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Specific impairments of rule induction in different frontal lobe subgroups☆

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Abstract

The neural correlates of inductive reasoning are still poorly understood. In order to explore them, we administered a revised version of the Brixton test [Cortex 32 (2) (1996a) 241], a rule attainment task, to a group of 40 patients with a focal frontal brain lesion of mixed aetiology and to 43 control subjects. To interpret an impairment on the test as suggesting an inductive reasoning deficit a number of alternative hypotheses need first to be considered, namely whether the Brixton impairment could be explained by: (i) a working memory deficit; (ii) a monitoring deficit; (iii) a difficulty in applying an already induced rule; (iv) greater impulsivity. The patients with left lateral (LL) frontal lesions were significantly impaired on the Brixton test; more importantly they were the only group in which none of the alternative hypotheses we explored proved able to explain the flawed performance. In sharp contrast, right lateral lesion patients did not make significantly more errors on the Brixton test than controls, but they produced three times more capture errors (a sign of impaired monitoring processes). The results were interpreted as suggesting functional dissociations between inductive reasoning, monitoring and working memory and a localisation of key processes for induction in left lateral frontal cortex and in right lateral cortex for monitoring and checking.

Keywords: Inductive reasoning; Frontal lobe; Working memory; Monitoring and checking; Executive functions

1. Introduction

Reasoning is the activity of generating and evaluating arguments. Theories of reasoning distinguish, on the basis of the relationship that holds between premises and conclusions, two main kinds of inference: induction and deduction. An inference is a deduction if the conclusion must be true whenever all the premises are true. Consider, for example, the premises "All men are mortal" and "Socrates is a man" and the conclusion "Socrates is mortal". As the latter statement must be true whenever both the former ones are, the inference at hand is a (valid) deduction; by contrast, the premises "Socrates is mortal" and "Socrates is a man" do not entail "All men are mortal"; in the latter case the premises provide only limited grounds for accepting the conclusion: these kinds of inferences are called inductions (Rips, 1999). Induction can also be defined as any process of thought yielding a conclusion which increases the semantic information contained in its premises (Johnson-Laird, 1993).

Recently, there has been a growing interest in elucidating the neuroanatomy of the inductive reasoning processes. A series of imaging studies have been devoted to this issue (Duncan et al., 2000; Goel & Dolan, 2000; Goel, Gold, Kapur, & Houle, 1997; Osherson et al., 1998; Parsons & Osherson,

☆ The study was carried out in the Santa Maria della Misericordia Hospital (Udine, Italy) and in SISSA (Trieste, Italy).

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2001; Strange, Henson, Friston, & Dolan, 2001). In most of these, the frontal lobes, particularly their lateral aspects, are activated while the participants are carrying out inductive inferences; but they are often part of a large network of areas.

Many putative processes may be involved in the carrying out of the cognitive operations necessary when any real-life induction occurs. Information must be comprehended and held in working memory; checking may or may not occur. All of these may involve multiple subprocesses. The determination of what the relevant subprocesses are and where they are localised is a complex task, as in our current state of knowledge processes that are not yet tightly definable or operationalisable may be relevant. In addition, the necessary complexity of inductive reasoning tasks often makes it hard to carry out a task analysis. Since, it is difficult to ensure that the processing of all stages *other* than any hypothetical critical one remain constant across conditions, factorial designs are, in general, difficult to apply using the imaging methodology (but see Duncan et al., 2000).

To determine whether processes necessarily involve a particular region, neuropsychological studies (as other inactivation techniques such as TMS) can provide an important source of evidence (D'Esposito & Postle, 1999; Goel, in press; Price, Mummery, Moore, Frakowiak, & Friston, 1999).

Moreover, with tasks requiring a considerable number of high-level processes, the results of lesion studies are generally easier to interpret *functionally* as it is not necessary to characterise in detail processes that are unimpaired. Moreover one has (additional) evidence on function from, for instance, the nature of errors as well as from observed dissociations. This provides a second reason for also using the lesion methodology.

A number of tasks, used in lesion studies, have an inductive component. For instance fluid intelligence tests, such as the Raven test (Basso, Capitani, Luzzatti, & Spinnler, 1981; Gainotti, D'Erme, Villa, & Caltagirone, 1986) or the culture fair intelligence test (Duncan, Burgess, & Emslie, 1995) have a major inductive component (Carpenter, Just, & Shell, 1990). However, the structure of the tests makes it difficult to isolate the inductive component neuropsychologically and the variety of item types does not allow a quantitative error analysis to be simply carried out. Theoretically, it would be necessary, in order to assess the inductive component, to administer, along with a fluid intelligence tests, other tests that can assure one of the integrity of the other components. Duncan et al. (1995) indeed used a related procedure. However, as only three subjects were tested, the number of patients studied was too few to strongly sustain any localisation claim. Concept attainment tasks, such as the Weigl test, the Wisconsin card sorting test (WCST) (Drewe, 1974; Milner, 1964; Stuss et al., 2000) and the Brixton spatial rule attainment test (Burgess & Shallice, 1996a) also have an inductive component. The Wisconsin card sorting test is the best known clinical signature of frontal lobe dysfunction. The "discovery" part of the test may indeed stress the inductive competence of cognitively impaired subjects. However, in order to attain normal performance, other abilities are involved, in particular attentional switching, monitoring and sensitivity to negative feedback. In fact, there is some evidence that suggests that the failure observed in frontal patients may arise from impairments to processes other than the inductive one. Thus, perseverative errors, the more distinctive feature of the difference between the performance of frontal patients and that of controls, suggests—although it does not necessitate—a central role for a switching deficit. Moreover, even if frontal patients are told which the relevant criteria of classification are, they can still have pathological performance (Stuss et al., 2000). This relates to the clinical observation that patients often verbalize the three sorting criteria but are unable to use this knowledge effectively (Stuss et al., 2000).

A recently proposed rule attainment task, the Brixton test (Burgess & Shallice, 1996a), is likely to be better suited as a measure of inductive competence in patients. In this test, the participant is presented with a card containing a 2×5 display of circles of which one only is filled. The participant must predict where the circles would be completed on the next card. Nine simple rules are used each of which is in operation between three to eight trials. In the Brixton test, the rules which have to be attained pertain to the relation among succeeding stimuli. Thus, the inductive process will be more stressed than on the WCST where the rules directly relate to perceptual features on single cards. In addition, the stimuli will be less prone to automatically trigger overlearned stimulus–response associations and so are less liable to induce perseverative behaviour (see Burgess & Shallice, 1996a). This means that a possible deficit in induction will be less contaminated by other factors. Finally, the variety of different rules used allows a richer error analysis. In the study of Burgess and Shallice (1996), frontal patients as a group both showed a pathological level of performance on the Brixton test, but did not produce a significantly larger number of perseverative errors. Posterior patients, by contrast, performed at a similar level to controls on both measures. This result suggests that the frontal lobes could have a crucial role in inductive reasoning. However, a number of alternative possibilities need to be considered for the pattern of results shown by the patients with frontal lesions:

(i) A low score on the Brixton test could be due to a working memory deficit (Baddeley, 1997). To carry out any kind of inference, in fact, it is necessary to be able to hold the relevant information in mind. Moreover, it is generally acknowledged that some components of the system underlying working memory performance rely on frontal lobe networks and particularly their ventrolateral and dorsolateral aspects (D'Esposito & Postle, 1999; Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Paulesu, Frith, & Frackowiak, 1993; Petrides, 2000). Thus, it cannot be excluded that the deficit on the Brixton test observed in frontal patients could be generated by a more basic impairment in holding information online. This possibility could not be ruled out given the condi-

tions used in the original study of Burgess and Shallice (1996).

