evidence that the ACG is an essential structure for modulation of attention and intention, particularly for complex tasks [7,21,46,57]. The precise neural operations that underlie this modulation and control are not known. They have been operationally described in various ways, usually determined by the nature and terminology of the tasks used to probe for them: selection of information for attention to action, particularly if inhibition of another competing response is required [3,50,57]; in divided but not necessarily selective attention conditions [14,54]; monitoring and compensating for errors [20]; on-line general performance monitoring independent of errors [8]; facilitating correct and suppressing inappropriate responses [52]; cognitively demanding tasks in general [25,26,61]; and anticipation in doing a task rather than actual task processing [48].

This study had several hypotheses: (1) The frontal lobes are, perhaps in many regionally specific ways, essential for the Stroop test. Comparing patients with frontal and non-frontal lesions should confirm the usefulness of the Stroop as a clinically specific measure of frontal function. (2) Left ventrolateral frontal regions are involved in lexical-semantic generative tasks. Excluding patients with damage to posterior structures essential for early processing of colors and words, i.e. with significant visual impairment, neglect, alexia or aphasia, only left lateral frontal lesions uniquely should cause slowing of color naming and/or word reading. An independent role of the left frontal regions in the incongruent condition was not expected, even though this is the brain region frequently associated with the Stroop effect. (3) The superior medial frontal lobes play a critical position in response modulation, especially when complexity or response competition are present. Controlling for overall slowness, only SM lesions should cause greater impairment in the Stroop incongruent condition. Within the SM region, the ACG should be most critical.

2. Methods

2.1. Subjects

Fifty-one patients with focal brain lesions and 26 normal subjects (CTL) were administered the Stroop task as part of a longer neuropsychological examination. Informed consent was obtained in all cases. This study was approved by the joint ethical committee of the University of Toronto and Baycrest Centre for Geriatric Care. Patients met the following inclusion criteria: presence of a single focal frontal or non-frontal lesion, with three instances of frontal damaged individuals with small non-frontal lesions; availability of a CT or MRI scan for lesion localization; time since onset greater than 1.8 months. One patient with a left non-frontal lesion who met the above criteria was not included because of extreme slowness in reaction time compared to all other patients. Patients were excluded if neuropsychological screening demonstrated significant comprehension deficit, alexia, or neglect.

All subjects were native English speakers with intact color identification (red, blue, green), adequate ability to read, and no prior history of any neurological or psychiatric disorders.

The patients were subdivided into five lesion groups using standard anatomical methods of lesion classification: right frontal (RF) ($n = 14$), left frontal (LF) ($n = 8$), bifrontal (BF) ($n = 15$), right non-frontal (RNF) ($n = 7$) and left non-frontal (LNF) ($n = 7$). Lesion volume was quantified using a pixel tabulation method. For each patient, for each axial slice in which a lesion was evident, the size of the lesion was quantified by superimposing the lesion on a constant pixel diagram and counting the number of pixels. This number was then divided by the total pixel count for all axial slices in order to obtain a total percentage of brain affected. The lesions for each patient are depicted in Fig. 1. For a small number of cases, precise verbal descriptions of lesion locations were used because scans had been available for lesion localization but were subsequently lost for quantification and depiction (RF–2; BF–1; RNF–1; and LNF–4). The lesion location, lesion size, etiology of injury, and chronicity of the injury for each patient in each group are summarized in Table 1. There were no significant group differences in lesion size (Kruskal–Wallis $\chi^2 = 2.1$, $P = 0.7$). While etiology varied, previous research had indicated no effect of etiology in other studies of frontal lobe functions [6] [67]. Tumors excised were benign primary tumors. Lobectomies were performed for correction of epileptic seizures. In the trauma cases, care was taken to exclude individuals with significant diffuse axonal injury. There was a significant group difference in lesion chronicity (Kruskal–Wallis $\chi^2 = 13.8$, $P < 0.01$). Orthogonal post hoc comparisons with a Bonferroni corrected alpha

indicated that the lesion chronicity of the frontal groups was significantly shorter than the non-frontal groups (Mann–Whitney $U = 98.5$, $P < 0.01$) but the frontal groups did not differ from each other (all $P > 0.2$).

The patient groups and the control group did not differ significantly in age, education, gender, and handedness, with all differences being greater than $P > 0.05$ (see Table 2). The National Adult Reading Test (NART) [49], Digit Span forward and backward, and the Boston Naming Test (BNT) [33] were administered to assess the comparability of the groups on other cognitive processes and to use in correlational analyses as an index of processes relevant to the completion of the Stroop task. There was a significant group difference only on the NART [$F(5, 71) = 4.0$, $P < 0.01$] and the BNT [$F(5, 67) = 3.6$, $P < 0.01$]. Post hoc examination of the group difference in the NART revealed that the CTL group had significantly higher scores than the RF and the BF groups while the BNT effect was due to the poorer performance of the LNF group compared with RNF and CTL groups (see Table 2).

2.2. Materials

The Stroop task used in this study, based on Comalli and colleagues [13], consisted of three conditions presented in a fixed order: word reading (WR), color naming (CN), and incongruent color naming of color words (INC). In each condition, subjects were presented with an $8\frac{1}{2} \times 11$ in. sheet of paper with 100 items arranged in 10 rows and columns. For each condition the subject was instructed to read the words (or name the colors) row by row as quickly and accurately as possible, without skipping any. In the WR condition, stimuli were three color words (red, blue, and green)

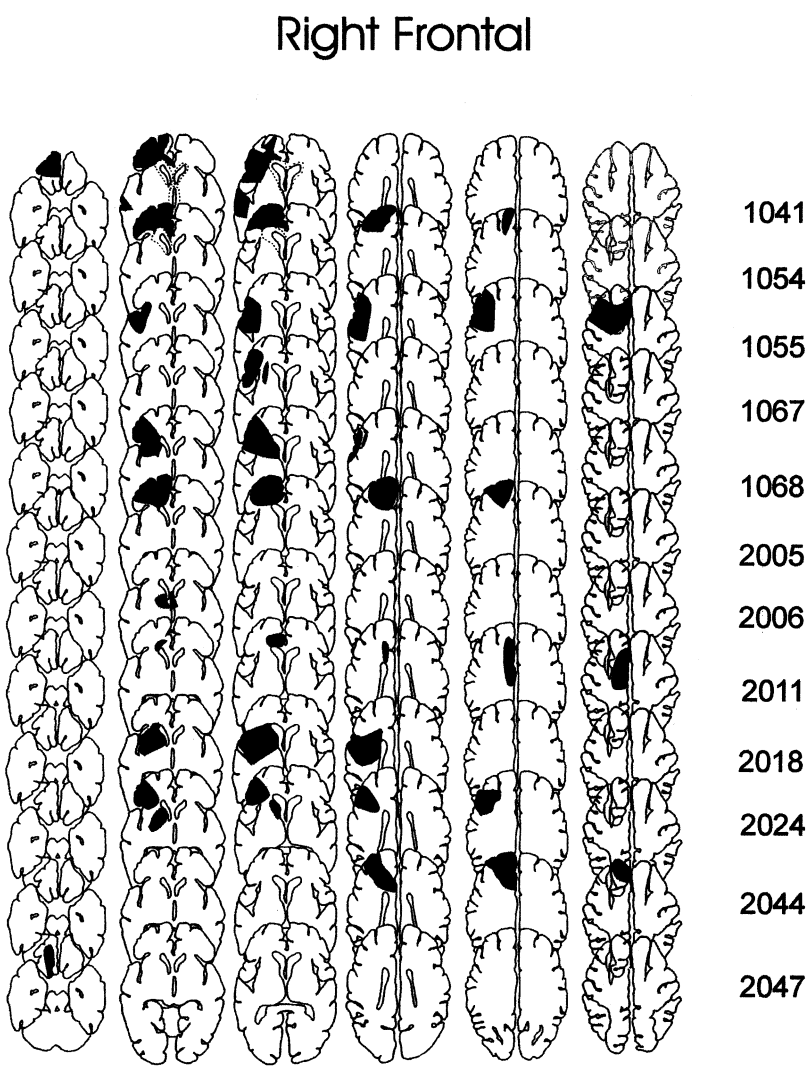

Fig. 1. Lesion location of each subject within the five patient groups. The bilateral lesion of subject 2042 is evident on axial slices that are not represented here. In some cases, scans were available for localization but subsequently lost (see text). Verbal descriptions of lesion locations for all patients are listed in Table 1. RF, right frontal; LF, left frontal; BF, bilateral frontal; RNF, right non-frontal; LNF, left non-frontal.

