Pergamon

0028-3932(95)00106-9

Neuropsychologia, Vol. 34, No. 4, pp. 247–261, 1996

Printed in Great Britain
0028-3932/96 $15.00+0.00

Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal lobe damage

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(*Received* 23 *November* 1994; *accepted* 20 *June* 1995)

Abstract—Impairments in the identification of facial and vocal emotional expression were demonstrated in a group of patients with ventral frontal lobe damage who had socially inappropriate behaviour. The expression identification impairments could occur independently of perceptual impairments in facial recognition, voice discrimination, or environmental sound recognition. The face and voice expression problems did not necessarily occur together in the same patients, providing an indication of separate processing. Poor performance on both expression tests was correlated with the degree of alteration of emotional experience reported by the patients. There was also a strong positive correlation between the degree of altered emotional experience and the severity of the behavioural problems (e.g. disinhibition) found in these patients. A comparison group of patients with brain damage outside the ventral frontal lobe region, without these behavioural problems, was unimpaired on the face expression identification test, was significantly less impaired at vocal expression identification and reported little subjective emotional change. The expression identification deficits in ventral frontal patients may contribute to the abnormal behaviour seen after frontal lesions, and have implications for rehabilitation.

Key Words: face expression; emotion; face recognition; frontal cortex; orbitofrontal cortex.

Introduction

Patients with frontal lobe damage, produced for example by closed head injury or cerebrovascular accident (CVA), may show altered emotional and social behaviour [18, 34]. The changes can include disinhibited or socially inappropriate behaviour, impulsiveness, inappropriate sexual advances, euphoria, jocularity, irritability and misinterpretation of people's moods. In humans, there is little understanding of the processing which normally takes place in the affected region, or even of exactly which part of the frontal lobe is crucial for the changes. On the other hand, there have been many advances in the last few years in understanding the neural basis of emotion in non-human primates, and the location, connections, and functions of the frontal region involved in emotion, which these studies indicate is in the orbitofrontal cortex in the ventral part of the frontal lobe. It is the aim of the research described here to take this fundamental research and to investigate its implications for understanding and treating the symptoms in these patients and for their rehabilitation.

The analysis of one part of the frontal cortex, the orbitofrontal cortex, suggests that one way that it is important in emotions is because it is involved in emotion-related learning [21, 25, 26, 28]. For example, the learning deficits associated with damage to the orbitofrontal cortex in non-human primates include impaired extinction and impaired visual discrimination reversal. The impairment produced by orbitofrontal damage in extinction and reversal consists of continued responding for the previously rewarded stimulus. These two deficits can be understood as a failure to break, or adjust, previously learned associations between stimuli and primary reinforcers. In modern approaches to emotion, emotions are often considered to be states elicited by rewarding and punishing stimuli [21, 26], so that any failure to alter behaviour when the reinforcement value of environmental stimuli changes will lead to

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inappropriate emotional and social behaviour. In tests of patients with ventral frontal lobe damage with such emotional and social problems, deficits in the extinction and reversal of visual discrimination tasks have been found, and difficulty with such correction of behaviour to environmental reinforcers may at least partly underlie the behavioural problems of such patients [29].

The question then arises of the nature of the environmental stimuli which are normally detected and used as reinforcers to alter behaviour. There are certainly many such signals, but one set of such signals to which we must react continuously in normal social and emotional behaviour is provided by the expression on the face or in the voice. It is known that there are areas in the primate temporal visual cortex where faces are represented, and there is a specialised population of neurons concerned with face expression [11, 22–24, 27]. There is a direct projection from these temporal visual cortex areas into the orbitofrontal/inferior frontal convexity cortex [1]. In addition there is a route via the amygdala, where face-responsive cells are also found [17], to the orbitofrontal cortex [21–24]. This suggests that information about faces, for example about face expression, may reach the orbitofrontal cortex, where it could be used to provide reinforcing signals used when social and emotional behaviour must be altered. Consistent with this, Rolls and Critchley (unpublished observations) and Wilson *et al.* [36] have found cells in the orbitofrontal cortex which do respond to faces. Further, damage to this region reduces the emotional response of monkeys to emotion-provoking stimuli such as faces, snakes and food [21]. Inputs from the superior temporal auditory association cortex also reach the orbitofrontal cortex [1, 19].