(ii) In WCST, Stuss et al. (2000) have attributed loss of set errors and perseverative errors in patients with right frontal lesions to a problem of sustained attention or monitoring. An impairment of monitoring and checking could also affect the performance of the Brixton test. Checking and monitoring processes (Burgess & Shallice, 1996b) may also be necessary to satisfactorily evaluate and verify putative newly generated rules or schemas in Shallice & Burgess's (1996) terminology. Recently, Henson and his collaborators have proposed that this process can be localised in the right dorsolateral prefrontal cortex. Their primary evidence was derived from fMRI studies using episodic memory paradigms (Henson, Shallice, & Dolan, 1999; Henson, Shallice, Josephs, & Dolan, 2002 but also see Fletcher & Henson, 2001; Shallice, 2002 for reviews and Petrides, 2000 for an alternative approach to monitoring). For instance, Fletcher, Shallice, Frith, Frackowiak, and Dolan (1998) found that retrieval of a long list of items where the subject needs to monitor his or her output for repeats (Stuss et al., 1994) activates right dorsolateral prefrontal cortex much more than does the equivalent amount of retrieval of one-off paired associates. Can one, however, obtain direct neuropsychological evidence, as these imaging studies suggest, that monitoring or checking processes are relatively lateralised in the frontal cortex?

(iii) Patients could fail on the Brixton test because of an inability to apply a rule they had already induced. For instance Strange et al. (2001), in their efMRI study on explicit abstract rule induction, suggested that the left dorsolateral frontal cortex is necessary for rule application per se.

(iv) Finally, a greater impulsivity leading to excessively rapid responding (Burgess & Shallice, 1996a; Miller, 1985, 1992; Miller & Milner, 1985) could be responsible, in some patients, for the higher number of errors.

Our aims in the current study were three-fold. First, it was to obtain a more precise localisation of regions within prefrontal cortex which give rise to impaired Brixton performance. Secondly, it was to examine whether any such impairment could be explained as a result of malfunctioning of any of the non-induction processes discussed above used in the carrying out of the task. If they are intact, it makes it more plausible to attribute any impairment on the task to malfunctioning of more basic processes involved in induction. Thirdly, we wished to examine using a paradigm different from the imaging paradigms involving episodic memory whether there was any evidence for differential lateralisation of organisational processes on the one hand from checking ones on the other.

As far as localisation is concerned, a procedure developed by Stuss, Alexander and their co-workes has been applied which allows one to produce a somewhat finer localisation of prefrontal impairment than that of simply compare unilateral left and right frontal patients (e.g. Stuss et al., 1998, 2000). It does so by creating groups that are reasonably coherent and sizeable given the natural history of lesions affecting the frontal cortex, while at the same time making parts which plausibly relate to functional divisions (i.e. lateral versus medial). Following Stuss and collaborators, four subgroups were used: left (LL) and right (RL) lateral, superior (SM) and inferior (IM) medial. However, because of some localisation claims related to induction (Strange et al., 2001) and monitoring (Carter et al., 2000), in a subsidiary analysis we also checked the possible effects, only on these two functions, of fronto-polar (FP) or anterior cingulated (AC) damage, respectively.

We examined the additional processes discussed above that might be involved in performing the Brixton test by using a variety of procedures. First, to carry out the induction of a rule the subject must actively hold information on a sufficient number of cards in mind, as the information will not be stored automatically in a phonological or visuo-spatial buffer (Mitchell, 1972; Phillips & Christie, 1977). An additional working memory task using similar material was designed; it involved one card more than the maximum number necessary in order to disambiguate the rules used. Secondly, an extended error analysis was used to examine whether perseveration was a particular problem. Third, the collection of response times enabled us to consider impulsivity. Finally, the issue of checking and monitoring was addressed by using a second version of the Brixton where interfering rules are potentiated and, for each correct rule, the subject has to avoid making a capture error, which would occur if they obey the interfering rule rather than the previously acquired one. This created the neuropsychological analogue of a situation occurring in a study involving imaging of episodic memory (Henson et al., 1999) which had produced a right dorsolateral activation. Participants had to decide of an item recognised as familiar whether it occurred at precisely the same spatial position and list as when presented. It therefore allowed for possible differential lateralisation of monitoring and checking processes to occur.

2. Material and methods

2.1. Participants

Forty patients with a single focal brain lesion as determined by a CT or an MRI scan were recruited from the Neurological and Neurosurgical ward of Ospedale Civile in Udine (Italy); all patients gave their consent to participate in the study. The aetiology was mixed: stroke, traumatic brain injury and neoplasm (Table 1). Exclusion criteria were: the presence in the clinical history of psychiatric disorders, substance abuse or previous neurological disease, neuroradiological evidence of diffuse brain damage, and age lower than 18 or higher than 70. We also considered for single case anal-

Table 1
Aetiology for each lesion group

	LL	RL	IM	SM	Overall
Meningioma	2	3	8	4	17
Glioma low grade	1	2		1	4
Glioma high grade	2	1	1	1	5
Metastases	1		1		2
Histiocytosis		1			1
Lymphoma		1			1
Stroke	4	2		3	9
TBI			1		1

Absolute frequencies of patients included in the study. IM: inferior medial frontal; SM: superior medial frontal; LL: left lateral frontal; RL: right lateral frontal; TBI: traumatic brain injury.

yses (see below in methods) four more patients with neuroradiological evidence of multiple brain lesions limited to the frontal lobes. The time since the lesion ranged between 7 and 495 days; this did not significantly differ between the lesion subgroups [Kruskal–Wallis test, χ^2 (3) = 0.825, $P > 0.1$] (Table 2). The starting point considered in the case of neoplasm is the day of surgery. Forty-three normal control volunteers also participated in the study; they were recruited from slipped disc patients at the same Udine hospital and from patients' relatives. The controls were matched with the patients for age and educational level. There were no significant differences either between the frontal patients overall and the controls for age [F (1, 81) = 2.08, $P > 0.1$], education [F (1, 81) = 0.003, $P > 0.1$] and sex [F (1, 81) = 0.543, $P > 0.1$] or between the frontal subgroups considered separately and the controls for age [F (4, 78) = 1.403, $P > 0.1$], education [F (4, 78) = 1.555, $P > 0.1$] and sex [F (4, 78) = 0.455, $P > 0.1$].