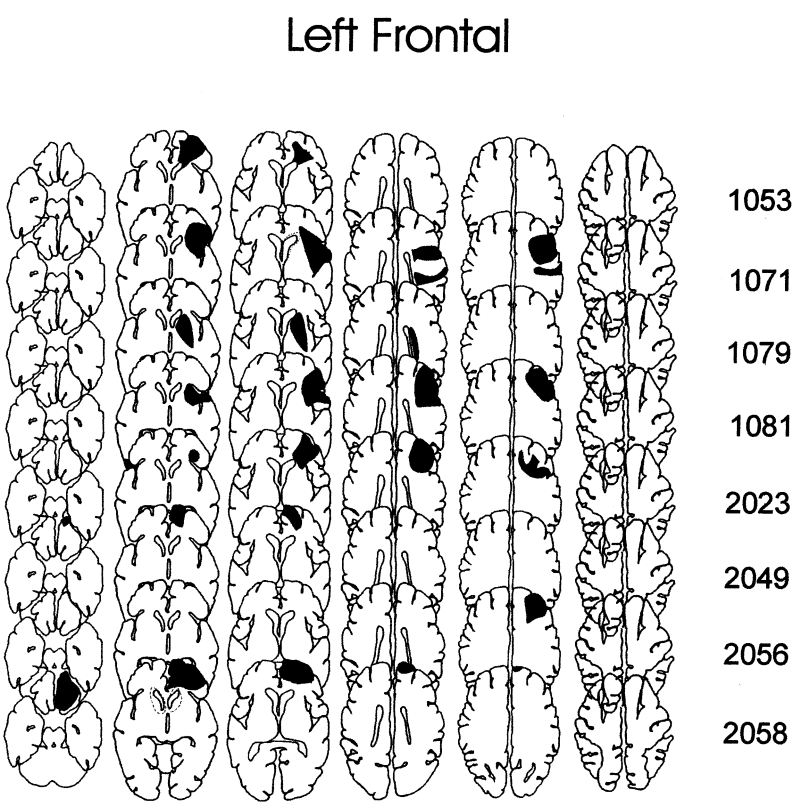

Fig. 1. (*Continued*)

printed in black ink. In the CN condition, stimuli were small (6×13 mm) red, blue, and green rectangles. Finally, in the INC condition, stimuli were the color words red, blue and green printed in an incongruent color. The subject had to name the color of the ink in which the word was printed, i.e. if the word 'blue' were printed in green ink, the correct response would be 'green'. For all conditions stimuli were arranged on the page in a pseudorandom order, and no more than two identical stimuli occurred together.

2.3. Dependent measures

Measures relating to speed and accuracy were assessed. There were three categories of speed measures. The first two speed measures were selected for comparison to previous research. The first was total time in seconds to complete each of the three conditions. Second, we computed difference scores for successive pairs of conditions. These were CN minus WR (CN − WR), and INC minus CN (INC − CN). Difference scores are usually the standard measure by which to determine the degree of interference that the incongruent task elicits [44]. To overcome baseline differences in response speed, the third speed measure was a proportion score which takes into account the difference in RT between two tasks as well as each individual's baseline speed. Based on previous research, proportion scores were calculated for each subject for CN related to WR, and INC related to CN. These were computed by dividing the difference score by the total time of the earlier condition (e.g. color naming to word reading = (CN–WR)/WR). Finally, three dependent measures concerning accuracy were calculated: (a) errors; (b) self-corrections (errors that were immediately self-corrected); (c) total errors and self-corrections.

2.4. Statistical analyses

The first set of analyses consisted of an investigation of group differences based on the standard anatomical group classification described above for each of the dependent measures. Corrections for non-normal distributions, such as logarithmic transformation of RT prior to calculation of the proportions, did not alter the results. Rather than excessively distort the dependent measures or trim the data we relied on the robustness of the ANOVA test. Since the groups had significantly different NART and BNT scores, these variables were initially used as covariates; however, the variance accounted for by BNT scores did not alter the pattern of results and therefore only NART scores were covaried. All post hoc analyses were done using the Bonferroni procedure. The accepted alpha level was $P < 0.05$ for all analyses.

The second set of analyses was performed to address our major objective of evaluating the specific brain regions involved in Stroop performance. Using our modified case study/group approach [66,67,71,72], patients were grouped by level of performance on a defined measure, and then specific brain correlates and relations to other processes examined. The number of errors rather than the speed measures was selected as the major ranking criterion for the reasons outlined in the review, and also because accuracy measures appeared to be more sensitive to task difficulty than speed. Total errors were chosen as the best index of performance accuracy because the resulting group of poor performers incorporated all subjects that made a disproportionate number of self-corrections or combined errors and self-corrections. Very few subjects committed WR errors. Using the criterion of a number of errors greater than 1.5 SD above the CTL mean, subjects were categorized as either good or poor performers for the CN and INC conditions separately. This binary classification of performance was used in order to avoid the inherent variance problems when attempting to analyze outliers. Two cases, which had moderately high number of errors although not reaching the criterion and whose proportion speed score was greater than 2.5 SD from the CTL mean, were included in the poor performer group because of the speed–accuracy trade-off. No non-frontal patients performed within the abnormal range on the stated error criterion. Lesion location analyses based on error performance

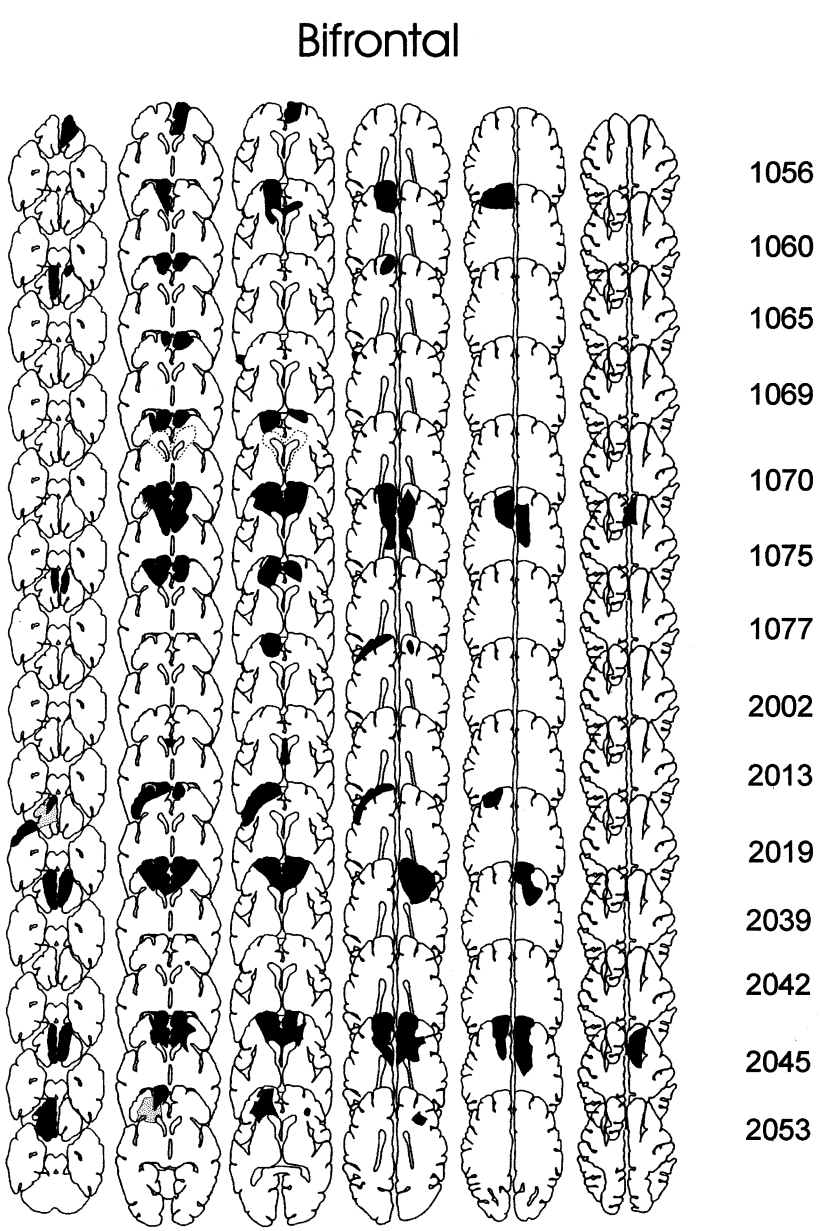

Fig. 1. (*Continued*)