Because of this evidence, we performed the investigation described here in order to determine whether patients with damage to the ventral part of the frontal lobe (which includes the orbitofrontal and inferior frontal convexity regions described above) have impaired responses to social and emotional signals derived from face and voice expression. If so, there would be implications for the management and rehabilitation of such patients. A subsidiary aim of this investigation was to determine whether there were dissociations between the ability to respond to these different (face and vocal) expression signals in such patients, for this would indicate not only localisation of function, but would also have implications for rehabilitation.

In previous studies in humans investigating changes in emotion-related processing which have focused on the critical brain regions rather than on hemisphere differences, some impairments associated with frontal lesions in recognising emotional expression from the face, the voice or from gesture have been found [15, 16]. More recently a PET study consistent with these studies showed that, during an expression-matching test in which normal subjects took part, the inferior frontal gyri bilaterally (as well as the right anterior cingulate) were activated [10]. Hopf *et al.* [12] found that patients with left frontal lesions were impaired at producing facial expressions of emotion, though unimpaired at non-emotional facial movements. Damasio *et al.* [7] found that patients with bilateral frontal lesions, who showed sociopathic behaviour, failed to respond autonomically to emotionally-charged pictures. However, with the exception of some single case studies by Ross and his colleagues [30, 31], very little attention has been paid to the possible relationship between impairments in the identification of emotional stimuli, alterations of subjective emotional experience, and the behavioural changes produced by frontal lobe damage.

One of the aims of the present study was to measure in the same group of patients their responses to face and voice expression, their changes in the experience of emotions following the brain damage and alterations in their behaviour. The behavioural changes in these patients have been described more fully elsewhere [29] but are analysed here in relation to impairments in emotional expression identification and to subjective emotional changes.

Methods

Facial expression identification and related tests

Photographs of the following expressions were used: sad, angry, frightened, disgusted, surprised, happy and neutral, taken from the Ekman and Friesen [8] series. There were four examples of each of these seven expressions made by different subjects, making a total of 28 trials.* One example of each expression occurred in the first seven trials. Thereafter the order of expression was randomised, with the constraint that no expression occurred twice or more in succession, and no subject appeared making an expression on any two consecutive trials. Patients were shown one photograph at a time. They were asked to choose from a list the adjective best describing the facial expression in each photograph. A list of the seven adjectives was presented underneath each photograph, in a vertical array and in a different order on each successive trial. Each photograph and list was presented for unlimited duration and no feedback was given to the patient about whether the choice was correct. Patients who were unsure or used an adjective not on the list to describe the expression were asked to choose whichever adjective on the list seemed most appropriate.

The mean and standard deviation of the percentage correct on the face expression identification test for 11 normal subjects (of a comparable age range as that of the patient groups) was $87 \pm 8.9\%$, giving a 5th centile cutoff of 72% correct.

To determine whether effects found with the above test were in some cases specific to face expression identification, patients

*The photographs used were as follows: sad (54A, 57B, 42Practice, 43P); angry (42A, 57A, 26P, 27P); frightened (20D, 29A, 12P, 13P); disgust (29B, 30A, 16P, 21P); happy (43C, 44A, 29P, 31P); neutral (32B, 45C, 53P, 54P).

were also tested for their ability to recognise a series of faces after a delay (Warrington facial recognition test [35]).

Vocal expression identification and related tests

Patients listened to a tape of emotional sounds corresponding to the following emotions: sad, angry, frightened, disgusted, puzzled, contented and neutral. As in the facial expression test, they were asked to name each sound from a list.

Previous studies of the identification of vocal emotion have generally used neutral sentences spoken in different tones of voice. Some exceptions exist however, in which some non-verbal sounds were used with normal subjects, and in which a left-ear advantage was found, e.g. laughing, crying, moaning, sighing, sneezing and coughing [14], and crying, shrieking, and laughing [6]. In the present study non-verbal expressions (of approximately the corresponding emotions as were used in the facial expression identification test) were used instead of emotionally modulated speech. This was because producing these sounds (which patients were required to do in a later session) was considered easier for patients than reading neutral sentences in different tones of voice.