2.2. Neuroradiological assessment

For all patients but one, we obtained at least a CT or an MRI scan. The patients were assigned to four anatomically defined subgroups depending on their lesion site, following the procedure of Stuss et al. (1998): inferior medial region (IM), in which the lesion involves the orbital surface and/or the inferior medial surface of one or both lobes; superior medial (SM) in which the superior part of the medial cortex of one or both lobes is damaged, in SM patients the orbitofrontal cortex is always spared; left lateral (LL) and right lateral (RL), which have unilateral damage of the frontal lobe convexity (Fig. 1). In order to classify lesions, the scans were evaluated by two senior neuroradiologists blind to the experimental results. The neuroradiologists were provided with the template of the Stuss et al.'s (1998) paper, Fig. 3. We also checked, for each patient, the anterior cingulate (AC) and the frontal pole (FP) involvement (in this latter case only the neuroradiologist was provided with another template, namely the one in Stuss & Levine, 2002, Fig. 1). Lesion size was estimated using the "Curry" software by NeuroScan version 4.5, for the 27 patients for whom a digitalized version of the scans was available. Lesions boundaries were traced by a senior neuroradiologist blind to the experimental findings. Finally, since in some cases, we administered tests 7 days after surgery, we also examined the possible effect of oedema using the patients' scans nearest to the testing session date when more than one scan was available. We assigned each patient to one grade of a four level scale:

0 absence of oedema;
1 mild oedema involving <20% of one lobe;

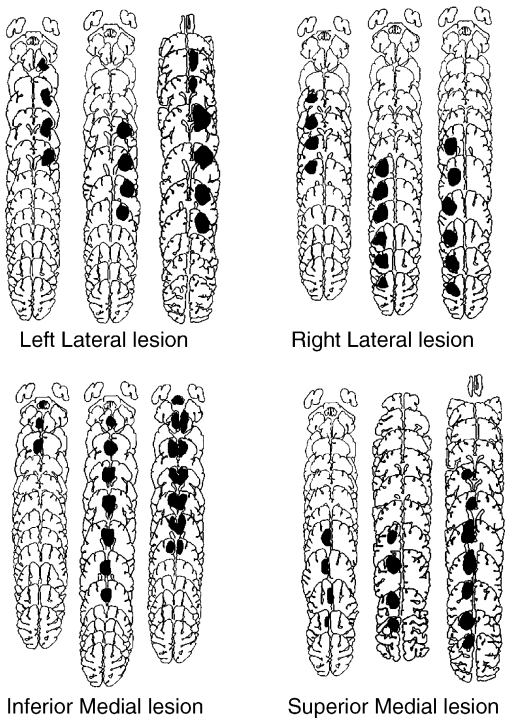

Fig. 1. Lesion templates of three subjects within each of the four patient groups. The three patients were selected from among the others of the same lesion group on the basis of lesion size: small (25th percentile), medium (50th), large (75th).

Table 2
Demographic variables for each lesion group and for control subjects

	LL	RL	IM	SM	Overall	CTL
N	10	10	11	9	40	43
Age [mean (S.D.)]	55 (13)	46 (16)	51 (14)	55 (13)	52 (15)	48 (10)
Education [mean (S.D.)]	10.9 (4.0)	8.2 (4.1)	7.8 (2.5)	10.3 (3.4)	9.4 (3.7)	9.2 (3.24)
Gender [% female]	50	60	36	56	49	42
Days since lesion [median (range)]	42.5 (7–332)	27.5 (9–325)	27 (7–321)	33 (7–495)	31 (7–495)	
Oedema index [mean (S.D.)]	1.40 (1.17)	1.63 (1.30)	2.09 (0.83)	1.13 (0.99)	1.59 (1.09)	

Subgroups are defined in the caption of Table 1.

Table 3A
FirstHalf Brixton cards

Cards	Rule	*N* cards in the rule
2 3 4 5 6	+1	5
5 4 3 2 1 10	−1	6
6 10 6 10 6 10 6	Alternate	7
1 7 2 8 3 9 4	Top down	7
3 2 1 10 9	−1	5
10 10 10 10 10 10	Stay the same	6
9 10 9 10 9 10 9	Alternate	7

The numbers in the first column refers to the position of the circle in the 2 row × 5 column array.

2 moderate oedema involving >20% and <50% of one lobe;
3 severe oedema involving >50% of one lobe.

Patients with time since lesion greater than 60 days were all attributed to grade 0.

2.3. Materials

We devised a new version of the Brixton test (Burgess & Shallice, 1996a). In the FirstHalf, 43 cards were presented, one at time, on a touch screen monitor. Each card contains a 2 × 5 array of numbered circles (1–5 first raw, left to right; 6–10 s raw, left to right); one only being blue, the rest being white. The blue circle moves from one card to the next following seven rules of five different kinds. On average, a rule changes after six cards (range 5–7), without any explicit warning (Table 3A). The participant's task is to touch the circle where s/he thinks the blue circle will be on the card following the one currently presented. Participants are told that the coloured circle never moves randomly and that rules change without warning. An example of a series of answers scored as correct is, for the first 10 cards (Table 3A), the following: ignored-4-5-6-7-4-3-2-1-10. Note that for all rules we counted a prediction about the last card to which it applied as correct if it followed the rule in force, even though the next card actually appeared elsewhere. For example, the response considered correct for the last card of the first rule (Table 3A) is "7" even if the cards that actually follows has the blue circle in position 5. In the SecondHalf, 56 blue circle cards were presented with the blue circle following seven rules of five different kinds, one rule being active for, on average, eight cards (range 6–10). An interference procedure occurs once for each rule, one to three cards before the end of the "blue" series; this allows most of the participants to acquire the rule before the interference begins. It consists of a sequence of four cards similar to that in the first part except that they contain a *red*-filled circle instead of a *blue*-filled one. These four cards always follow a rule which is different from that of the blue ones which immediately precede or follow them (Table 3B). The succession of the interfering red cards is arranged so that the position of the first blue card which follows the red ones fit with both the rules obeyed by the blue cards preceding the interference and that of the red interfering cards. This allows a theoretically interesting error type to occur, namely capture errors (see below). Participants are clearly instructed and given an example that: (i) the red cards have nothing to do with the blue ones; (ii) after the red cards the blue circle will always continue to follow the *same rule as before* the interruption; (iii) with the blue cards the task is identical to that in the first part: they have to predict the position of the blue circle on the card following the one currently presented; (iv) with red cards they simply have to touch the red-filled circle, which remains on the screen until they touch it (i.e. the card does not "turn" if they touch another circle). An example of a string of responses scored as correct is, for the first 15 cards (Table 3B), the following: ignored-2-1-10-9-5-6-7-8-8-7-8-3-8-3. In this case, answering with position 10 instead of 8 on the first blue card after the interference would have been scored as a capture error (see also below in the "variable section").