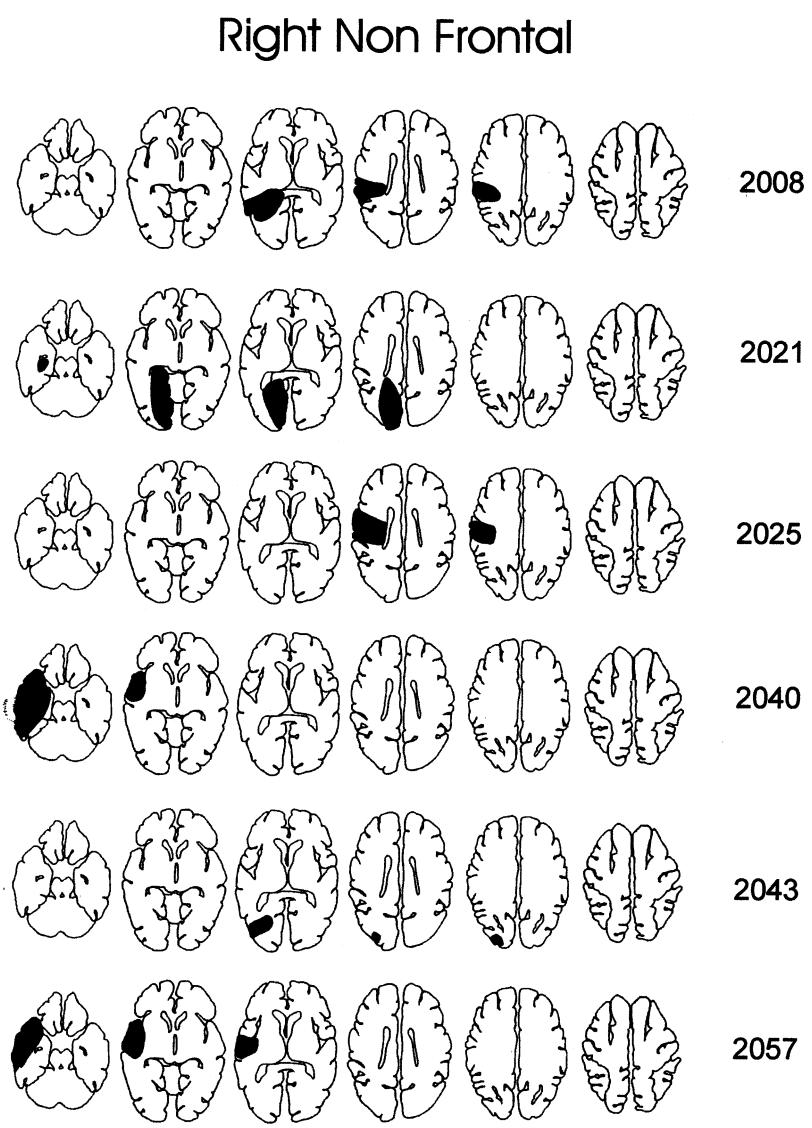

Fig. 1. (*Continued*)

were therefore completed only with the 37 frontal damaged patients.

To investigate whether impairment on the Stroop task could be isolated to more specific regions within the frontal lobes, a template based on divisions of the frontal lobes proposed by Alexander in Stuss et al. [72] and the cytoarchitectonic areas reported in Petrides and Pandya [55] was devised and superimposed on drawings of each subject's lesion. The following frontal lobe regions were delineated: left and right lateral (Petrides and Pandya Areas (PPA) 9/46, 44, 45, 46, 6A, 6B, 8A); polar (most anterior portion of PPA 8B, 9, 10); inferior medial (PPA 10, 11, 14, inferior portion of 32), superior medial (PPA 8B, 9, and superior portion of 32), anterior cingulate (PPA 24, 25), superior posteromedial (medial and superior aspect of PPA 6B), left and right caudate, and the septal region. Each of these frontal lobe regions were coded as 0 (no lesion present) or 1 (lesion present).

3. Results

3.1. Standard anatomical group differences

Using total time in seconds as the dependent measure, significant group differences were observed for all three conditions: WR [$F(5, 71) = 8.06$, $P < 0.\ 001$]; CN [$F(5, 71) = 11.86$, $P < 0.001$]; and INC [$F(5, 71) = 7.45$, $P < 0.001$]. In the WR and INC conditions, the LF group was impaired relative to all other groups except the RF group, and the RF group was impaired relative to the CTL group only. For the CN condition, the LF group was impaired relative to all other groups while

the RF group differed only from the CTL group. The LF slowing was also observed for the CN − WR difference measure, the LF group having a significantly higher difference score than all other groups except for the LNF group. There were no significant differences between groups for the defined proportion scores. (See Appendix A for means and standard deviations of each group for all conditions.)

3.2. Summary of standard anatomical group comparisons

First, patients with left and right frontal lesions (particularly those with left) were slower throughout. Conclusions about the frontal lesions and the Stroop incongruity effect must account for this general slowing effect. Second, the LF group had difficulty with color naming, reflected both in speed of response and in the number of errors committed. Third, none of the patient groups had excessively disproportionate slowing on the incongruent task. The large variance, however, suggested a need to regroup the frontal damaged patients in a manner other than standard anatomical classifications.

3.3. Anatomical grouping by error scores

Because we had coded each of the brain regions as damaged or non-damaged, we were able to investigate the relationships between a defined performance measure and each specific region using the Phi coefficient, a correlation measure for dichotomous data [9]. Although lesions in any individual may extend to several areas, this procedure enabled us to isolate the key location across individuals.

3.4. Color naming performance

For the CN condition, 30 of the frontal damaged subjects were classified as good performers and seven as poor performers. There were no significant differences between these performance-based groups in education, gender, handedness, lesion size, chronicity (in months), Boston Naming Test, or NART scores. There was a significant age difference between groups, such that the poor performers were older (mean = 59.6, SD = 9.5) than the good performers (mean = 49.0, SD = 12.6) [$t(35) = 2.08$, $P < 0.05$].

