BRAIN INJURY, 2003, VOL. 17, NO. 6, 479–496

Taylor & Francis
healthsciences

Relationships between olfactory discrimination and head injury severity

PAUL GREEN†, MARTIN L. ROHLING‡, GRANT L. IVERSON§ and ROGER O. GERVAIS†

† Neurobehavioural Associates, Edmonton, Alberta, Canada ‡ University of South Alabama, Mobile, AL, USA § University of British Columbia & Riverview Hospital, Vancouver, BC, Canada

(*Received 30 March 2001; accepted 20 November 2002*)

The goal of this study was to examine the relationship between brain injury severity and scores on both an olfactory identification test and on many widely used neuropsychological tests in 367 patients with head injuries of varying levels of severity. It was hypothesized that valid olfactory test scores would correlate highly with injury severity because both the olfactory nerves and the primary olfactory cortices are especially vulnerable to damage in closed head injury. After removing data of doubtful validity from cases failing effort tests, olfactory test scores were related to Glasgow Coma Scale scores (GCS), post-traumatic amnesia and radiological abnormalities more strongly than any of the neuropsychological test scores. Based on the assumption that post-traumatic amnesia is caused by a different mechanism than loss of core consciousness, it was also predicted that there would be no cases with a GCS less than 13 and with no post-traumatic amnesia. As predicted, there were no cases in this group. The results support previous studies showing greater olfactory impairment with increased severity of head injury.

Introduction

The current research evolved from the routine use of a very inexpensive and convenient olfactory discrimination test (Alberta Smell Test) as part of a battery of neuropsychological tests with patients with head injuries and neurological diseases being assessed for disability [1]. In previous studies with patients with head injuries [1, 2], effort was measured with two Symptom Validity Tests (SVT); the Computerized Assessment of Response Bias (CARB) [3] and the Word Memory Test (WMT) [4–8]. In those with the most severe brain injuries who passed both of these SVTs, the odds of having impairment of olfactory discrimination were 10 times higher than in patients with the most mild head injuries. Yet, in those making insufficient effort to pass the SVTs, there was no relationship between olfactory scores and head injury severity. The patients with mild head injuries who failed the SVTs were 4.5 times more likely to produce impaired olfactory test scores than those who passed them, whereas those passing the effort tests scored no differently from an orthopaedic control group [1, 2]. Thus, exaggeration of impairment can greatly influence olfactory test scores.

Correspondence to: Paul Green, 201-17107-107 Avenue, Edmonton, Alberta, Canada, T5S 1G3. e-mail: paulgreen@shaw.ca

Brain Injury ISSN 0269–9052 print/ISSN 1362–301X online © 2003 Taylor & Francis Ltd
http://www.tandf.co.uk/journals
DOI: 10.1080/0269905031000070242

There remains some confusion regarding the relationship between olfactory changes and brain injury severity. For example, Doty *et al.* [9] reported that duration of loss of consciousness was unrelated to impairment of olfactory discrimination. Sampling characteristics partly explain the unusually high (87%) incidence of impairment in their patients, who were self-referred to a smell and taste clinic complaining of impaired sense of smell. Also, well-validated effort tests were not employed to screen out exaggerated olfactory impairment, which could make a substantial difference because there was a 26% failure rate on effort tests in cases of mild head injury in the study of Green and Iverson [1]. Other researchers have reported increased impairment of olfactory discrimination in patients with more severe brain injuries [10–13]. However, the amount of effort applied to testing was also not measured in any of these studies, which might explain why the estimates of olfactory impairment in groups with mild head injuries varied from less than 5% to more than 40% of cases. Differences in the composition of the mild head injury groups, such as including those with both uncomplicated and complicated injuries, might also have affected these results. To control for exaggerated impairment in the current study, the main analyses were conducted only after removing people who failed the CARB or WMT effort tests.

This study further examines the relationships between olfaction and head injury severity, using hypotheses derived from anatomical considerations. Costanzo and Zasler [13] listed the known and presumed mechanisms underlying impairment of sense of smell as an effect of head injury as follows: (1) traumatic damage to the nasal epithelium; (2) shearing of the olfactory fila, arising from the nasal epithelium, prior to entering the olfactory bulbs, as a presumed consequence of movement of the brain relative to, and/or fracture of, the cribriform plate; and (3) contusion or oedema effecting the olfactory bulb or the lateral or medial olfactory tracts. All of these mechanisms would cause impairment in the ability to detect odours. The authors also stated, 'It is possible that most cases of anosmia are caused by damage to central olfactory brain regions' (p. 21), in which case olfactory discrimination could be impaired without any loss of the ability to detect odours.

Costanzo and Zasler ([13], p. 20) presented a diagram of the central olfactory brain regions, including the anterior temporal lobes and orbital-frontal poles, both of which are among the most likely sites of contusions after a closed head injury [14]. Levin *et al.* [10] reviewed many studies showing that the temporal lobes and orbital frontal regions contribute to olfactory discrimination. They concluded that non-missile head injury could produce impairment of olfactory recognition, despite preserved olfactory detection, just as an auditory cortex lesion can affect speech perception without causing deafness. On an important historical note, it was pointed out that the classic amnesic patient, H.M., with bilateral mesial temporal lesions, could detect odours but had impaired olfactory discrimination.

In closed head injuries, it is likely that the sphenoid wings, which are in contact with the anterior borders of both temporal lobes, play a major role in producing temporal lobe damage and dysfunction. The upper border of the sphenoid bone is sharply angulated, like the edge of a shelf. In the centre of the skull base, the sphenoid wings or ridges terminate in two bony projections called the anterior clinoid processes, derived from the Latin word for 'bedposts'. They are adjacent and immediately anterior to the uncus on both sides and point posteriorly towards the foramen magnum ([15], pp. 844, 880). Hence, with a violent forward movement of the brain relative to the skull, the anterior clinoid processes would be