A test to assess participants' ability to process the working memory requirements of the Brixton test was also given. The same type of "red" and "blue" cards are used as far the Brixton test. Three cards with a randomly positioned blue circle are shown to participants one at time (any of the rules used can be induced from three cards). Four cards with a red coloured circle, which the participants must touch, follow; as in the Brixton test. Finally they must state the positions of the three blue-filled circles. Ten trials were administrated to each participant. The dependent variable is the proportion of correct responses. Standard Raven matrices (series A1 A2 A3 B C D) were also administered.

Table 3B
SecondHalf Brixton cards

Cards	Rule	*N* cards in the rule (+ *n* after the I)	Interference cards (following normal cards)	Interference rule
4 3 2 1 10 (I) 9 8	−1	5 (+2)	I5 I6 I7 I8 (9 8)	+1
3 8 3 8 3 8 3 (I) 8 3 8	Alternate (3–8)	7 (+3)	I2 I1 I10 I9 (8 3 8)	−1
9 10 1 2 3 4 (I) 5 6	+1	6 (+2)	I5 I8 I5 I8 (5 6)	Alt (5–8)
5 4 3 2 1 (I) 10	−1	5 (+1)	I6 I7 I8 I9 (10)	+1
5 9 4 8 3 7 2 (I) 6	Top down	7 (+1)	I6 I6 I6 I6 (6)	Stay (6)
7 6 7 6 7 6 7 (I) 6 7	Alternate (6–7)	7 (+2)	I10 I9 I8 I7 (6 7)	−1
7 7 7 7 7 7 (I) 7 7	Stay (7)	6 (+2)	I7 I2 I7 I2 (7 7)	Alt (2–7)

The numbers in the first and forth column refers to the position of the circle in the 2 row × 5 column array respectively of the standard "blue cards" and of the interference "red cards". I: interference.

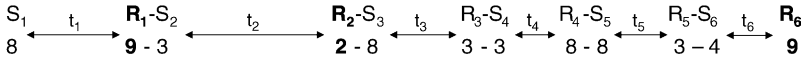

Fig. 2. Brixton FirstHalf: schema of stimuli presentation. S: Stimulus; R: Response; t: time. Wrong responses in bold.

2.4. Variables

2.4.1. Brixton FirstHalf

(i) Proportion of correct responses in the first part (hereafter "FirstHalf score"), the one without interference.

(ii) Perseveration of the response (PRe): an incorrect response which is the same as the immediately preceding one (e.g. incorrect response *n*: 5, incorrect response *n* + 1: again 5).

(iii) Perseveration of the preceding rule (PPRu): an incorrect response in which the rule that precede the currently active one is applied. Since, when the correct rule has not been attained, each Brixton rule has its own rate of utilisation as an "attempt" for each participant (e.g. some could tend to use more often "+1", others "−1" as the first try), to measure this kind of error appropriately it is needed to estimate how the baseline rate of production of a particular rule *n* is modified by the fact that the rule *n* was the last active one. The index used is an odds ratio between the average probability of rule *n* given the preceding one was *n* and the average probability of rule *n* given preceding one was not *n*.

(iv) Same rule (SR): incorrect responses in which subject continues to apply the same incorrect rule, even when they have been negatively reinforced (e.g. the subject continues to use *a* +1 rule even after the first unsuccessful attempt; thus with the −1 rule active a pattern such as this could be obtained: S: 6 R: 7; S: 5 R: 6; S: 4 R: 5 and so on);

(v) Bizarre errors: where the participant is incorrectly behaving in accordance with an "implausible" or unfruitful heuristic. We considered as plausible, attempts that follow any proximity heuristic: any S–R pair where the two positions are: (a) adjacent; or (b) at the two extremes of a row.

(vi) Move errors: where a subject has correctly attained a rule, but then goes on to make an error. Treating at least two successive correct responses as evidence that the participant has attained a rule, we calculated the number of times each subject subsequently made an error before the rule changed. We considered here the ratio of the number of move errors to the number of attained rules.

For each error type, apart from PPRu and move, the rate at which each error type occurs was evaluated. This taxonomy is neither mutually exclusive nor exhaustive. All error types were computed on the Brixton FirstHalf only.

2.4.2. Interference

(i) Interference failures: the proportion of times on which the subject loses a rule after the red interfering cards, given that the rule has been acquired before the interruption.

(ii) Capture errors: the participant incorrectly applies the interference rule to the first standard blue card following the interference. We considered the ratio between the number of capture errors following an attained rule and the number of trials on which this error type was possible, i.e. the number of attained rules in the SecondHalf of the Brixton test.

(iii) Recovery failure: the proportion of times in which subjects recover the same correct rule on the second card after the interference.

2.4.3. Reaction times

We analysed: (i) the median RTs for correct and wrong responses (as baseline duration for the response, we used the RTs to the two last stimuli in the interference procedure); (ii) the difference in the median RTs *after* (i.e. when the feedback is delivered) correct and error responses, i.e. we calculated [median (t_4, t_5, t_6)] – [median (t_2, t_3)] (see Fig. 2); (iii) for each error type the RT both before each example of the error type and before all the other errors but the one being considered. The relevant RTs here are t_1, t_2, t_6 (see Fig. 2). These RTs have been first sorted depending on the error type which follows (i.e. depending on R_1, R_2, R_6), then the median has been computed. Only participants who produced more than three errors of the type under examination have been considered. Analyses (i) and (ii) were carried out using only FirstHalf RTs; in contrast for the indices described in (iii) the RTs throughout the Brixton test were evaluated, in order to increase the computation base.

2.5. Statistical analysis

2.5.1. Group analysis

The raw data was first checked for normal distribution using the Kolmogorov–Smirnov test and for homogeneity of variance by the Levene test. Variables differing significantly from the normal distribution or having inhomogeneous variances between groups underwent logarithmic transformation. If one of the assumptions necessary to apply for the analysis of the covariance (ANCOVA) was still not valid then after transformation, a non-parametric test, the Mann–Whitney was used. In this latter case, *P*-values were estimated using the Monte Carlo method. Where an ANCOVA was carried out, the effects on the dependent variables were evaluated covarying for age, education and sex.[1] Given our expectation on

[1] The covariation for age, education and sex cause a reduction (−3, one for each factor) of the degrees of freedom (d.f.) of the *F* distribution denominator in all our ANCOVAs.

the direction of the effects for most of variables considered, we generally used one-tailed tests if not otherwise specified. Effects were considered significant at the $P < 0.05$ level.