The only region that showed a significant relationship using the Phi correlational analysis with the number of errors on color naming was the left lateral area ($\phi = 0.584$, $P < 0.001$). When the speed measures were compared between the good and poor performers on the error measures, there were significant differences in straight color naming time [mean (SD), good performers = 80.9s (25.8), poor performers = 108.6 (35.8); $t(35) = 2.4$, $P < 0.05$] and for the WR/CN proportion

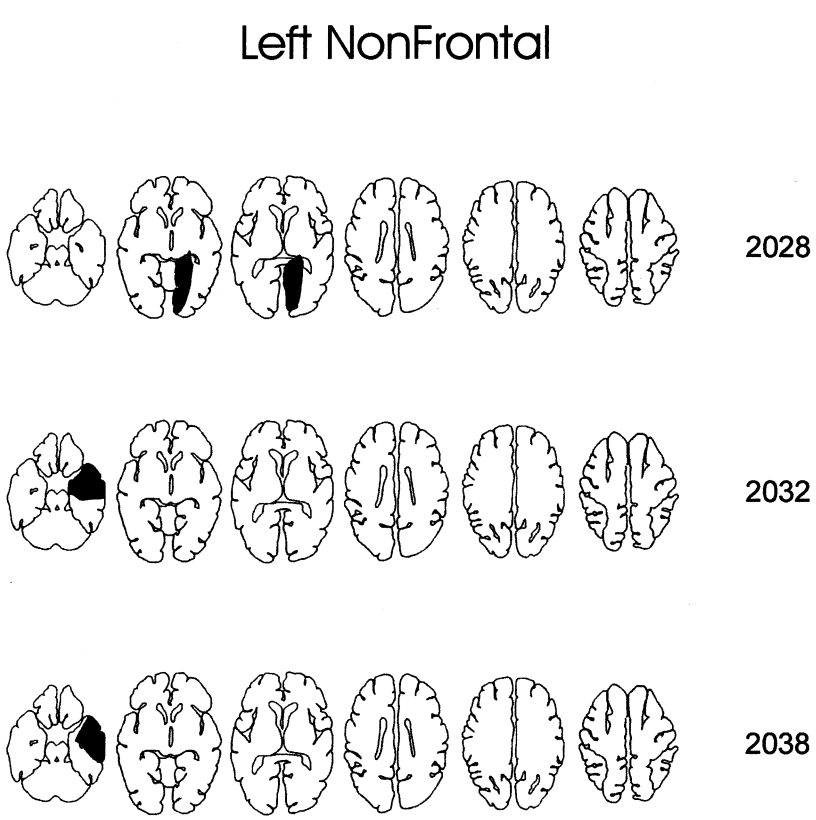

Fig. 1. (*Continued*)

Table 1
Lesion location and etiology within patient groups

Subject no.	Lesion location	Etiology	Lesion size[a]	Chronicity[b]
Right frontal				
1041	Dorsolateral, inferior medial	Lobectomy	3.41	4.2
1054	Inferior medial, dorsolateral, ACG	Tumor	3.60	24.7
1055	Superior medial, dorsolateral	Infarct	4.16	10.9
1064	Dorsolateral, striatal	Stroke	NA	14.7
1067	Dorsolateral	Stroke	1.13	21.0
1068	Dorsolateral, striatal	Stroke	3.43	7.4
2005	Medial, dorsolateral, ACG	Tumor	3.76	3.6
2006	Striatal, inferior medial, septal	Stroke	0.25	22.5
2011	Superior medial, ACG	Stroke	2.15	3.6
2018	Dorsolateral, striatal	Stroke	2.50	4.5
2024	Dorsolateral, striatal	Stroke	2.30	2.5
2027	Dorsolateral, striatal	Stroke	NA	3.6
2044	Superior medial, ACG	Tumor	1.63	3.6
2047	Inferior medial	Stroke	0.75	3.5
Mean (SD)			2.4 (1.3)	9.3 (8.1)
Left frontal				
1053	Dorsolateral	Trauma	1.46	291.1
1071	Dorsolateral, parietal	Stroke	4.23	12.7
1079	Striatal	Stroke	1.14	10.7
1081	Dorsolateral	Hemorrhage	2.85	10.0
2023	Dorsolateral, occipital	Stroke	3.82	2.4
2049	Medial, polar	Hemorrhage	0.85	3.4
2056	Dorsolateral	Tumor	0.83	10.4
2058	Medial, dorsolateral	Tumor	2.32	74.5
Mean (SD)			2.2 (1.3)	51.9 (99.5)
Bifrontal				
1056	Inferior medial, ACG	Stroke	1.53	33.1
1060	Medial, ACG	Stroke	2.17	6.1
1065	Inferior medial	Trauma	1.29	15.6
1069	Inferior medial	Tumor	0.33	2.5
1070	Medial, ACG(R)	Stroke	2.25	2.6
1075	Medial, ACG	Hemorrhage	8.79	22.1
1077	Inferior medial	Trauma	1.75	10.2
2002	Medial, dorsolateral, ACG(L)	Infarct	1.63	4.6
2013	Inferior medial, septal, ACG	Stroke	0.46	8.9
2014	Inferior medial	Stroke	NA	18.7
2019	Dorsolateral, medial, temporal	Trauma	2.69	8.3
2039	Medial, ACG	Hemorrhage	8.03	1.8
2042	Inferior medial	Trauma	0.13	5.9
2045	Medial, septal, ACG	Stroke	9.55	59.8
2053	Inferior medial(R), dorsolateral(L), ACG(R)	Trauma	3.18	3.4
Mean (SD)			3.1 (3.2)	13.6 (15.5)
Right non-frontal				
2008	Temporal, parietal	Tumor	2.90	9.3
2021	Temporal, occipital	Stroke	4.97	4.5
2025	Parietal	Stroke	2.89	4.8
2040	Temporal	Lobectomy	2.79	89.3
2043	Occipital	Stroke	0.78	36.3
2055	Temporal	Hemorrhage	NA	55.3
2057	Temporal	Lobectomy	4.05	134.6
Mean (SD)			3.1 (1.4)	47.7 (49.4)
Left non-frontal				
1049	Temporal	Tumor	NA	17.8
2028	Temporal, occipital	Stroke	1.13	28.5
2031	Parietal	Stroke	NA	15.9
2032	Temporal	Lobectomy	2.02	49.6
2036	Temporal	Lobectomy	NA	91.3
2038	Temporal	Lobectomy	1.58	144.7
2054	Temporal	Lobectomy	NA	142.6
Mean (SD)			1.6 (0.4)	70.1 (56.4)

[a] Percent whole brain.
[b] Months post incident.

score [mean (SD), good performers = 0.38 (0.29), poor performers = 0.75 (0.42); $t(35) = 2.7$, $P < 0.01$] but not for the other measures ($P > 0.1$). These differences did not alter when the effect of age was removed. In the poor performer group, neither color naming errors nor color naming time were related to naming ability as measured by the BNT ($r = -0.14$, $P > 0.70$; $r = -0.07$, $P > 0.85$, respectively), verbal fluency ($r = -0.10$, $P > 0.76$; $r = -0.39$, $P > 0.22$, respectively), or semantic fluency ($r = 0.05$, $P > 0.87$; $r = -0.43$, $P > 0.17$, respectively).

The differences in lesion location in relation to performance are illustrated in Fig. 2.

3.5. Incongruent performance

For the INC condition, 25 of the frontal damaged subjects were classified as good performers and 12 as poor performers based on the number of errors in the INC condition. There were no significant differences between these good and poor performer groups on any demographic variable or neuropsychological measure.

The lesion location analysis indicated that bilateral superior medial lesions as well as right superior posteromedial lesions were significantly related to performance ($\phi = 0.317$, $P = 0.05$; $\phi = 0.503$, $P = 0.002$, respectively) (see Fig. 3). The poor performers were also slow, being significantly different in response speed for the CN [mean (SD), good performers = 78.3s (26.7), poor performers = 102.5 (29.3); $t(35) = 2.5$, $P < 0.05$] and the INC condition [mean (SD), good performers = 145.0 s (32.2), poor performers = 219.3 (85.6); $t(12.5) = 2.91$, $P < 0.05$] and almost significant for the WR condition [mean (SD), good performers = 56.3 s (21.4), poor performers = 69.9 (14.3); $t(35) = 1.99$, $P < 0.06$).

3.6. Further anatomical observations

There were a total of 14 patients who had involvement of the anterior cingulate gyrus, 10 bilateral and four right. On CN, no patients with ACG damage were in the poor performer group. In the INC group, six of the total 14 anterior cingulate damaged patients (four BL and two R) made a sufficient number of errors to be in the poor performer group. However, these six subjects also had superior medial damage. There were four subjects with medial damage restricted to inferior cortical areas, including the ACG. These four performed normally. The correlational analysis indicated that the ACG was not significantly related to poor performance for the INC condition ($\phi = 0.174$, $P > 0.29$). There were no significant differences between patients with and without ACG involvement on any speed measure.