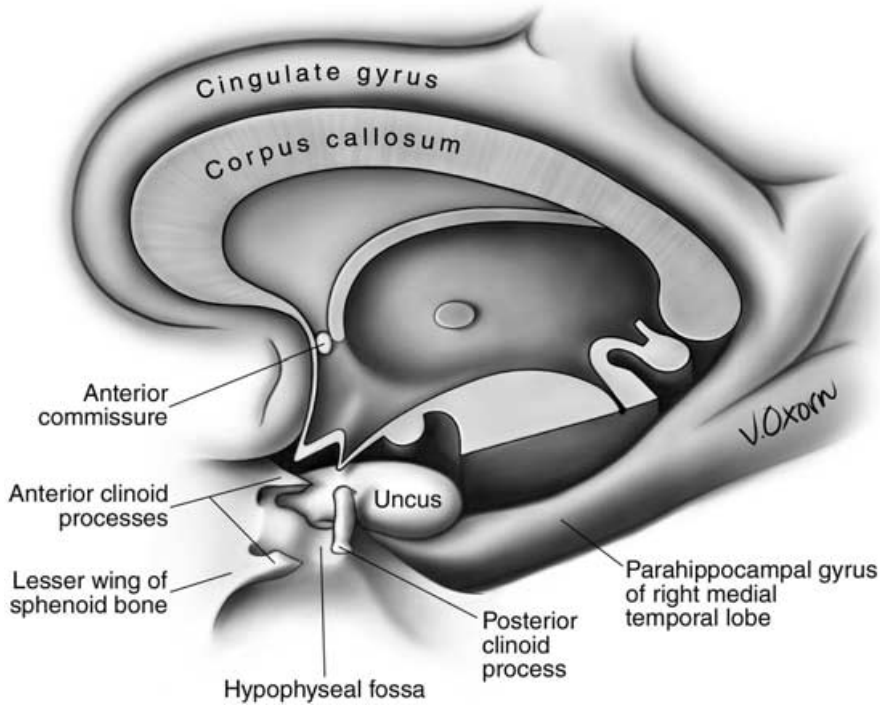

Figure 1. Proximity of uncus to anterior clinoid process.

expected to produce contusions in the region of the uncus (figure 1). If so, the primary olfactory cortex on each side would be especially vulnerable to head trauma (the uncal-clinoid theory). Smith ([16], p.180) states that the primary olfactory cortex is on the anteromedial part of the uncus on each side. The olfactory tract is attached to the outer aspect of the hemisphere, whereas other sensory pathways enter the hemisphere through the anterior capsule. Hence, the impact of the skull's jagged interior upon the uncus could damage the primary olfactory cortex or the terminal portion of the olfactory nerve or both.

Because the uncus is so vulnerable to trauma, it may be hypothesized that olfactory discrimination scores will be related to measures of head injury severity, especially duration of post-traumatic amnesia (PTA) and CT or MRI abnormalities. One way of examining this question is to employ multiple regressions, with various measures of head injury severity as the dependent variables, using many neuropsychological test scores from head injury patients as the independent variables. Neuropsychological tests, by definition, are sensitive to the impairment produced by traumatic brain injury (TBI). It was hypothesized that olfactory discrimination scores would explain at least as much of the variance in head injury severity as any of the most widely used neuropsychological tests (Hypothesis 1). This hypothesis implies that there will be significant negative correlations between olfactory discrimination and head injury severity, defined by PTA duration, GCS or CT/MRI brain abnormalities. It is also implied that scores on many neuropsychological tests will be lower in brain injury patients with impairment of olfactory discrimination than in those with normal olfaction. On the other hand, olfactory test scores would not be expected to predict the severity of neuropsychological impairment in a group

of patients with heterogeneous and non-traumatically produced neurological lesions, scattered around many parts of the brain (e.g. the lesions in multiple sclerosis, stroke or brain tumour).

Most lesions causing amnesia are found in the medial temporal lobes. Damasio [17] highlights a classic case with bilateral medial temporal lobe lesions caused by encephalitis, in which there was profound amnesia but intact consciousness. Conversely, he identifies other parts of the brain in which lesions or neurochemical dysfunction are known to impair consciousness, especially the brainstem but also the hypothalamus, ventro-medial forebrain and cingulate cortex. Following the same rationale, one might deduce that contusions or neurochemical disturbance in the anterior and medial temporal lobes arising from head trauma would not cause impairment of consciousness but would produce PTA. The brainstem, as it emerges through the foramen magnum, is also in contact with the edge of a bony structure. However, in a moderate-to-severe head injury, the brainstem would be more protected than the anterior temporal lobes because it is relatively small and the surface of the posterior fossa is not as sharp and angulated as the sphenoid wings or the clinoid processes. As stated in the *Merck Manual* ([18], p. 1428) 'Non-penetrating trauma is more likely to affect the cerebral hemispheres and underlying diencephalon, which are larger and more exposed than the brain stem'. Other brain areas in which lesions could cause loss of consciousness would, similarly, be less at risk of traumatic injury than the anterior temporal lobes.

The brain areas subserving memory and olfaction are both affected more or less directly by impact with the sphenoid wings and anterior clinoid processes, which can occur with or without significant trauma to the brainstem or other brain regions. If the temporal lobes are traumatized, causing transient amnesia, the olfactory cortex will also be affected and vice versa. Therefore, one would expect increased PTA duration to correlate with decreases in smell test scores. Loss of consciousness may result from impact between the skull and the brainstem or stretching forces acting on the brainstem or from other mechanisms. None of these would directly affect olfaction because the primary olfactory cortex is remote from the brainstem and from other regions in which lesions would cause loss of consciousness. However, smell test scores and GCS would be expected to intercorrelate significantly, because the greater the head injury severity and the lower the GCS the more likely that olfaction would be impaired through impact between the sphenoid bone and the anterior temporal lobes. Perhaps one would not expect GCS and PTA to behave like equivalent variables if they are caused by damage to different brain regions.

Based on the above argument, one would assume that it is possible to have a blow to the head of sufficient severity to produce PTA and damage or dysfunction in the anterior temporal regions, without producing what Damasio [17] refers to as a loss of core consciousness, corresponding approximately with a GCS of 12 or lower. Hence, one would expect to find many cases of head injury with some PTA in whom there has been relatively mild or no impairment of consciousness (GCS 13 or higher). On the other hand, one would predict that it is very unlikely that a closed head injury would cause a loss of core consciousness without also disrupting the anterior temporal lobes and causing transient post-traumatic amnesia. Hypothesis 2 in the current study was that all cases in whom core consciousness was lost (GCS $<$ 13) would display post-traumatic amnesia after the head injury (where PTA is timed from the point of recovery of consciousness).

It was predicted that both olfactory discrimination test scores and neuropsychological test scores would be significantly different between the four groups defined by combinations of PTA (present/absent) and GCS (13–15 or less than 13). The largest impairment of olfaction would be expected in the group with a GCS less than 13 and with some PTA, whereas the smallest would be expected in the group with no PTA and a GCS of 13 or higher (Hypothesis 3). Finally, the areas of the cerebral cortex most likely to be affected by head injury, in addition to the anterior temporal lobes, are the frontal poles [14]. Therefore, scores on tests that are the most sensitive to frontal lobe functioning would be expected to show the greatest correlations with impairment of olfactory discrimination (Hypothesis 4). Selective impairment of executive functions in conjunction with olfactory impairment has previously been reported by Callahan and Hinkebein [11].

Method

Subjects

Participants were patients referred to a private practice in Edmonton, Alberta, Canada for psychological or neuropsychological assessment, related to evaluation of impairment and disability resulting from a work-related or non-work related accident or a neurological disease. All patients were involved in some form of compensation or medical disability claim at the time of their evaluation.