2.5.2. Correlation analysis

We evaluated the correlations between a set of potentially relevant variables and FirstHalf score. A multiple regression analysis was performed with the variables of interest (FirstHalf score was always the dependent measure). The percentage of the variation explained (R^2) controlled for demographic factors was extracted and F statistics calculated for this value.

2.5.3. Single case analysis

A multiple single case analysis was also run. The performance of each patient was evaluated to examine if a particular measure had a value that was significantly different from the one predicted by the regression analysis on the control group with age, education and sex as independent variables. The significance level for each comparison was set to 0.05 (one-tailed).

3. Results

3.1. Demographic factors

Education is the only factor significantly affecting FirstHalf score in controls [$R^2 = 0.11$, F (1, 39) = 6.38, $P < 0.05$ two-tailed] while in patients age is the only one [$R^2 = 0.44$, F (1, 36) = 33.81, $P < 0.001$ two-tailed].

3.2. Days from onset, dimension of the lesion, aetiology and oedema

We evaluated the presence of an effect of each of these four variables on FirstHalf score. For the first two variables, we performed a regression analysis with age, years of education and sex as covariates and FirstHalf score as a dependent variable. In none of the cases was the effect significant either for days from onset, after logarithmic transformation [$R^2 = 0.003$, F (1, 35) = 0.213, $P > 0.1$] or dimension of the lesion [$R^2 = 0$, F (1, 22) = 0.018, $P > 0.1$]. For the aetiology and oedema, we used an ANCOVA, again with demographic factors as covariates. Apart from traumatic brain injury ($n = 1$) four aetiologies were involved: meningioma ($n = 17$), glioma ($n = 9$), stroke ($n = 9$), and other brain neoplasm ($n = 4$). A difference in aetiology did not affect FirstHalf score [F (3,32) = 0.630, $P > 0.1$]. Finally, the degree of oedema is similar across groups [F (3,35) = 0.176, $P > 0.1$]. The extension of the oedema did not affect FirstHalf score significantly [F (3,33) = 1.513, $P > 0.1$].

3.3. Raven matrices

Frontal patients overall did not have a significantly lower score on Raven Matrices than the controls [F (1, 78) = 1.218, $P > 0.1$]; at subgroup level, only the inferior medial patients had poorer performance [F (1, 49) = 4.228, $P < 0.05$], with the left lateral subgroup displaying a trend in the same direction [F (1, 48) = 2.714, $P < 0.1$].

3.4. Working memory test

This test was straightforward for controls, all but one of whom had a score equal to or greater than 8 out of 10 (the outlier had a score of 5). A regression analysis with sex, age and education as covariates was performed using the control group (excluding the outlier). The contribution of this variance to the WM variable was negligible [$R^2 = 0.001$, F (1, 37) = 0.046, $P > 0.1$]. The frontal group, by contrast, showed a clear impairment. They produced a significantly higher error rate compared to the Control Group [Mann–Whitney, $z = 2.909$, $P < 0.001$]. However, the deficit is not found among all the lesion groups: only the LL [Mann–Whitney, $z = 3.3$, $P < 0.01$], and the IM [Mann–Whitney, $z = 2.522$, $P < 0.01$] subgroups were significantly worse than the control group (Fig. 3).

3.4.1. Brixton FirstHalf

The combined frontal group gave significantly fewer correct responses on FirstHalf score than did the control group [F (1, 78) = 2.884, $P < 0.05$]. Two subgroups were impaired relative to controls: LL, IM. The LL group had higher proportion of errors on Brixton FirstHalf [F (1, 48) = 6.117, $P < 0.01$]. A significant effect was also obtained in the IM subgroup [F (1, 49) = 3.698, $P < 0.05$] (Table 4). In a secondary analysis, we checked if damage to the frontal pole region has a negative impact on Brixton performance: FP patients did not show a significant deficit on FirstHalf score [F (1, 52) = 1.040, $P > 0.1$]. FP is a subgroup that overlaps with others (it is composed by 1 LL, 3 RL, 8 IM, and 2 SM): if FP patients are removed from the LL subgroup the FirstHalf accuracy is

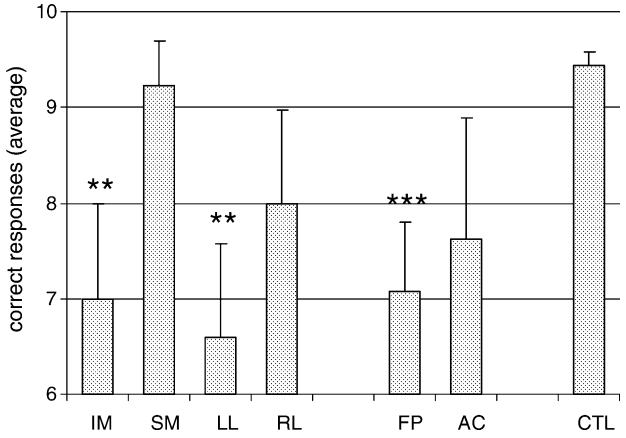

Fig. 3. Working Memory test: average of correct responses. IM: Inferior medial frontal; SM: superior medial frontal; LL: left lateral frontal; RL: right lateral frontal; FP: frontal pole; CTL: control group. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$ for the control group vs. patient subgroup comparisons.

Table 4
Error types for each lesion subgroup and controls

	LL	RL	IM	SM	Overall	CTL
Brixton FirstHalf (proportion of errors)	0.62 (0.15)**	0.47 (0.20)	0.60 (0.16)*	0.54 (0.12)	0.56 (0.17)*	0.48 (0.15)
PRe errors	0.07 (0.06)	0.06 (0.08)	0.04 (0.05)	0.03 (0.04)	0.05 (0.06)	0.06 (0.06)
PPRu (odds ratio)	3.43 (3.19)	3.36 (1.27)	3.52 (2.30)	3.47 (1.40)	3.45 (2.12)	3.07 (2.63)
SR	0.22 (0.18)	0.21 (0.15)	0.24 (0.24)	0.17 (0.13)	0.21 (0.18)	0.15 (0.11)
Bizarre	0.20 (0.16)	0.07 (0.14)	0.15 (0.15)	0.19 (0.15)	0.15 (0.15)	0.11 (0.12)
Move errors	0.21 (0.17)	0.25 (0.21)	0.17 (0.32)	0.27 (0.14)	0.22 (0.22)	0.20 (0.15)
Errors after interference	0.61 (0.35)*	0.50 (0.32)*	0.37 (0.33)	0.49 (0.19)	0.49 (0.31)*	0.34 (0.21)
Capture errors	0.10 (0.16)	0.27 (0.15)***	0.15 (0.15)	0.19 (0.16)	0.18 (0.16)*	0.11 (0.13)
Recovery	0.50 (0.44)**	0.74 (0.35)	0.63 (0.49)	0.76 (0.37)	0.65 (0.41)**	0.88 (0.26)

IM: inferior medial frontal; SM: superior medial frontal; LL: left lateral frontal; RL: right lateral frontal; CTL: control group; PRe: perseveration of the response; PPRu; perseveration of the preceding rule. Values significantly different from control group are underlined. Averages with S.D. in parentheses are reported.
* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

still significantly lower than controls [F (1, 47) = 5.661, $P < 0.05$]. The same kind of analysis cannot be performed for IM because of the strong overlap between IM and FP.