3.7. Summary of anatomical groupings by errors

The error analysis clarified the finding that the left frontal group was impaired in color naming. This result is due to the left *lateral* frontal group's performance. For the INC condition, patients with lesions in either bilateral superior medial or right superior posteromedial areas made the most errors. They also had the slowest overall response times in all conditions.

4. Discussion

The essential claim for the Stroop incongruity effect is that color naming is disproportionately slowed in the presence of lexical-semantic interference, the printed color name, compared to straightforward color naming. This is a normal finding in all subjects and patients. The essential diagnostic claim in neuropsychology is that this effect is exaggerated after frontal lobe damage. We found that the Stroop incongruity effect, measured as disproportionate time for the interference condition, is not affected by frontal lobe damage in general, nor by undifferentiated left or right or bilateral lesions. Patients with frontal lesions, particularly left sided, are simply slow on all conditions.

Table 2
Demographic characteristics and neuropsychological test[a] results of patient groups and matched control subjects

Group [b]	Gender		Hand			Age		Education		BNT		BDI		DSF		DSB		NART	
	M	F	L	R	A	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
RF	7	7	0	13	1	52.3	12.2	12.1	2.8	52.2	5.0	3.9	5.5	6.2	1.2	5.1	1.4	104.6	10.5
LF	6	2	0	8	0	58.0	13.6	13.5	3.5	49.9	7.9	3.7	2.8	6.0	0.9	4.5	2.5	105.0	8.4
BF	9	6	2	12	1	46.1	11.5	11.4	2.5	50.6	7.7	7.7	5.2	6.7	0.9	4.6	1.3	102.2	7.7
RNF	5	2	1	6	0	49.0	16.6	13.4	2.9	55.4	6.6	5.0	4.6	7.3	1.1	6.0	2.0	110.9	8.7
LNF	2	5	0	7	0	44.1	16.4	13.4	1.6	44.3	9.9	3.2	1.3	5.8	i.2	4.7	1.5	104.4	9.4
CTL	10	16	5	21	0	55.3	14.2	13.8	2.4	55.0	4.2	3.3	2.3	6.9	1.4	5.9	1.1	119.1	20.1

[a] BNT, Boston Naming Test; BDI, Beck Depression Inventory; DSF, Digit Span Forward; DSB, Digit Span Backward; NART, National Adult Reading Test.

[b] RF, right frontal; LF, left frontal; BF, bifrontal; RNF, right non-frontal; LNF, left non-frontal; CTL, matched control.

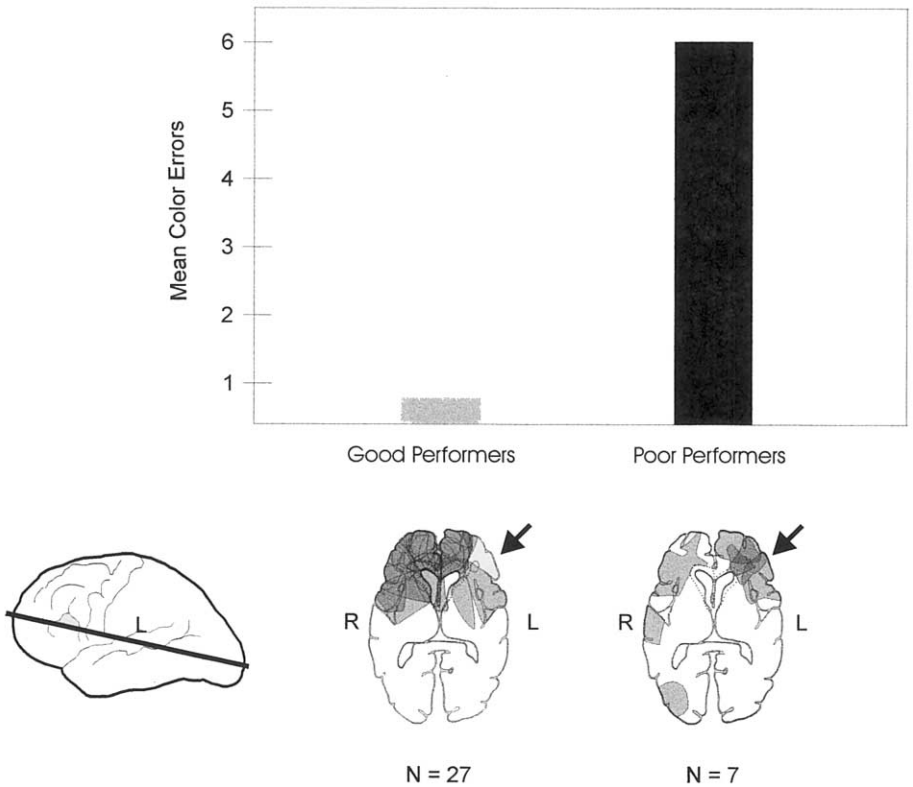

Fig. 2. Mean color errors and lesion locations of performance-based (error) groups for the color naming (CN) condition. Poor color naming performance was associated with lesions in the region indicated by the arrow (left lateral, $\phi = 0.584$, $P < 0.001$). There were three missing scans in the good performer group.

Left frontal lesions impaired direct color naming, complicating any interpretation of a Stroop effect. Slow color naming in the left frontal group was part of generally slow responses in all conditions in that group. A subgroup of the left frontal lesions — those with lateral frontal damage — had increased errors and particularly slow performance in color naming, but even they did not show disproportionate interference in the incongruent condition. Previous studies that claimed that left frontal injury is associated with exaggerated Stroop effect did not control for direct color naming [29,53]. The current study, the first with control for baseline color naming and with precise lesion localization, demonstrated that the left lateral frontal effect is for color naming directly.

Why color naming should be more vulnerable than word reading after left frontal damage is not certain. Patients with aphasia were excluded from the study. There was no correlation between performance on the color naming portions of the Stroop and other measures of language — the BNT or FAS. The Stroop color naming deficit seen with left lateral frontal lesions does not, therefore, appear to be a non-specific manifestation of impaired naming in recovered mild aphasia. There is evidence for left hemisphere dominance in color processing [42] and naming, both in normal [18] and brain injured [16,17] individuals. Thus, there could be a left hemisphere network for color naming. Within the network, lateral frontal structures would be recruited when considerable control of the response output is demanded by the continuous presentation of exemplars for response within one semantic class. The same argument — left posterior dominance and left frontal response control — could be made for oral word reading, however, so the color naming results remain only tentatively explained.

Exaggeration of the Stroop interference effect was observed in patients with superior medial frontal lesions, usually bilateral but some right sided alone. These regions are not considered part of semantic networks. Both left and right superior medial frontal regions are essential for initiation, activation, and spontaneity [1,22,45,73]. They are required for and activated in generative mental tasks [66]. The involvement of bilateral superior medial frontal regions is consistent with theories about the mental process necessary for Stroop performance. These theories emphasize maintenance of the strength of the activated intention (i.e. to name colors and not read the words), a strength which exists on a gradient and can wax and wane

[10,37,44,77]. The ability to perform the incongruent condition successfully requires consistent activation of the intended response mode. This is precisely the role we postulate for the superior medial frontal region. These data also give credence to other suggestions of the importance of the superior medial frontal regions in Stroop performance [20,60].

Our finding of a right superior frontal effect for the INC condition has support in the imaging [3,50] and lesion [75] data, but not necessarily for the same region as we reported. A modest right dominance for this task would be consistent with other proposals that the right frontal region is critical for sustaining attention [39,47,61,70,79].

The region identified as associated with exaggerated Stroop effect is *superior* medial. Studies that do not differentiate among patients with medial lesions, such as infarctions in the territory of the anterior cerebral artery, will fail to demonstrate a comparable lesion effect. In the current study, 15 out of 21 individuals who had inferior medial lesions performed within normal limits on all aspects of the Stroop. This finding is consistent with the negative results on the Stroop for patients with orbitofrontal lobotomies [69], and prior assertions that the inferior medial frontal region is not involved in maintaining sustained activation of an intention, at least for cognitive tasks [66].