The first diagnostic group included 367 consecutive head injury cases, varying from very minor to very severe. The average age of these patients was 38.9 years (SD = 12.4) and their average education was 11.8 years (SD = 2.7). The sample was 79% male. All available details of head injury severity were recorded, including the lowest recorded GCS scores within 24 hours of injury, the presence or absence of intracranial CT or MRI brain abnormalities, the duration of PTA and the duration of loss of consciousness (LOC). If no GCS was recorded in the file, as in the case of patients who did not consult a doctor on the day of the accident, it was assumed to be 15, as long as there was no evidence that a patient lost consciousness, suffered any PTA nor exhibited any radiological brain abnormalities. Self-reports of PTA were not accepted unless they were independently confirmed by previous medical reports written shortly after the accident. When the emergency room notes indicated unspecified but short duration of amnesia, estimates of PTA were based partly on medical reports, partly on self-reports of the accident and immediate consequences on comprehensive interviewing and partly on reports of relatives who were with the patient shortly after the accident. Details of PTA duration, GCS scores and CT/MRI results are provided in table 1 for the 197 patients from this group (i.e. 68.4% of cases), chosen for the main analyses because they passed both of the effort tests and had a PTA estimate.

The second group consisted of 64 patients with neurological diseases (56% men) and heterogeneous brain lesions. The mean age of the neurological group was 46.6 (SD = 9.4) and they had a mean 13.5 years of education (SD = 3.7). There were 13 cases with ruptured cerebral aneurysms, 15 with strokes, 11 with multiple sclerosis, seven with tumours, three with epilepsy and 15 with miscellaneous conditions, such as herpes simplex encephalitis, Von Hippel-Lindau disease, hypoxic event, orbital frontal abscess, venous thrombosis and dorsal mid-brain haemorrhage. In 55 cases, brain CT or MRI results were available, and the findings were abnormal in 95% of

Table 1. Indicators of head injury severity in patients passing both effort tests, broken down by levels of PTA

Days of PTA	*n*	% with abnormal CT/MRI*	Median GCS	Mean GCS (SD)	Median PTA in hours	Mean PTA (SD)
No PTA	90	9%	15	14.9 (0.2)	0	0
< 1 day	46	23%	14	14.2 (0.9)	1	3.0 (4.5)
1–10 days	35	63%	14	11.5 (3.9)	72	88.5 (55)
> 10 days	26	100%	6	6.3 (3.3)	504	726 (650)

* The percentage with abnormal CT/MRI shown is the number of abnormal scans as a percentage of the total of cases at each PTA level. Figures on GCS are for those cases for whom both GCS and PTA were available.

these cases. The radiologically identified locations of cortical abnormalities in the neurological patients were diverse, including frontal 44%, temporal 26%, parietal 18% and occipital 11%, with lesions in more than one region in 22% of all cases. There were three ruptured anterior communicating artery aneurysms and four cases involving brain stem, basal ganglia or cerebellar lesions.

As a non-neurological control group for normative comparison purposes, 196 people were included with recent orthopaedic injuries not involving the head who had passed both of the tests designed to detect exaggerated cognitive deficits (i.e. the CARB and the WMT). These orthopaedic patients, 90% of whom were men, were given the Alberta Smell Test but not the other neuropsychological tests. The average age of the sample was 37.3 years (SD = 9.5) and their average education was 10.8 years (SD = 2.2).

Assessment procedures

The head injured and neurological patients in this study were all given the Alberta Smell Test and two SVTs to measure effort (CARB and WMT). Failure on these effort tests was defined using the criteria found in the test manuals, which are not reported here for test security purposes. The data from people failing effort tests were *not* included in the main analyses. All patients were given 1.5 days of testing and at least 2 hours of interviewing, and an attempt was made to give all patients the 43 neuropsychological measures shown in table 2. In some cases, this was not possible, largely owing to patient behaviour, including slowness, demands to rest and refusal to continue testing. The mean number of neuropsychological test scores obtained per case was 36, in addition to the smell test and the SVTs.

The Alberta Smell Test [1, 2] involves 10 trials in each nostril, in which the person is required to identify the odour presented. Subjects were required to close their eyes, to close one nostril at a time and to sniff when told to do so. Scented markers with authentic essences of lemon, orange, licorice, mint, raspberry, cinnamon, grape and melon were presented half an inch below one nostril at a time, in a standard order, alternating between nostrils after every four trials. Subjects were asked to identify the odours from a list of the eight options printed on a sheet of paper. The results were the number of correct responses out of 10 for each nostril. The non-toxic food-grade, scented water-colour markers are widely available, very inexpensive and have been manufactured by Sanford USA since 1972 under the trade name 'Mr Sketch' scented markers for use by young children. A mean smell

Table 2. Forty-three ability measures contributing to the Overall Test Battery Mean (OTBM), grouped by domain

Domain	*n*	Ability measures
EF, Executive Functioning	6	Wisconsin Card Sorting Test–Categories achieved & Perseverative errors; Category Test–Errors; Thurstone Word Fluency; Ruff Figural Fluency–Total score; Gorham's Proverbs
ML, Memory and Learning	15	CVLT–Total, Trial 5, SDFR, LDFR, Recognition hits; Warrington Recognition Memory Test–Words & Faces; Cognisyst Story Recall Test–Immediate & Delayed Recall; Word Memory Test–Paired Associates, Multiple Choice, Delayed recall, Long delayed recall; Rey CFT–Delayed Recall & Recognition
AW, Attention & Working Memory	8	Trail Making Test–Forms A & B; Digit Span–Forward & Backward; Visual Memory Span–Forward & Backward; CVLT–Trial 1 & List B
VC, Verbal Comprehension	4	WAIS-R Verbal IQ or MAB Verbal IQ; WRAT-III–Reading, Spelling & Arithmetic
PO, Perceptual Organization	4	Rey Complex Figure Test–Copy & Recall; Benton's Judgement of Line Orientation; WAIS-R or MAB PIQ
PS, Psychomotor Skills	6	Finger Tapping, Grip Strength & Grooved Pegboard (all for both dominant and non-dominant hands)

test score of 2.4/10 was at the 5th percentile for the 196 orthopaedic patients, and so this score was used to define impairment in the other groups (i.e. a mean of less than or equal to 2.4 is impaired). Their mean raw smell test scores were 5.6 (SD = 2.3) in the right nostril (naris), 6.0 (SD = 2.3) in the left nostril and 5.8 (SD = 2.1) for both nostrils. Scores in the right and left nostril correlated with each other at 0.6 in the orthopaedic controls. The correlations between the right and left nostrils in the head injury and neurological patients who passed the effort tests were 0.7 and 0.53, respectively.

Scores from all the neuropsychological tests shown in table 2 were calculated as *Z*-scores relative to external norms (i.e. scaled to a normal mean of 0 and SD of 1). After conversion to *Z*-scores, the test results were summarized in two ways. First, all *Z*-scores except olfactory discrimination were averaged to produce an overall test battery mean (OTBM), using the method reported in Rohling *et al.* [19]. In addition to this global measure of neuropsychological test performance, tests were clustered into the six domains of ability shown in table 2, which were memory and learning (ML), attention and working memory (AW), executive functions (EF), psychomotor skills (PS), verbal comprehension (VC) and perceptual organization (PO).