3.4.2. *The effect of WM capacity on Brixton FirstHalf score*

We split the lesion subgroups into patients who scored in the normal range on the WM measure (WM+) and patients with a score below the normal range (WM−). The WM- subgroups (combined frontal, LL, IM) all had a FirstHalf score significantly lower than the control group; while in the case of WM+ subgroups, only the LL subgroup still continued to show a significant impairment [F (1, 42) = 6.058, $P < 0.01$]. However, this was not the case for the IM subgroup (Fig. 4 see also Fig. 5). Moreover, if the WM measure is introduced as a covariate in the ANCOVA, the basic effect is no longer obtained in the IM subgroup but it remains significant in the LL one [F (1, 47) = 4.04, $P < 0.05$].

3.4.3. *Effects of the interference*

After the interfering stimuli, the frontal patients overall failed to apply the rule they had previously attained significantly more often than did the control group [F (1, 78) = 4.299, $P < 0.05$]. At the subgroup level the LL group showed the same effect [F (1, 48) = 4.083, $P < 0.05$] and so did the right lateral (RL) group [F (1, 48) = 4.98, $P < 0.05$]. Recovery of the rule on the second post-interference trial was also significantly more difficult for the frontal group than for the control group [Mann–Whitney, z = 2.492, $P < 0.01$]. The effect is largely ascribable to the LL subgroup [Mann–Whitney, z = 2.771, $P < 0.01$], which is the only subgroup which shows a significant effect.

The combined frontal groups made significantly more capture errors on the first blue card than did the control group [F (1, 78) = 4.679, $P < 0.05$]; this finding can be mainly attributed to the RL subgroup [F (1, 48) = 10.577, $P < 0.001$ one-tailed] (Fig. 6). The effect in the RL subgroup is still present even if we exclude from the analysis patients who scored outside the normal range on the working memory test [F (1, 45) = 13.056, $P < 0.001$]. To further statistically support, the double dissociation between capture errors and FirstHalf score, we performed additional analyses: a direct comparison on z-scores between LL and RL both for capture errors [F (1, 18) = 8.228, $P < 0.01$ two-tailed] and FirstHalf score [F (1, 18) = 8.570, $P < 0.01$ two-tailed], and an ANOVA 2 (groups) × 2 (error types, within factor) in order to check for

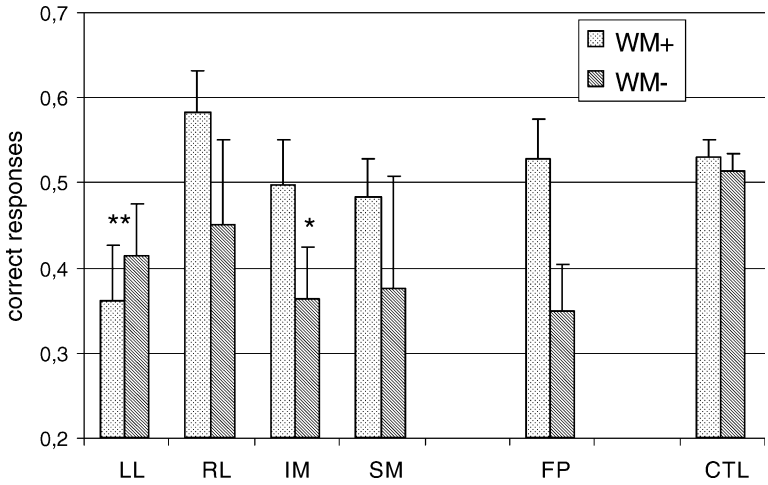

Fig. 4. Brixton FirstHalf: Performance of patients' subgroups according to whether they scored in (WM+) or below (WM−) the normal range on the working memory test.

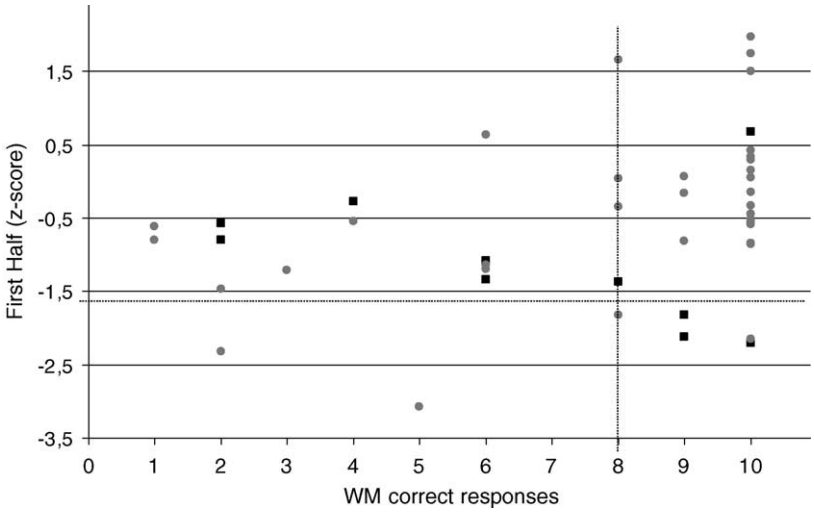

Fig. 5. Performance on Brixton FirstHalf (*z*-scores) and WM test (number of correct responses) for each patient. Black squares are subjects belonging to the left lateral lesion group; dotted lines represent the normal limits.

the interaction [F (1, 18) = 18.533, $P < 0.001$ two-tailed]. Dissociations between capture errors and FirstHalf score were also found in single cases. None of the four patients with a capture errors score significantly higher than the control group also had a poor FirstHalf score; moreover two of them were above the controls' mean. None of the seven cases with a significantly below normal FirstHalf score produced a number of capture errors outside the confidence limits based on normal control data; furthermore four of them were even below the control mean. A significant correlation was not found between capture errors and FirstHalf score for the combined frontal group [R^2 = 0.013, F (1, 35) = 1.014, $P > 0.1$]. As a subsidiary analysis, we also checked the anterior cingulate group for the rate of occurrence of capture errors: they made significantly more errors of this kind than the control group [F (1, 46) = 5.831, $P < 0.05$]. Since classification in this subgroup is not mutually exclusive with the others, we verified if the effect could be due to participants having both structures, AC and RL, damaged. Excluding such patients did not change the pattern [RL: F (1, 43) = 7.982, $P < 0.01$; AC: F (1, 43) = 3.060, $P < 0.05$]. As for the RL subgroup, the impairment in the AC subgroup remains significant even when the patients with a WM score below the normal range are removed [F (1, 43) = 6.479, $P < 0.01$].