Our three hypotheses are partly supported. First, the Stroop test does have specificity for frontal lesions compared to posterior lesions, but not for all frontal regions. When proportional scores were used in our standard anatomical grouping analyses, there were no frontal lobe Stroop effects. Exaggeration of the Stroop interference effect does not hold for frontal lesions in general. It emerges only with specific regional lesions. The primary effect of frontal lesions (with some specificity within the frontal lobes) is overall slowing on all conditions. General slowing must be considered in the analysis of the Stroop. For instance, in normal elderly subjects, 85% of variance in the Stroop incongruent condition is due to overall processing speed [62].

Second, left lateral frontal lesions produce impaired color naming in the context of the two Stroop condi-

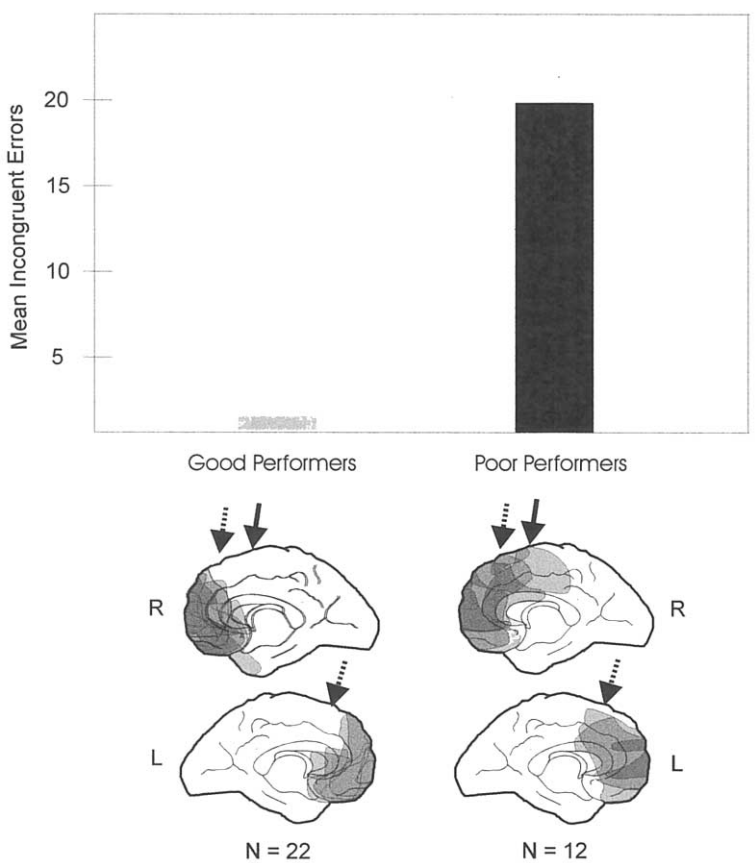

Fig. 3. Mean incongruent errors and lesion locations of performance-based (error) groups for the incongruent (INC) condition. Poor incongruent performance was associated with lesions in the regions indicated by the arrows (bilateral superior medial, $\phi = 0.317$, $P = 0.05$; right superior posteromedial, $\phi = 0.503$, $P = 0.002$). There were three missing scans in the good performer group.

tions that require rapid repeated naming. Since this specific subgroup is slow and makes more errors, the slowing is not secondary to a speed–accuracy trade-off. This effect may reflect a modest generative lexical-semantic demand of rapid repeated naming. This effect is not seen for word reading, supporting claims that word reading is more automatic, or simply perception driven, than naming [58].

Third, superior medial frontal lesions, particularly on the right, do exacerbate the interference effect. We could not demonstrate that this consequence of superomedial lesions was specifically due to ACG damage. In fact, even bilateral ACG injury did not cause difficulty. There are different possible reasons for our failure to demonstrate a critical effect of ACG lesions. The ACG is not a homogeneous structure [22], and the ACG lesions in our 14 patients may have been too heterogeneous within the ACG to isolate a key region. Davis and colleagues [19], in a microelectrode single neuron recording in the human ACG during attention demanding cognitive tasks including the Stroop, found that only 19% of the ACG neurons tested were clearly modified in one or more attention tasks. In addition to lesion location specificity, the chronicity of the lesion is important. Janer and Pardo [32] found attentional dysfunction in a cingulotomy lesion case study only in the subacute post-operative stage, with no deficit at 6 month re-examination. Our patients were tested in the chronic phase of recovery to minimize acute recovery effects.

Task context has been shown to be an important factor in demonstrating relationship of ACG to attentional tasks. These include task difficulty or demand on attentional resources [19,51], the relative frequency of presentation of incongruent and congruent stimuli [38], and the amount of response competition [2]. In our study, we used a clinical version of the Stroop, in which the three conditions were presented in a blocked format. This would minimize competition among competing responses, a context that enhances ACG functional activity [2]. Moreover, presenting all the stimuli on a single page, as opposed to individually, may change task context such that response selection is lessened. All three conditions in our study may require some degree of executive control [75], and neither activation subtractions in normal subjects nor lesion-behavior studies can distinguish the depth of control. It has also been proposed that the ACG activation in functional imaging may also just be unrelated to the actual performance of a task [74].

We have previously proposed that there are separate processes in an anterior attentional system that can be operationalized as: (1) selection of a response schema; (2) energizing (activation) of that schema; (3) inhibition of competing schemata; (4) if-then logical analysis; and (5) monitoring of performance [72]. If the ACG is essential for response selection (e.g. [5,8]), or in conditions that maximize competition among alternative responses [2], then its role in the clinical blocked condition version of the Stroop task would be minimized. The response is already given; no selection is required. What is necessary is maintenance or energizing of that given response over the many trials. The fact that the errors occur sporadically throughout the test provides some support for Kornblum et al.'s [37] theoretical proposal of a waxing and waning of activation. The medial frontal lobe may have at least two roles in the anterior attentional control system. The ACG is important for performance monitoring and response selection, and would be less relevant in the format of Stroop task we used. The superior medial prefrontal area is central to the activation or implementation of control, analogous to the role of the BA 9 region proposed by MacDonald and colleagues [43]. In task contexts such as ours, it is the frontal region downstream in the system that plays the major role. If the Stroop were implemented in a different context, perhaps as in an unblocked version, the demands for response selection should be increased, and the role of the ACG would emerge.

The critical lesion in this study population, using our version of the Stroop, is in non-cingulate, superior medial frontal lobe — the supplementary motor area bilaterally. This region has been described as the intersection or transformation point between afferent pathways, including subcortical activation pathways, particularly dopaminergic (e.g. [34]), and perceptual systems, and efferent pathways projecting bilaterally to frontal cortex and striatum. Severe damage in these regions produces abulia and bradykinesia, the ultimate expressions of loss of energizing. With less injury, or with time to recover from more severe injury, the effects of poor energizing are apparent in the Stroop. If this hypothesis is correct, one implication is that performance on the Stroop, and perhaps on real life activities with comparable cognitive demands, might improve with dopaminergic medication.

Acknowledgements

This research was funded by the Canadian Institutes of Health Research (DTS) and NINDS (Grant # 26985) to the Memory Disorders Research Center of Boston University (MPA). We are indebted to the subjects for their participation, the reviewers for very helpful comments on the discussion, and the following for patient referral, testing, statistical advice, figure and manuscript preparation: M. Binns, S. Bisschop, L. Buckle, R. Dempster, D. Franchi, L. Hamer, D. Izukawa, R. McDonald, P. Mathews, S. Patterson, and A. Savas.