A tape of emotional sounds corresponding to the following emotions: sad, angry, frightened, disgusted, puzzled, contented and neutral was prepared with the help of three normal subjects: a man, a woman and a child. These three volunteers were asked to make whatever sounds they felt best expressed these emotions, with the following limitations:

- that the sounds should all be voiced and of approximately equal volume and length;
- that, although two sounds were allowed, they should avoid inserting consonants (which could make these sounds more easily recognizable through a quasi-verbal route, e.g. "ugh" or "yuck" for disgust);
- that no sound should contain repetitions which itself might disambiguate the sound (e.g. they should not produce a series of sobs for sad).

For neutral they were asked to hum briefly on one note.

Each volunteer produced two sounds for each emotion. All of the different emotions occurred once in the first seven trials: thereafter they were presented randomly, with the constraint that no emotion should appear more than twice in succession. As far as was possible, given the randomised order of emotions, the three different voices appeared in rotation.

As in the facial expression test, patients were asked to name the emotion expressed by each sound from a vertically-arrayed list in which the order of emotions was altered on each trial. When patients were in doubt, they were asked to name whichever emotion seemed the most appropriate. No feedback was given.

The mean and standard deviation of the percentage correct on the vocal expression identification test for 18 normal subjects was $80 \pm 10.3\%$, giving a 5th centile cutoff of 63.1% correct.

To determine whether effects found with the above test were in some cases specific to voice expression identification, patients were also tested for their ability to identify non-emotion-related sounds of comparable complexity, as follows.

In a *voice discrimination test* patients were required to discriminate between tape-recorded female voices speaking the names of different months of the year. There were 32 trials. On 16 trials the same voice spoke the names of a series of 6 months, read at the rate of 1 month per sec, with a 1 sec pause after the first 3 months. On the other 16 trials a different voice spoke the second 3 months, again after a 1 sec pause. The voices of a total of 15 different normal female subjects were recorded and used to construct the test tape. Trials consisting of same and of different voices occurred in random order, with the constraint that neither type of trial occurred more than three times in succession. Patients were asked to listen carefully to the names of the 6 months spoken on each trial, and to say whether they thought the first 3 months were spoken by the same voice as the second 3 months, or by a different voice. No feedback was given. The mean and standard deviation of the percentage correct for a group of 15 normal subjects on this test were $76 \pm 8\%$, giving a 5th centile cutoff of 61% correct.

In an *environmental sounds test* patients were required to name a series of 44 recorded sounds that were neither emotional nor vocal (such as water dripping, a vacuum cleaner, etc.). Patients were asked to listen to the sounds and name them. In tests with a group of nine normal subjects, it was found that the mean and standard deviation of the percentage correct were $87 \pm 3\%$, giving a 5th centile cutoff of 82% correct.

In a later session, some patients were asked to imitate the sounds occurring on the vocal expression identification test, and then to identify the sounds as before. The imitations were recorded and subsequently played to normal subjects who were asked to identify the emotion the patient was attempting to imitate.

Subjective emotional change questionnaire

To obtain evidence on whether there was a relation between problems in identifying face expression and/or voice expression, and subjective emotional changes since their illness or injury, the following questionnaire was completed by the patients.

Patients were asked whether they had experienced, since their illness or injury, any general change in their ability to experience emotion. They were then questioned about any change in the intensity or frequency of the following emotions:

Sadness (or regret)
Anger (or frustration)
Fear (or anxiety)
Disgust (in the sense of physical revulsion)
Excitement or Enjoyment.

These emotions were chosen because they correspond to the main emotions which occurred in the tests of facial and vocal expression identification in which the patients also took part. Examples of the kinds of situations likely to provoke these emotions were described to the patients. (For 'sadness' patients were asked, for example, how they felt watching sad films; for 'disgust', they were asked how they would feel if they discovered maggots wriggling in some food they were about to eat. For the positive emotions, they were asked whether they still took the same pleasure in or had the same appreciation of various aspects of life as before, including social (e.g. whether they still felt affection for those close to them), aesthetic (e.g. music), natural beauty, sport, hobbies, etc.).

Positive scores were given for changes in either direction (increase or decrease) in any of the five emotions. A slight change scored 0.5; a definite change scored 1.0; and a very extreme change or the complete absence of an emotion scored 1.5. (As there were five emotions, the score for any patient was in the range 0–7.5.)