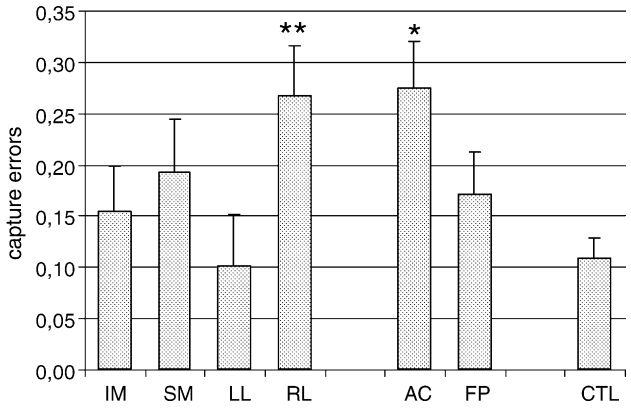

Fig. 6. Capture errors for each subgroup. Conventions as in Fig. 3.

3.4.4. Error type analysis

The frontal patients overall did not showed an increase in any of the error kinds considered: either for PRe [F (1, 78) = 0.201, $P > 0.1$], PPRu [F (1, 78) = 0.333, $P > 0.1$], SR [Mann–Whitney, z = 1.240, $P > 0.1$], bizarre errors [F (1, 78) = 0.378, $P > 0.1$] or move errors [Mann–Whitney, z = 0.098, $P > 0.1$]. At the lesion subgroup level (Table 4), the picture was the same. In particular for the two groups with a pathological FirstHalf score, we did not obtain significant effects either for PRe [LL: F (1, 48) = 0.025, $P > 0.1$; IM: F (1, 49) = 0.796, $P > 0.1$], PPRu [LL: F (1, 48) = 0.015, $P > 0.1$; IM: F (1, 49) = 0.024, $P > 0.1$], SR [LL: F (1, 48) = 2.450, $P > 0.05$; IM: Mann–Whitney, z =0.957, $P > 0.1$], bizarre errors [LL: F (1, 48) = 0.807, $P > 0.1$; IM: F (1, 49) = 0.459, $P > 0.1$] or move errors [LL: F (1, 48) = 0.294, $P > 0.1$; IM: Mann–Whitney, z = 1.710, $P > 0.1$]. Since there is, in the LL subgroup, a statistical trend for SR errors to be above the control level an additional analysis was carried out to check whether the LL subgroup still have a Brixton FirstHalf score significantly worse than controls if all the trials in which an error SR was produced are excluded from the computation of the Brixton FirstHalf score. The answer is positive [F (1, 48) = 4.230, $P < 0.05$].

3.4.5. Reaction times analysis

Frontal patients were not significantly slower or faster than controls either if the RTs are computed on error trials, on correct responses or on all the responses; a similar outcome was obtained for all subgroups apart from the superior medial one; this group showed a significant increase in the median RTs computed on all responses [Mann–Whitney, z = 2.020,

$P < 0.05$ two-tailed] and before errors [Mann–Whitney, $z = 1.996$, $P < 0.05$ two-tailed] compared to control group. Both the control and the frontal group showed a significant increase in median RTs on error trial in comparison with the RTs on correct responses [frontal patients: paired $t(39) = 4.833$ $P < 0.001$; CTL: paired t (35) = 7.315 $P < 0.001$ two-tailed]. In general, participants were significantly slower after errors, in the case of both controls [paired $t(42) = 7.510$ $P < 0.001$ two-tailed] and patients [paired $t(39) = 4.094$ $P < 0.001$ two-tailed]. The size of the slowing down was not statistically different between control group and either frontal patients overall or any of the subgroups. There were no significant differences in the PPRu, SR and bizarre errors RT index between the control group and either frontal patients overall or any of the subgroups. PRe and move error RTs were not analysed because too few participants produced enough errors of this type. Frontal patients were significantly faster when producing SR errors than other error types [paired $t(35) = 2.774$ $P < 0.01$ two-tailed]; the control group showed a statistical trend in the same direction [paired $t(33) = 1.811$ $P < 0.1$ two-tailed]. The control participants, but not frontal patients, were slower producing bizarre errors than the other types of error combined [paired $t(21) = 3.334$ $P < 0.01$ two-tailed].

4. Discussion

The Brixton spatial rule attainment task (Burgess & Shallice, 1996a) is a procedure devised to investigate impairments in rule induction and rule following. It is less prone to perseverative types of responding than the WCST, but still produces deficits following prefrontal lesions. A key process in the Brixton test is held to be rule induction. However, a number of other processes/abilities may be required for satisfactory task performance. We considered the following processes, all of which can be involved in the task: working memory, monitoring and checking, rule-following, set shift, and impulsivity.

Our study had these aims:

(i) to determine if the difficulties frontal patients have with the Brixton test (Burgess & Shallice, 1996a) could be explained by the deficits other than the inductive reasoning one;
(ii) to attempt to localise difficulties in the Brixton test performance to specifically frontal cortex areas that were not examined in the original study;
(iii) to examine checking a monitoring processes in particular by using an analogue of the Jacoby exclusion procedure in episodic memory research.

The frontal patient group was impaired on the main performance measure, i.e. the proportion of correct responses on the FirstHalf score of the revised version of the test, where there are no interfering stimuli. The left lateral and inferior medial groups showed impaired performance on this measure; by contrast, the right lateral and superior medial groups scored well within the normal range. These different levels of performance are not due to any lack of equivalence on any of the demographic variables considered, or in the distribution of the lesion sizes, aetiology or of the presence of oedema. Moreover, none of these factors, apart from age, correlate significantly with FirstHalf accuracy. Is it possible to explain the failure of these frontal subgroups by any of the alternative hypotheses considered in Section 1? We examine the possibilities in turn.

4.1. Alternative hypotheses

4.1.1. Working memory hypothesis

If all participants with a WM span out of normal range were excluded from the subgroup analyses, the differences between controls and inferior medial patients were no longer present. In contrast, the left lateral subgroup was still significantly impaired. Moreover, in the left lateral subgroup with normal WM, as in the control group, the correlation between Brixton and WM score was not significant. Indeed, five out of seven patients with a significantly impaired Brixton score achieved a normal WM span. Therefore, even if an ability to store relevant information is necessary in rule discovery tasks such as Brixton, as suggested for example by the significant correlation between WM and FirstHalf accuracy or by the major difficulty that the patients with low WM capacity had on the Brixton, by itself this is not sufficient to satisfactorily accomplish the task. A failure by frontal patients on the Brixton task cannot always be reduced to a working memory problem.