Appendix A

Mean and standard deviation of stroop RT, difference, and proportion measures by lesion group

Group[a]	RT (s)						Difference (s)						Proportion					
	Word (W)		Color (C)		Incongruent (I)		C−W		I−C		I−W		(C−W)/W		(I−C)/C		(I−W)/W	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
RF	61.1	13.8	81.3	16.1	164.8	35.5	20.1	14.4	83.5	27.3	103.6	35.4	0.4	0.3	1.1	0.3	1.8	1.0
LF	73.8	32.7	117.1	44.8	213.6	103.0	43.4	32.8	96.5	84.6	139.9	105.9	0.7	0.5	0.9	0.6	2.2	1.6
BF	53.4	74.1	74.1	16.4	149.3	51.8	20.7	10.3	75.1	41.9	95.9	40.8	0.4	0.3	1.0	0.5	1.8	0.5
RNF	42.3	59.9	59.9	13.3	108.3	18.7	17.6	6.2	48.4	7.7	66.0	12.5	0.4	0.8	0.8	0.2	1.6	0.2
LNF	48.9	71.0	71.0	15.6	135.0	30.6	22.1	6.6	64.0	19.8	86.1	22.4	0.5	0.1	0.9	0.3	1.8	0.4
CTL	41.3	59.3	59.3	11.8	111.7	27.6	18.0	9.0	52.4	20.9	70.4	23.1	0.5	0.2	0.9	0.3	1.7	0.5

[a]RF, right frontal; LF, left frontal; BF, bifrontal; RNF, right non-frontal; LNF, left non-frontal; CTL, matched control.

References

[1] Alexander MP, Benson DF, Stuss DT. Frontal lobes and language. Brain and Language 1989;37:656–91.

[2] Barch DM, Braver TS, Sabb FW, Noll DC. Anterior cingulate and the monitoring of response conflict: evidence from an fMRI study of overt verb generation. Journal of Cognitive Neuroscience 2000;12:298–309.

[3] Bench CJ, Frith CD, Grasby PM, Friston KJ, Paulesu E, Frackowiak RSJ, Dolan RJ. Investigations of the functional anatomy of attention using the Stroop test. Neuropsychologia 1993;31:907–22.

[4] Benson DF, Ardila A. Aphasia. New York: Oxford University Press, 1996.

[5] Botvinick M, Nystrom LE, Fissell K, Carter CS, Cohen JD. Conflict monitoring versus selection-for-action in anterior cingulate cortex. Nature 1999;402:179–81.

[6] Burgess PW, Shallice T. Response suppression, initiation and strategy use following frontal lobe lesions. Neuropsychologia 1996;43:263–72.

[7] Cabeza R, Grady CL, Nyberg L, McIntosh AR, Tulving E, Kapur S, Jennings JM, Houle S, Craik FI. Age-related differences in neural activity during memory encoding and retrieval: a positron emission tomography study. Journal of Neuroscience 1997;17:391–400.

[8] Carter CS, Braner TS, Barch DM, Botvinick MM, Noll D, Cohen JS. Anterior cingulate cortex, error detection and the online monitoring of performance. Science 1998;280:747–9.

[9] Cohen J, Cohen P. Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences, 2nd. Hillsdale, NJ: Lawrence Erlbaum Associates, 1983.

[10] Cohen JD, McClelland JL, Dunbar K. On the control of automatic processes: a parallel distributed processing account of the Stroop effect. Psychological Review 1990;97:332–61.

[11] Cohen JD, Servan-Schreiber D. Context, cortex and dopamine: a connectionist approach to behavior and biology in schizophrenia. Psychological Review 1992;99:45–77.

[12] Cohen JD, Usher M, McClelland JL. A PDP approach to set size effects within the Stroop task: reply to Kanne, Balota, Spieler, and Faust, 1998. Psychological Review 1998;105:188–94.

[13] Comalli PE, Wapner S, Werner H. Interference effects of Stroop colour–word test in childhood, adulthood, and aging. Journal of General Psychology 1962;100:47–53.

[14] Corbetta M, Miezin FM, Dobmeyer S, Shulman GL, Petersen SE. Attentional modulation of neural processing of shape, colour, and velocity in humans. Science 1990;248:1556–9.

[15] Corcoran R, Upton D. A role for the hippocampus in card sorting? Cortex 1993;29:293–304.

[16] Damasio AR, Damasio H. The anatomic basis of pure alexia. Neurology 1983;33:1573–83.

[17] Damasio AR, McKee J, Damasio H. Determinants of color anomia. Brain and Language 1979;7:74–85.

[18] David AS. Stroop effects within and between the cerebral hemispheres: studies in normals and acallosals. Neuropsychologia 1992;30:161–75.

[19] Davis KD, Hutchison WD, Lozano AM, Tasker RR, Dostrovsky JO. Human anterior cingulate cortex neurons modulated by attention-demanding tasks. Journal of Neurophysiology 2000;83:3575–7.

[20] Dehaene S, Posner MI, Tucker DM. Localization of a neural system for error detection and compensation. Psychological Science 1994;5:303–5.

[21] D'Esposito M, Detre JA, Alsop DC, Shin RK, Atlas S, Grossman M. The neural basis of the central executive system of working memory. Nature 1995;378:279–81.

[22] Devinsky O, Morrell M, Vogt BA. Contributions of anterior cingulate cortex to behavior. Brain 1995;118:279–306.

[23] Dulaney CL, Rogers WA. Mechanisms underlying reduction in Stroop interference with practice for young and old adults. Journal of Experimental Psychology: Learning, Memory and Cognition 1994;20:470–84.

[24] Dunbar K, MacLeod CM. A horse of a different colour: Stroop interference patterns with transformed words. Journal of Experimental Psychology: Human Perception and Performance 1984;10:622–39.

[25] Fletcher PC, Frith CD, Grasby PM, Shallice T, Frackowiak RSJ, Dolan RJ. Brain systems for encoding and retrieval of auditory–verbal memory: an in-vivo study in humans. Brain 1995;118:401–16.

[26] Frith CD, Friston K, Liddle PF, Frackowiak RS. Willed action and the prefrontal cortex in man: a study with PET. Proceedings of the Royal Society of London 1991;244:241–6.

[27] Fuster JM. The Prefrontal Cortex: Anatomy, Physiology, and Neuropsychology of the Frontal Lobes. New York: Lippincott-Raven, 1997.

[28] Gardner R, Holzman PS, Klein GS, Linton H, Spence DP. Cognitive control: a study of individual differences in cognitive behavior. Psychological Issues 1959;1:1–185.

[29] Golden CJ. Identification of brain disorders by the Stroop color–word test. Journal of Clinical Psychology 1976;32:654–8.

[30] Golden CJ, Osmon DC, Moses J, Berg RA. Interpretation of the Halstead-Reitan Neuropsychological Test Battery: A Casebook Approach. New York: Grune & Stratton, 1981.