Results

Predicting head injury severity

Hypothesis 1 was that, after removing all cases failing the SVTs, olfactory discrimination scores would explain at least as much of the variance in head injury severity as any of the most widely used neuropsychological tests. This was tested by running

15 regressions with the dependent variables of PTA duration, GCS or presence/absence of CT/MRI brain abnormalities. In addition to mean smell test scores, five sets of independent variables were entered into separate stepwise regressions: (1) individual scores from each of the tests from three of the domains described above and listed in table 2 (executive functioning, memory and learning and attention and working memory); (2) individual scores from tests in the domains of processing speed, perceptual organization and verbal comprehension; (3) the domain scores for executive functioning, attention and memory; (4) the domain scores of verbal comprehension, psychomotor skills and perceptual organization, and (5) all six domain scores. The results are shown in table 3.

Smell test scores entered into the regression equation first 14 times out of 15 and, five times, no other variable entered the equation. Smell test scores were the best predictors of CT/MRI abnormalities in all five regressions, the mean *R* for smell being 0.4. Similarly, smell test scores were the best predictors of PTA duration in all five regressions, the mean *R* for smell being 0.27. With GCS as the dependent variable, smell test scores loaded first in four out of five regressions, with a mean *R* of 0.27. In one regression, smell test scores did not add anything to the variance in GCS, which was already explained by Warrington's Recognition Memory Test for faces and Wisconsin perseverative errors. Because of a lack of space, regressions employing loss of consciousness as the dependent variable are not shown in table 3, but, when the mean smell test score was entered along with the OTBM and all domain scores, the only variable to load was the mean smell test score.

Overall, smell test scores predicted CT abnormalities, PTA, GCS and LOC better than any of the other 43 neuropsychological test scores and also better than any of the neuropsychological domain scores composed of clusters of related tests. The correlation between PTA and GCS was −0.76, congruent with the fact that both of these measures are widely regarded as measures of head injury severity. CT abnormalities correlated −0.56 with GCS and 0.43 with PTA duration. Mean

Table 3. Variables loading first and second in regression equations including Alberta Smell Test scores and 43 neuropsychological variables with PTA, GCS and CT/MRI abnormalities as dependent variables

		First and second test to load for each dependent variable (and *r*-value for first variable)					
Independent variables	Test scores/ domains	CT/MRI abnormal	*r* for first variable	PTA	*r* for first variable	GCS	*r* for first variable
Smell, EF, AW, ML	All test scores	Smell	0.42	Smell	0.31	WRM-F★ WCST-P	0.33
Smell, VC, PS, PO	All test scores	Smell G Peg★	0.5	Smell PIQ	0.4	Smell	0.37
Smell, EF, AW, ML	Domain scores	Smell AW	0.36	Smell	0.31	Smell ML	0.24
Smell, VC, PS, PO	Domain scores	Smell PS	0.36	Smell VC	0.17	Smell	0.25
All domains	Domain scores	Smell AW	0.37	Smell AW	0.17	Smell ML	0.25

★ WRM-F = Warrington's Recognition Memory Test (faces), WCST-P = Wisconsin Card Sorting Test (perseverative errors), G Peg = Grooved Pegboard (left hand). All other two-letter entries represent domain scores as in table 2 (e.g. ML = memory and learning tests).

smell test scores correlated 0.31 with CT abnormalities, after controlling for GCS, LOC and PTA using a partial correlation. Smell test scores correlated with PTA duration (-0.23), GCS (0.29) and with LOC (-0.17; all significant at $p < 0.01$).

Mean smell test scores by CT/MRI abnormality, level of PTA and GCS

The head injured patients with normal CT or MRI scans who passed the effort tests obtained a mean smell test score of 5.7 (SD = 2.3, $n = 75$), which was significantly higher than the mean of 3.4 (SD = 2.8) from those with abnormal brain scans ($n = 86$; $F(1, 159) = 30.6$, $p < 0.0005$). In these head injury patients, smell test scores correlated significantly with CT abnormalities ($r = -0.35$) and also with OTBM ($r = 0.18$).

In table 4, the differences between the mean smell test scores by level of PTA are significant ($F(3, 193) = 13.3$, $p < 0.0005$). *Post-hoc* Bonferroni comparisons show that the two groups with the lowest PTA scored significantly lower on the smell test than the group with no PTA ($p < 0.0005$). The two groups with the lowest PTA did not differ from each other, nor did the two groups with the highest PTA. The mean smell test score of 5.9 out of 10 (SD = 2.0) in the patients with no PTA was not significantly different than the mean of 5.8 out of 10 (SD = 2.1) from the orthopaedic control group. In those with no PTA, 6.7% of cases had an impaired smell test score, compared with 37.5% in those with PTA of at least 1 day and 41% of those with 10 days of PTA or more.

Using GCS as the measure of severity (table 5), those with the more severe brain injuries (GCS < 13) scored significantly lower on the smell test than those with the least severe injuries (GCS 14 or 15; $F(2, 144) = 7.1$, $p < 0.001$). The group with GCS scores of 13–15 scored higher than the group with scores between 9–12 (Bonferroni $p < 0.05$) and higher than the group with GCS scores less than 9 ($p < 0.05$). The mean smell test scores were not significantly different in the group with the lowest GCS (3–8) compared with the intermediate GCS group (9–12).

Half (i.e. 49%) of the patients with a GCS of 12 or less had an impaired mean smell test score of 2.4 or lower, compared with 23% of the group with a GCS of 13 or greater (counting only those given a scan). In the mild injury group, 90% of those

Table 4. Smell test scores and levels of PTA duration in patients with head injuries, with and without CT or MRI brain abnormalities

	All cases		Normal CT or MRI			Abnormal CT or MRI		
PTA duration (days)	*n*	Mean smell test (SD)	Median PTA (hours)	*n*	Mean smell test (SD)	Median PTA (hours)	*n*	Mean smell test (SD)
0	90	5.9 (2.0)	0	37	6.2 (2.1)	0	8	4.3 (2.9)
< 1 day	46	4.8 (2.6)	1	19	5.3 (2.1)	4.3	11	2.5 (2.6)
1–10 days	35	3.5 (2.7)	72	10	4.6 (1.9)	60	22	3.3 (2.6)
> 10 days	26	3.1 (2.5)	—	0	—	672	26	3.2 (3.0)
Orthopaedic	196	5.8 (2.1)						

Note: Some of the less severe cases did not undergo CT or MRI and reliable PTA estimates were not available in some cases.