4.1.2. Perseveration hypothesis

In the original work on the Brixton task, perseveration of preceding responses was combined with that of preceding rules. No difference was found between anterior and posterior groups in the proportion they formed of total errors. In the current study, we extended the investigation of perseverative errors both by differentiating (Sandson & Albert, 1984) between recurrent perseveration and stuck-in-set perseveration (perseveration of response errors and perseveration of the preceding rule errors, respectively) and by using a more detailed lesion analysis. However, our results confirm the original study; neither the frontal patients overall nor any of the lesion subgroups showed a significant increase of either type of perseveration. So the hypothesis that the Brixton impairment arises from the interfering effects of the previously active rule or the preceding response, as may be the case for the WCST (Stuss et al., 2000), can be rejected. A related issue concerns same response errors (i.e. when the same incorrect rule is repeatedly applied, even if it has been negatively reinforced). They can be considered either as just another kind of stuck-in-set perseveration or a measure of the participant's sensitivity to negative feedback. In this second case, the participants would not be "trapped" into repeated application of a highly activated action schema, but instead they are ignoring the feedback, continuing to answer as if they were right.

What speaks in favour of this second interpretation is that SR errors are produced even with rules not strongly activated, since they are not always preceded by a series of positively reinforced answers that follow the same rule, as for PPRu errors. In any case, none of the frontal subgroups produced significantly more SR errors than the control group.

4.1.3. Rule application hypothesis

Under this hypothesis, participants would not apply the rule to the next position of the blue circle. If this were the case, we would anticipate an increase of move errors that is, an evidence of trouble in working out the next blue circle position, after having demonstrated the attainment of the rule. However, neither frontal patients overall nor any of the frontal subgroups made more move errors than controls. This is consistent with the reports of patients, who often complain about their inability to find the correct rule and indeed frequently raise doubt that any is actually present, but never mention their difficulty in doing what they have in mind; this is a pattern clearly different from the one reported by Stuss et al. (2000) for the WCST.

4.1.4. Impulsivity hypothesis

If higher impulsiveness had been present in any of the frontal groups, they would have been expected to have faster reaction times than controls, especially for wrong responses. However, none of the frontal groups behaved in this fashion.

4.1.5. Monitoring and checking hypothesis

The final alternative possibility to be assessed is that the impaired performance on the Brixton test is secondary to a problem in monitoring and checking. We examined this possibility by using an interference paradigm in the SecondHalf of the revised Brixton procedure. In this part of the procedure, after enough cards had been presented so that most subjects had acquired the rule, the participants are exposed to a second interfering rule and must then revert to applying the rule previously attained. In so doing they have to avoid making the potential capture errors that the interfering rule induces. If there are no grounds for assuming that the subject has forgotten the original rule, then actual capture errors suggest a failure to monitor or check whether the correct rule is being applied. The right lateral group had a significantly larger proportion of capture errors than the control group making nearly three times their rate. The group had no overall problem on the working memory task. Removing individual patients who do show such a problem still leaves an excess of capture errors. Moreover, on the second trial after the interference the subgroup is quite normal unlike the left lateral subgroup who do not retain well even rules they actually acquired. Thus, an explanation of the high rate of capture errors of the right lateral group as a memory problem is implausible.

Our results suggest that there is no causal role of a monitoring deficit on the basic failure in the Brixton test. In fact none of the two frontal subgroups impaired on the FirstHalf score showed a significant excess of capture errors after the interference procedure. In a subsidiary analysis, the anterior cingulate group showed an effect similar to the right lateral group.

If one attributes the capturing error problem in the right lateral group to a failure of monitoring or checking, why should the right lateral group be unimpaired on the FirstHalf score? If we assume that the monitoring and checking processes come into play when effective management of a conflict is needed (as in the case of the two plausible rules in the first cards after the interference), we can hypothesize that during the induction phase in the Brixton FirstHalf participants usually generate only one possible rule for each card presented: consequently they do not need to start a checking procedure.

4.2. Localisation of functions

4.2.1. Monitoring and checking

Frontal patients overall produced significantly more capture errors than controls. At the subgroup level only the right lateral patients showed an increase; in addition subsidiary analyses show an effect of anterior cingulate lesions. This pattern remains unchanged even when we excluded participants who had lesions involving both the right lateral and the anterior cingulate cortices. The right lateral localisation obtained is consistent with a series of studies, which used episodic memory paradigms to explore the neuroanatomical substrate of monitoring processes (see Shallice, 2002). Thus, in an efMRI study, Henson and collaborators (Henson et al., 1999 see also Rugg, Otten, & Henson, 2002) administered a verbal source memory task to participants. Right dorsolateral activation was interpreted in terms of control of monitoring or checking process. Neuropsychological findings are also available which suggest an on-line monitoring failure. In a task of episodic free recall (Stuss et al., 1994) patients with a right frontal lesion were the only group to make more item repetitions than controls. Yet, they had normal recall performance; it appeared that they did not check their output adequately. In our study, the anterior cingulate subgroup also showed an effect on capture errors. This finding can be related to a line of research which proposes that this area is required for the processes involved in conflict detection between incompatible response tendencies (e.g. Carter et al., 2000). Typical tasks used to elicit conflict situations are versions of the Stroop, the Simon or the Flanker test (Fan, Flombaum, McCandliss, Thomas, & Posner, 2003). In our case, a conflict would be generated by the competition between the interfering rule and the previously active one: if a participant does not notice its presence, s/he would carry on using the most recent rule (i.e. the one of the interference), thus committing a capture error. However, the findings are also compatible with broader characterizations of the functions of the anterior cingulate such as those of Posner and DiGirolamo (1998) and Critchley et al. (2003). On the whole our results suggest that the neural network necessary to successfully manage a potentially conflicting situation, should involve both the right lateral and the anterior cingulate cortices.

4.2.2. Induction

This leaves the fundamental process being examined in this paper, induction. For the inferior medial group it is possible to interpret the deficit on the basic Brixton task as secondary to a working memory problem. This is not possible for the deficit of the left lateral group: the LL subgroup which had intact performance on our measure of working memory was still impaired on the basic Brixton measure, FirstHalf score. Combining the present findings with results of the earlier study by Burgess and Shallice (1996a), which showed a lack of any difficulty on Brixton in posterior patients, we can suggest that a key process necessary to carry out inductive inference is localized in the left convexity of frontal cortex. This is consistent with most of the imaging literature reported in Section 1: in the majority of the studies the areas activated also involved the left lateral prefrontal cortex (Duncan et al., 2000; Goel et al., 1997; Osherson et al., 1998; Parsons & Osherson, 2001). Furthermore, our study suggests that when other frontal activations are elicited, as frequently occur, they are not crucial. This convergence between functional imaging and neuropsychological evidence is especially valuable given the variety of complex processes that any realistic induction task requires.

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