[31] Holzt P, Vilkki J. Effect of frontomedial lesions on performance on the Stroop test and word fluency tests. Journal of Clinical and Experimental Neuropsychology 1988;10:79–80.
[32] Janer KW, Pardo JV. Deficits in selective attention following bilateral anterior cingulotomy. Journal of Cognitive Neuroscience 1991;3:231–41.
[33] Kaplan EF, Goodglass H, Weintraub S. The Boston Naming Test, 2nd. Philadelphia: Lea and Febiger, 1983.
[34] Kimberg DY, D'Esposito M, Farah MJ. Effects of bromocriptine on human subjects depend on working memory capacity. NeuroReport 1997;8:3581–5.
[35] Kimberg DY, Farah MJ. A unified account of cognitive impairments following frontal lobe damage: the role of working memory in complex organized behavior. Journal of Experimental Psychology: General 1993;122:411–28.
[36] Klein M, Pondo R, Haux PJ, Jolles J. Effect of test duration on age-related differences in Stroop interference. Journal of Clinical and Experimental Neuropsychology 1997;11:421–7.
[37] Kornblum S, Stevens GT, Whipple A, Requin J. The effects of irrelevant stimuli I: the time course of stimulus–stimulus and stimulus–response consistency effects with stroop-like stimuli, Simon-like tasks, and their factorial combinations. Journal of Experimental Psychology: Human Perception and Performance 1999;25:688–714.
[38] Leung HC, Skudlarski P, Gatenby JC, Peterson BS, Gore JC. An event-related functional MRI study of the Stroop colour word interference task. Cerebral Cortex 2000;10:552–60.
[39] Levine B, Stuss DT, Milberg WP, Alexander MP, Schwartz M, Macdonald R. The effects of focal and diffuse brain damage on strategy application: evidence from focal lesions, traumatic brain injury, and normal aging. Journal of the International Neuropsychological Society 1998;4:247–64.
[40] Lezak M. Neuropsychological Assessment, 3rd. New York: Oxford University Press, 1995.
[41] Lindsay DS, Jacoby LL. Stroop process dissociations: the relationship between facilitation and interference. Journal of Experimental Psychology: Human Perception and Performance 1994;20:219–34.
[42] Lueck CJ, Zeki S, Friston KJ, Deiber MP, Cope P, Cunningham VJ, Lammertsma AA, Kennard C, Frackowiak RS. The color center in the cerebral cortex in man. Nature 1989;340:386–9.
[43] MacDonald AW, Cohen JD, Stenger VA, Carter CS. Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. Science 2000;288:1835–8.
[44] MacLeod CM. Half a century of research on the Stroop effect: an integrative review. Psychological Bulletin 1991;109:163–203.
[45] McLean A, Temkin AR, Dikmen S, Wyler AR. The behavioral sequelae of head injury. Journal of Clinical Neuropsychology 1983;5:361–76.
[46] Mesulam M-M. Large-scale neurocognitive networks and distributed processing for attention, language, and memory. Annals of Neurology 1990;28:597–613.
[47] Milner B, Petrides M. Behavioral effects of frontal-lobe lesions in man. Trends in Neuroscience 1984;7:403–7.
[48] Murtha S, Chertkow H, Beauregard M, Dixon R, Evans A. Anticipation causes increased blood flow to the anterior cingulate cortex. Human Brain Mapping 1996;4:103–12.
[49] Nelson HE, O'Connell A. Dementia: the estimation of premorbid intelligence levels using the New Adult Reading Test. Cortex 1978;14:234–44.
[50] Pardo JV, Pardo PJ, Janer KW, Raichle ME. The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. Proceedings of the National Academy of Sciences 1990;87:256–9.
[51] Paus T, Koski L, Caramanos Z, Westbury C. Regional differences in the effects of task difficulty and motor output on blood flow response in the human anterior cingulate cortex: a review of 107 PET activation studies. NeuroReport 1998;9:37–47.
[52] Paus T, Petrides M, Evans AC, Meyer E. Role of the human anterior cingulate cortex in the control of oculomotor, manual and speech responses: a positron emission tomography study. Journal of Neurophysiology 1993;70:453–69.
[53] Perret E. The left frontal lobe of man and the suppression of habitual responses in verbal categorical behavior. Neuropsychology 1974;12:323–30.
[54] Petersen SE, Corbetta M, Miezin FM, Dobmeyer M. Selective attention modulates usual processing of form, colour, velocity: III. Areas related to higher-order selective processes. Society for Neuroscience Abstracts 1989; p. 624.
[55] Petrides M, Pandya DM. Comparative architectonic analysis of the human and macaque frontal cortex. In: Boller F, Grafman J, editors. Handbook of Neuropsychology, vol. 9. Amsterdam: Elsevier, 1994:17–57.
[56] Ponsford JL. Attention. In: Groth-Marsnat G, editor. Neuropsychological Assessment in Clinical Practice. New York: John Wiley & Sons, 2000:355–400.
[57] Posner MI. Structures and functions of selective attention. In: Boll T, Bryant BK, editors. Clinical Neuropsychology and Brain Function: Research, Measurement, and Practice. Washington: American Psychological Association, 1988:171–202.
[58] Price CJ, Friston KJ. Cognitive conjunction: a new approach to brain activation experiments. Neuroimage 1997;5:261–70.
[59] Rand G, Wapner S, Werner H, McFarland JH. Age differences in performance on the Stroop color–word test. Journal of Personality 1963;31:534–58.
[60] Richer F, Decary A, Lapierre M, Rouleau I, Bouvier G, Saint-Hilaire J. Target detection deficits in frontal lobectomy. Brain and Cognition 1993;21:203–11.
[61] Rueckert L, Grafman J. Sustained attention deficits in patients with right frontal lesions. Neuropsychologia 1996;34:953–63.
[62] Salthouse TA, Meinz EJ. Aging, inhibition, working memory, and speed. Journal of Gerontology 1995;50:297–306.
[63] Smith GJW. Comparisons between adaptive patterns in two serial experiments. Acta Psychologica 1959;16:302–15.
[64] Spieler DH, Balota DA, Faust ME. Stroop performance in healthy younger and older adults and in individuals with dementia of the Alzheimer's type. Journal of Experimental Psychology: Human Perception and Performance 1996;22:461–79.
[65] Stroop JR. Studies of interference in serial verbal reactions. Journal of Experimental Psychology 1935;18:643–62.
[66] Stuss DT, Alexander MP, Hamer L, Palumbo C, Dempster R, Binns M, Levine B, Izukawa D. The effects of focal anterior and posterior brain lesions on verbal fluency. Journal of the International Neuropsychological Society 1998;4:265–78.
[67] Stuss DT, Alexander MP, Palumbo CL, Buckle L, Sayer L, Pogue J. Organizational strategies with unilateral or bilateral frontal lobe injury in word learning tasks. Neuropsychology 1994;8:355–73.
[68] Stuss DT, Benson DF. The Frontal Lobes. New York: Raven Press, 1986.
[69] Stuss DT, Benson DF, Kaplan EF, Weir WS, Della Malva C. Leucotomized and non-leucotomized schizophrenics: comparison on tests of attention. Biological Psychiatry 1981;16:1085–100.
[70] Stuss DT, Eskes GA, Foster JK. Experimental neuropsychological studies of frontal lobe functions. In: Boller F, Grafman J, editors. Handbook of Neuropsychology, vol. 9. Amsterdam: Elsevier, 1994:149–85.
[71] Stuss DT, Levine B, Alexander MP, Hong J, Palumbo C, Hamer L, Murphy KJ, Izukawa D. Wisconsin Card Sorting Test performance in patients with focal frontal and posterior brain damage: effects of lesion location and test structure on separable cognitive processes. Neuropsychologia 2000;38:388–402.

[72] Stuss DT, Shallice T, Alexander MP, Picton TW. A multidisciplinary approach to anterior attentional functions. Annals of the New York Academy of Science 1995;769:191–211.
[73] Stuss DT, van Reekum R, Murphy KJ. Differentiation of states and causes of apathy. In: Borod J, (editor). The Neuropsychology of Emotion, New York:Oxford University Press, in press.
[74] Taylor SF, Kornblum S, Lauber EJ, Minoshima S, Koeppe RA. Isolation of specific interference processing in the Stroop task: PET activation studies. Neuroimage 1997;6:81–92.
[75] Vendrell P, Junque C, Pujol J, Jurado MA, Molet J, Grafman J. The role of prefrontal regions in the Stroop task. Neuropsychologia 1995;33:341–52.
[76] West R. Age differences in lapses of intention in the Stroop task. Journal of Gerontology 1999;54:1–10.
[77] West R, Baylis GC. Effects of increased response dominance and contextual disintegration on the Stroop interference effect in older adults. Psychology and Aging 1998;13:206–17.
[78] West R, Bell MA. Stroop colour–word interference and electroencephalogram activation: evidence for age-related decline of the anterior attention system. Neuropsychology 1997; 11:421–7.
[79] Wilkins AJ, Shallice T, McCarthy R. Frontal lesions and sustained attention. Neuropsychologia 1987;25:359–65.
[80] Zhang H, Kornblum S. The effects of stimulus-response mapping and irrelevant stimulus–stimulus overlap in four-choice Stroop tasks with single-carrier stimuli. Journal of Experimental Psychology: Human Perception and Performance 1998;24:3–19.