Table 5. Mean Alberta Smell Test scores by levels of GCS and normal vs abnormal brain scan, showing median GCS per group

		All cases		Normal scan			Abnormal scan		
GCS	*n*	Mdn GCS	Mean smell test (SD)	Mdn GCS	*n*	Mean smell test (SD)	Mdn GCS	*n*	Mean smell test (SD)
13–15	112	15	5.18 (2.6)	14.7	21	5.6 (2.5)	–	0	—
9–12	12	10.5	2.92 (2.9)	11	1	2.0 (1 case)	10	11	3.0 (3.0)
3–8	23	6	3.41 (2.7)	–	0	–	6	12	3.26 (2.9)

Note: Many of the milder head injury cases did not undergo brain scanning and, in some cases, GCS was not recorded.

with impaired smell had either an abnormal brain scan or some PTA. Hence, a blow that caused a GCS of 13 or higher with no PTA and no CT scan abnormality was very unlikely to produce impaired smell test scores in those who passed effort tests. However, impaired smell test scores were no more likely in those with a GCS of 3 than in those with a GCS of 12.

Combinations of GCS and PTA

Hypothesis 2 was that all patients with loss of core consciousness (GCS < 13) would also have some period of PTA, after recovery from coma. Each of the head injury patients was placed into one of four groups, based on the presence or absence of PTA and a GCS score of 13 or greater vs 12 or less. The groups were, in rank order of severity of head injury, from most mild to most severe: (A) GCS $\geq$ 13 with no PTA; (B) GCS $\geq$ 13 with some PTA; (C) GCS $\leq$ 12 with no PTA; and (D) GCS $\leq$ 12 with some PTA. The number of cases in each of these four groups are presented in table 6. As predicted, there were no cases with a GCS less than 13 and no PTA (group C).

Hypothesis 3 was that both olfactory discrimination test scores and neuropsychological test scores would be the highest in group A and the lowest in group D. Consistent with the hypothesis, the OTBM *Z*-scores were significantly different between groups ($F(3, 135) = 4.4$, $p < 0.005$). The mean OTBM values

Table 6. Mean Alberta Smell Test scores by combinations of GCS level and PTA duration, broken down into those with and without abnormal brain scan results

Group	% abnormal CT or MRI	*n*	All cases	*n*	Normal scan	*n*	Abnormal scan
GCS $\geq$ 13							
(A) No PTA	15%	56	5.8 (2.3)	29	6.1 (2.1)	5	3.9 (3.1)
(B) Some PTA	59%	40	3.2 (2.7)	14	5.1 (2.3)	20	3.4 (2.9)
GCS $\leq$ 12							
(C) No PTA	–	0	–	0	—	0	–
(D) Some PTA	96%	27	3.1 (2.7)	1	2.0 (–)	26	3.1 (2.8)

Note: % CT abnormalities applies only to those given a scan. Most of the milder head injuries were never given a CT or MRI scan.

were as follows: Group A, −0.08 (SD = 0.57); Group B, −0.15 (SD = 0.52); Group D, −0.41 (SD = 0.71). Group C had zero cases.

It can be seen in table 6 that the patients' scores on the smell test were significantly different between groups A, B and D ($F(2, 120) = 11.4$, $p < 0.0005$). The mean smell test score was 5.8 (SD = 2.3) in group A, which had a median GCS of 15 and a median PTA of zero (mean GCS = 14.95, SD = 0.2, mean PTA duration = 0 hours). The mean smell test score was 4.3 (SD = 2.7) in group B, whose median GCS was 14, with a median PTA of 2.75 hours (mean GCS = 14.4, SD = 0.6, mean PTA duration = 28.6 hours, SD = 79). The lowest mean smell test score was 3.1 (SD = 2.7) in group D, with a median GCS of 7 and a median PTA of 336 hours (mean GCS = 6.9, SD = 3, mean PTA duration = 384 hours, SD = 322). Using *post-hoc* Bonferroni comparisons, the mean smell test scores were significantly different between groups A and B ($p < 0.01$) and also between groups A and D ($p < 0.0005$) but not between groups B and D. The percentages of patients with mean smell test scores of 2.4 out of 10 or less were as follows: Group A, 8%, group B, 20%, group D, 36% ($\chi^2 = 9.8$, $p < 0.01$).

Relation between smell and executive functioning

Hypothesis 4 stated that scores on tests that are the most sensitive to frontal lobe functioning would be expected to correlate to the greatest degree with olfactory discrimination. There were small, significant correlations between smell test scores and the following neuropsychological domain scores (table 2): Executive Functioning (EF, 0.22), Verbal Comprehension (VC, 0.2), Psychomotor Skills (PS, 0.16) and Attention-Working Memory (AW, 0.15). Non-significant correlations were found between smell test scores and both Memory-Learning (ML) and Perceptual Organization (PO).

The domain scores correlated with the OTBM at levels ranging from 0.53–0.92, all of which are much higher than the correlation between smell test scores and the OTBM (0.18) or any of the domain scores, suggesting that smell test scores tap a factor that is independent of most neuropsychological tests. However, in 30 patients, who scored more than 1.5 SD below the normal mean on the memory and learning domain, the mean smell test *Z*-score (−1.1, SD = 1.1) was significantly lower than the mean from 237 patients whose memory and learning score was above the −1.5 SD cutoff (−0.42, SD = 1.3; $F(1, 265) = 6.8$, $p < 0.01$). Also, there were 30 patients who scored more than 1.5 SD below the normal mean on the executive functions domain and their mean smell test *Z*-score (−1.3, SD = 1.0) was significantly lower than the mean from 233 patients whose EF score was above the −1.5 SD cutoff (−0.41, SD = 1.3; $F(1, 261) = 12.6$, $p < 0.0005$). There were small, significant negative correlations between PTA duration and the memory ($r = -0.3$) and executive function domains ($r = -0.23$).

Effects of exaggeration on hypothesis testing

None of the above major conclusions about olfactory performance and head injury severity applied to the patients who failed the effort tests. In those who failed the CARB or the WMT: First, there were no differences between smell test scores by levels of PTA ($n = 87$, $F(4, 82) = 0.95$, $p < 0.43$); Secondly, there were no significant differences between the smell test scores by GCS level in those with known

GCS scores ($n = 59$, $F(4, 49) = 0.4$, $p < 0.49$); Thirdly, in those who were given CT or MRI brain scans, there were no differences in mean smell test scores between those with and without radiological brain abnormalities ($n = 89$, $F(1, 87) = 1.93$, $p < 0.17$); Finally, there were no differences between the mean smell test scores in groups A ($n = 24$), B ($n = 11$), D ($n = 8$) and all other cases with either missing GCS or PTA data ($n = 72$), $F(3, 111) = 0.7$, $p = 0.5$.

The mean smell test score in those who failed the WMT (mean 4.3, SD = 2.5, $n = 112$) was significantly lower than the mean score in those who passed the WMT (mean 5.0, SD = 2.7, $n = 255$; $F(1, 365) = 5.3$, $p < 0.02$). Approximately 23% of patients who passed the effort tests had a score on the smell test lower than 2.4/10 in one or both nostrils, compared to 32% of the patients who failed the WMT (Mann Whitney test, $z = (2.1$, $p < 0.036$, two tailed). CT scan abnormalities were found in 53% of 185 cases who passed the WMT effort tests and in 32% of 109 cases who failed the WMT (Mann Whitney, $z = (3.6$, $p < 0.0005$). Hence, more impairment in smell test scores was seen in those with the least objective abnormalities of the brain but who exaggerated their impairment on testing with SVTs.

The removal from the main analysis of all cases failing either the CARB or WMT is further supported by the results from 104 patients with relatively mild head injuries who were selected for PTA less than a day and no CT or MRI abnormalities. The 70 mild head injury cases who passed the WMT scored significantly higher on the mean smell *Z*-score (-0.09, SD = 1.0) than the 34 exaggerators who failed the WMT (-0.7, SD = 1.4; $F(1, 102) = 6.1$, $p < 0.015$). An impaired mean smell test score was found in 32.4% of the exaggerating mild head injury cases, compared to only a 5.7% incidence of impaired smell test scores in the mild head injury cases making a genuine effort.

On the OTBM, representing an average from up to 43 neuropsychological tests, those patients with mild head injuries who failed the WMT scored 1 SD lower (-1.17, SD = 1.4) than the mild head injury cases who passed the WMT (mean -0.18, SD = 0.48), $F(1, 102) = 51$, $p < 0.0005$. They also scored significantly lower than the mean of -0.4 (SD = 0.7) from 91 more severely injured patients who passed the WMT but who had PTA of more than 1 day *and* abnormal brain scans ($p < 0.0005$). It is notable that, in cases of mild head injury with PTA less than 1 day, 27% of those who failed the WMT effort measures produced an impaired score in only one nostril (26 out of 94 cases), whereas only 10% of those who passed the effort measures produced unilateral impairment on the smell test (19 out of 190; $\chi^2 = 10.2$, $p = 0.001$).

Anosmia

In people who stated that they could not smell anything at all when presented with odours (anosmics), the test was stopped after four trials per nostril, producing a score of zero. The percentages of cases in each group scoring a mean of zero on the smell test were as follows: patients with less than 1 day of PTA = 0.7% of cases; patients with 1 day of PTA or more = 20.0% of cases; and neurological patients = 3.6% of cases. Hence, frank anosmia was virtually non-existent in patients with PTA of less than 1 day.

The patients with zero mean smell test scores had significantly lower GCS scores (mean 10.4, SD = 4 vs 13.3, SD = 3.3; $F(1, 151) = 9.6$, $p < 0.005$), significantly longer PTA duration (462 hours, SD = 813 vs 88 hours, SD = 250; $F(1,$

213) = 20.8, $p < 0.0005$) and significantly more abnormal CT/MRI brain scans (91% vs 46%; $F(1, 173) = 16.5$, $p < 0.0005$) than all other cases. There were significantly lower *Z*-scores on the executive function domain score in the patients with anosmia (mean −0.77, SD = 0.9) than in all other head injury cases (mean −0.4, SD = 0.8; $F(1, 261) = 4.1$, $p < 0.05$). There were no significant differences between these two groups on the OTBM or the other ability domains. These data show that the most severe impairment of olfactory discrimination, a total loss of olfactory detection in both nostrils, was present almost exclusively in head injury patients with the most severe brain injuries.

For every head injury patient with a mean smell test score of zero, there were 1.2 cases with a non-zero score at or lower than 2.5/10, suggesting some impairment. These patients did not differ significantly from the anosmics on GCS or incidence of abnormal CT/MRI scans, but the patients with frank anosmia had significantly longer mean PTA ($p < 0.008$). In patients with PTA of 1 day or more, 14.7% of cases had a score of zero in one nostril but not the other, whereas such a pattern of results was found in only 3.8% of those with less than 1 day of PTA. If one excludes all cases with a score of zero in either nostril, those with the greatest PTA (1 day or more) still scored significantly lower than those with the least PTA (less than 1 day) both in the right nostril (mean 6.2, SD = 1.9 vs 4.9, SD = 2.1; $F(1, 191) = 14.7$, $p < 0.0005$) and in the left nostril (mean 6.3, SD = 2.1 vs 5.1, SD = 2.3; $F(1, 191) = 12.2$, $p < 0.001$). This means that, in many cases, the more severe brain injuries caused a significant drop in smell test scores without necessarily producing a complete loss of the ability to detect and discriminate odours.

Sex differences

In head injury patients, there was a significant sex difference in favour of women ($n = 61$, mean 5.6, SD = 2.5) vs men ($n = 185$, mean 4.7, SD = 2.7; $F(1, 244) = 4.8$, $p < 0.03$), and this was not a function of differences in head injury severity because the men and women in this sample did not differ significantly from each other on PTA duration, GCS or presence of CT/MRI abnormalities. In the neurological patients and the orthopaedic controls who passed the effort tests no significant sex difference was found on the smell test, but there were very few women in either group.

Neurological patients

The neurological patients' mean smell test score (4.9, SD = 2.3) was significantly lower than that of the orthopaedic controls (5.8, SD = 2.1); $F(1, 250) = 7.3$, $p < 0.007$. As predicted, there were no significant correlations between mean smell test scores and the OTBM or CT/MRI abnormalities in the neurological patients, unlike the patients with head injury. The lack of correlation with radiological abnormalities could be misleading because very few neurological patients had normal CT scans, restricting the range on this variable. In addition, the pattern of olfactory deficits in the neurological patients was quite different than it was in patients with head injuries. There were 24% of cases with a right nostril score of two out of 10 or lower and 15% with a left nostril score of two or lower, but only 11.3% had a mean score of 2.4 or lower, reflecting predominantly unilateral decreases in olfactory discrimination in the neurological patients. Twenty per cent of neurological patients had unilateral impairment, almost twice the number with an

impaired mean score. There was a significant difference between the mean smell test scores from the neurological patients, head injury patients with PTA less than 1 day ($n = 156$, mean 5.54, SD = 2.3), patients with more than 1 day of PTA ($n = 68$, mean 3.4, SD = 2.7) and patients of undetermined PTA ($n = 22$, 5.5, SD = 3.2), $F(3, 295) = 11.9$, $p < 0.0005$. The head injury patients with more than 1 day of PTA scored significantly lower than the neurological patients ($p = 0.006$), lower than the head injury patients with less than 1 day of PTA ($p = 0.0005$) and lower than the group with undetermined PTA ($p = 0.004$). The latter three groups did not differ from each other in mean smell test scores.

Discussion

In those patients making an effort to do well on testing, olfactory scores decreased sharply in parallel with increasing head injury severity, whether severity was defined by longer PTA duration, lower GCS scores or the presence of CT abnormalities. The patients with the most mild head injuries produced smell test scores that were no different from those of the orthopaedic controls. Impaired olfaction was especially unlikely in those with a GCS of 15, no PTA and no CT abnormalities. Scores of zero in both nostrils occurred 28 times more often in patients with 1 day or more PTA than in those with less than 1 day of PTA. Impaired smell test scores (i.e. a mean of 2.4 or less) occurred six times more often in patients with 10 days or more PTA than in those with no PTA.

It is important to note that, in patients who *failed* effort tests, olfactory test scores were unrelated to all measures of head injury severity. This was probably the result of exaggeration of impairment. As a group, the mild head injury patients who failed the effort tests were assumed to be producing invalid test results. They were more than five times more likely to produce impaired smell test scores than those who passed the effort tests. Because effort exerts such major effects on olfactory test scores, it would be advisable in future studies to employ tests that are sensitive to response bias or exaggeration.

Based on the model outlined in the introduction, it was predicted that, when compared with a wide range of neuropsychological test scores, olfactory discrimination would be the strongest statistical predictor of brain injury severity in patients making a valid effort. This hypothesis was strongly supported because smell test scores loaded first in 14 out of 15 regressions predicting head injury severity (table 3). The group differences in tables 4 and 5 also show clear relationships between smell test scores, PTA, GCS and CT or MRI abnormalities. It has been known for a long time that the parts of the brain most likely to be damaged in a closed head injury include the anterior temporal lobes and the orbital frontal regions and that these same regions are associated with olfaction [13, 14]. Therefore, perhaps it is not surprising that one should find such strong relationships between olfactory discrimination scores and variables measuring the severity of head injury, including PTA duration. However, although the current results show that olfactory test scores are reduced in those with more severe brain injuries, they do not allow one to separate out the olfactory impairment resulting from central olfactory damage and that arising from peripheral damage, such as olfactory nerve shearing. Further research is needed to differentiate between loss of olfactory discrimination caused by shearing of the olfactory nerve at the cribriform plate vs 'damage to central brain regions' [13]. Jafek *et al.* [20] showed that presumed olfactory nerve shearing in five patients

with head injury led to degenerative changes and also some signs of incomplete regeneration in the olfactory neurons in the nasal epithelium, providing one possible marker of shearing of the olfactory nerve.

For heuristic purposes, it might be argued that olfactory nerve shearing is an all-or-nothing phenomenon. If so, then when shearing occurs it will result in a total loss of odour detection in the ipsilateral nostril, whereas damage to the olfactory cortex would not cause a total loss of odour detection. Based on these assumptions, one would conclude that olfactory nerve shearing is very rare in head injuries causing less than 1 day of PTA because only 0.7% of such cases had a total loss of olfaction in both nostrils (mean smell test score of zero) and only 3.8% had a score of zero in one nostril but not the other. However, based on current findings, one would assume that olfactory nerve shearing is likely to happen bilaterally in 20% of cases of head injury with at least 1 day of PTA and unilaterally in another 14.7%. If so, what effect would olfactory cortex damage be thought to have on olfactory test scores and how might one differentiate between the effects of olfactory nerve shearing and cortical damage?

First, damage to the olfactory cortex might explain why, in cases who do not display a total loss of detection in either nostril, smell test scores were still significantly lower in head injury patients with PTA of 1 day or more compared to those with lower levels of PTA. These patients could detect odours (presumably no shearing) but moderate or severe brain injuries lowered their olfactory discrimination ability.

Secondly, the current data show that zero smell test scores in both nostrils were associated with the most severe brain injuries. In these cases, therefore, one would expect concurrent damage to the olfactory cortex, for reasons outlined in the introduction. The practical problem one would then face is that, if shearing is present, causing the inability to detect odours, one would be unable to measure any impairment of olfactory discrimination that might otherwise have arisen from cortical damage. Undoubtedly, one would expect a few cases of olfactory nerve shearing with no central cortical damage as a result of frontal bone and ethmoid bone fractures with indicators of only mild brain injury, including normal brain CT scans. In cases of severe brain injury, however, olfactory nerve shearing might be expected to be superimposed on central cortical damage. If so, it may be argued that the currently observed correlations between smell test scores and measures of brain injury severity are actually manifestations of the association between central olfactory damage and damage in other brain regions, rather than representing a correlation between peripheral nerve shearing and generalized brain damage. In general, peripheral nerve damage does not predict cortical damage very well and one might assume that the same applies to olfaction.

The assumption that olfactory impairment reflects damage to or disruption of the ventromedial temporal lobes would be consistent with the observation of a steady decrease in olfactory discrimination scores with increasing levels of PTA duration (table 4). If both of these variables reflect damage or transient dysfunction in the anterior temporal regions, it would be easy to explain why they correlate. On the other hand, table 5 shows a discontinuity in the curve of GCS vs smell test scores. In the absence of PTA or CT abnormalities, GCS scores of 13 or greater were linked with normal smell test scores. Yet, olfactory discrimination was impaired to an equal degree whether the GCS scores were in the intermediate range (9–12) or the lowest range (3–8).

The model outlined in the introduction led to the prediction of no cases in the group with a GCS of 12 or less and no PTA, based on the assumption that it will take a less severe blow to the head to cause disruption of the anterior temporal lobes than it will to cause a loss of core consciousness, defined by a GCS in the moderate range. Therefore, it should be very unlikely to have a blow that is severe enough to cause loss of core consciousness without also causing some degree of PTA. On the other hand, there were many cases with recorded PTA and with a GCS of 13 or higher. These results reveal a clear dissociation between PTA and GCS, which would be more consistent with PTA and GCS having different causal mechanisms, rather than with PTA being a phase of recovery from a loss of core consciousness. Many brain injury rehabilitation clinics will have databases containing information about GCS and PTA duration in patients after closed head injuries. If the finding that all patients with GCS scores of 12 or less also have some PTA replicates, further studies might be warranted to investigate this phenomenon.

In the current study, neuropsychological impairment across many tests in patients with head injuries was the greatest in those with the lowest scores on the test of olfactory discrimination. However, smell test scores were not associated with overall neuropsychological impairment in the neurological patients, in whom the brain lesions were widely scattered, with no particular concentration of lesions in the anterior temporal lobes. Unilateral impairment was far more frequent in neurological patients than in people with head injuries. The presumed mechanism for impaired olfaction in most neurological patients was damage to the olfactory nerve. In some cases, unilateral impairment came from frontal craniotomy, which involved lifting one frontal lobe and severing the connections between one olfactory bulb and the olfactory epithelium, implying no presumed damage to the olfactory cortex. In others, the impaired olfaction arose from tumour, cyst or infection in the orbital frontal region, damaging one or both the olfactory nerves and possibly the olfactory cortex. Most of the other brain lesions in this patient group did not involve the ventromedial temporal or orbital frontal regions. Because impairment of olfactory discrimination probably resulted from discrete damage to the olfactory nerve in most cases, it had no predictive value with regard to the overall severity of neuropsychological impairment in neurological patients with heterogeneous diagnoses.

In this study, the tests of executive functioning did not correlate strongly with olfactory scores, unlike the findings reported by Callahan and Hinkebein [11]. However, the patients with anosmia in the current study had more severe head injuries and greater impairment of executive functions than those without anosmia. This would be consistent with Martzke *et al.*'s [21] report of a lower success rate in returning to work among head injury patients with anosmia. The most severe TBIs generally involve injuries to the ventromedial temporal lobes and orbital frontal regions, which could impair olfaction and also lead to behaviour changes such as impaired social judgement, inertia, amotivation and lack of spontaneity. On the other hand, these problems are not associated with mild head injury. The current results suggest that, in cases of mild head injury, the ratio of invalid-to-valid impaired olfactory discrimination scores is at least 5.5:1. Therefore, in mild head injury cases who produce impaired scores on an olfactory discrimination test and have a poor return to work, motivation might be an issue. Ideally, effort would be measured on the same day as olfactory testing, to determine if the impaired test scores are likely to be valid.

Most neuropsychological tests are traditionally regarded as being relatively insensitive to ventromedial temporal lobe and orbital frontal lesions. It may be argued that olfactory discrimination fills that gap. Varney *et al.* [22] reported orbital frontal hypofunction on PET scans in people selected for total anosmia after head injury. Also, Varney and Bushnell [23] correlated orbital frontal metabolism with reports of behaviour change by significant others. Yet, the patients in the latter studies were not classified as severe, and most were reported to be cases of mild-to-moderate brain injury, although the samples were small and highly selected. To optimize results from future studies of cortical metabolism and olfaction, it may be valuable to consider separately those cases with impaired olfactory test scores, who pass or fail effort tests. Clinically, before making inferences about brain function in people with mild head injury who are found to have impaired scores on tests of olfaction, it would be desirable to rule out the first and simplest hypothesis, which would be the possibility of invalid test scores due to poor effort.

This study cannot resolve the relative contributions of olfactory nerve shearing and olfactory cortex damage to impairment of olfactory discrimination. However, lesions in the ventromedial temporal regions, which are very likely in more severe brain injuries, might offer a simple explanation of why smell test scores are more related to head injury severity than all of the neuropsychological tests used in this study. Whatever the best explanation turns out to be, the data show clearly that olfactory discrimination is associated with head injury severity, as long as effort is controlled. Therefore, olfaction deserves more attention in studies of head injury than it has been given in the past, although clinically and in research studies care should be taken to identify and remove invalid olfactory test scores resulting from symptom exaggeration.

Acknowledgements

Acknowledgement and thanks for their help to Dr Keith Moore, anatomist, and Valerie Oxorn, medical illustrator.

References

1. Green, P. and Iverson, G. L.: Exaggeration of anosmia in 80 litigating head injury cases. *Archives of Clinical Neuropsychology*, **13**: 138, 1998.
2. Green, P. and Iverson, G. L.: Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *Neurorehabilitation*, **16**: 237–243, 2001.
3. Allen, L. M., Conder, R. L., Green, P. *et al.*: *CARB '97 Manual for the Computerized Assessment of Response Bias* (Durham, NC: CogniSyst, Inc.), 1997.
4. Green, P., Allen, L. M. and Astner, K.: *The Word Memory Test: a user's guide to the oral and computer-administered forms, US Version 1.1* (Durham, NC: CogniSyst, Inc.), 1996.
5. Green, P., Iverson, G. L. and Allen, L.: Detecting malingering in head injury litigation with the Word Memory Test. *Brain Injury*, **13**: 813–819, 1999.
6. Iverson, G. L., Green, P. and Gervais, R.: Using the Word Memory Test to detect biased responding in head injury litigation. *Journal of Cognitive Rehabilitation*, **17**: 4–8, 1999.
7. Allen, L. M. and Green, P.: Severe traumatic brain injury performance on CARB and the WMT. *Supplement to the CARB and WMT manuals* (Durham, NC: CogniSyst), 1999.
8. Green, P. and Allen, L. M.: Performance of neurological patients on CARB and the WMT. *Supplement to the CARB and WMT manuals* (Durham, NC: CogniSyst), 1999.
9. Doty, R. L., Yousem, D. M., Pham, L. T. *et al.*: Olfactory dysfunction in patients with head trauma. *Archives of Neurology*, **54**: 1131–1140, 1997.

10. Levin, H. S., High, W. M. and Eisenberg, H. M.: Impairment of olfactory recognition after closed head injury. *Brain*, **108**: 579–591, 1985.
11. Callahan, C. D. and Hinkebein, J.: Neuropsychological significance of anosmia following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, **14**: 581–587, 1999.
12. Ogawa, T. and Rutka, J.: Olfactory dysfunction in head injured workers. *Acta Otolaryngol Supplement*, **540**: 50–57, 1999.
13. Costanzo, R. M. and Zasler, N. D.: Epidemiology and pathophysiology of olfactory and gustatory dysfunction in head trauma. *Journal of Head Trauma Rehabilitation*, **7**: 15–23, 1992.
14. Courville, C. B.: Mechanisms of cerebral contusion. In: *Pathology of the Nervous System*, 2nd edn (Mountain View California: Pacific Press), p. 292, 1945.
15. Moore, K. L. and Dalley, A. F.: *Clinically orientated anatomy*, 4th edn (Baltimore, MD: Lippincott, Williams and Wilkins), 1999.
16. Smith, C. G.: *Basic Neuroanatomy* (Toronto: University of Toronto Press), 1961.
17. Damasio, A.: *The feeling of what happens: Body and emotion in the making of consciousness* (San Diego: Harcourt Inc.), 1999.
18. Beers, M. H., Berkow, R., Bogin, R. M. *et al.*: *The Merck Manual of Diagnosis and Therapy*, 17th edn (New Jersey: Merck Research Laboratories), 1999.
19. Rohling, M. L., Green, P., Allen, L. M. *et al.*: Effect sizes of impairment associated with symptom exaggeration versus definite traumatic brain injury. *Archives of Clinical Neuropsychology*, **15**: 839, 2000.
20. Jafek, B. W., Eller, P. M., Esses, B. A. *et al.*: Post traumatic anosmia: ultrastructural correlates. *Archives of Neurology*, **46**: 300–304, 1989.
21. Martzke, J. S., Swan, C. S. and Varney, N. R.: Post-traumatic anosmia and orbital frontal damage: neuropsychological and neuropsychiatric correlates. *Neuropsychology*, **5**: 213–225, 1991.
22. Varney, N. R., Pinkston, J. B. and Wu, J. C.: Quantitative PET findings in patients with posttraumatic anosmia. *Journal of Head Trauma Rehabilitation*, **16**: 253–259, 2001.
23. Varney, N. R. and Bushnell, D.: NeuroSPECT findings in patients with post-traumatic anosmia: a quantitative analysis. *Journal of Head Trauma Rehabilitations*, **13**: 63–72, 1998.