Particularly because of its potential relevance to rehabilitation, patients were also asked whether they were aware of any difficulty in the production of these expressions, either facially or vocally. In addition they were asked whether other people misinterpreted their moods or feelings and whether they had difficulty interpreting the moods and feelings of others. Finally, patients were asked whether they were aware of becoming more impulsive, or had noticed any difficulty in controlling their behaviour, since their illness or injury.

Behaviour questionnaire

In order to investigate whether there was a relationship between problems in identifying face expression and/or voice expression, subjective emotional change, and behavioural changes associated with their illness or injury, a questionnaire was completed by the member of the rehabilitation team who saw the patients regularly and knew them well, so that they could rate behaviour in a wide variety of situations, including meal-time, occupational therapy and behaviour on the ward. The questionnaire is described in detail elsewhere [29]. It was designed to provide a measure of the kinds of behaviour problems, especially social, which occurred in these patients.

Subjects

Twenty-three patients attending the Rivermead Rehabilitation Centre, Oxford, as either impatients or outpatients were studied. Most had suffered head injury or stroke. The sites of the brain damage in these patients were determined by magnetic resonance imaging (MRI) whenever possible, and when not possible, by CT scans, and are indicated, together with clinical details, in Table 1. Localisation was taken from the formal brain scan reports, and from detailed study of the scans, and patients were divided into two groups on the basis of the identified location of damage, as indicated in Table 1. (The rationale for investigating a group of patients with ventral frontal damage is given in the Introduction.) Twelve had lesions which included damage relating to the ventral (i.e. inferior) parts of the frontal lobes (ventral frontal group), and 11 did not have damage to this region (non-ventral group). The damage in the ventral group consisted of identifiable damage to the orbital or ventrolateral frontal cortex, or connected white matter, and where 'frontal' is used in the upper part of Table 1, it refers to this region. The lesions were located between the anterior 39 and anterior 65 mm planes (in the AC–PC coordinate system). An example of the lesion in one of the patients, case 11, is shown in Fig. 1. Case 11 was a patient with head injury, and in addition to diffuse atrophy, the scan evidence illustrated in the right ventral frontal region but not in the left shows that lesions in such patients can be focal in that for example they may be present on one side but not in the corresponding region of the other. This lesion was centred at 43 mm anterior. The 'ventral frontal' group here thus refers to a group of patients with damage related to the ventral part of the frontal lobes. What was common to this group was damage to the ventral frontal region, although the damage was not necessarily restricted only to this brain region in all the patients. The damage in the non-ventral group was either outside the frontal lobes, or, in two patients (cases 5 and 6) was in the dorsolateral frontal region, and in one patient (case 8) involved the posterior lateral surface of the frontal lobe, as shown in Table 1. The time since the brain damage was typically 6 months to 2 years. No patient was included unless

Table 1. Clinical information

Patient case no.	Sex	Age	Pathol.	Scan	Lesion site
Ventral frontal					
1	M	62	CVA	MRI	Right posterior frontal
2	M	72	CVA	MRI	Right orbitofrontal and temporal
3	M	42	HI	MRI	Right frontal and cerebellar
4	M	22	HI	MRI	Left frontal
5	M	35	HI	MRI	Bilateral frontal and right temporal
6	M	30	CYST	MRI	Right frontal and left hippocampus
7	M	38	HI	MRI	Right frontal and temporal
8	F	45	CVA	CT	Bilateral frontal
9	M	42	CVA	CT	Left frontal
10	M	28	HI	CT	Bilateral frontal
11	F	30	HI	MRI	Right orbitofrontal
12	M	51	CVA	MRI	Right MCA: ventral frontal involvement
Non-ventral					
1	M	54	CVA	CT	Left parietal
2	M	63	CVA	CT	Right temporal and right basal ganglion
3	M	56	CVA	MRI	Right parietal
4	M	52	CVA	MRI	Left internal capsule infarct
5	M	38	HI	MRI	Right MCA and ACA: dorsal frontal damage
6	M	22	HI	MRI	Dorsolateral bilateral frontal
7	M	26	HI	MRI	Right internal (and external) capsule and right striatum
8	F	45	CVA	CT	Right temporal and posterior lateral surface of right frontal lobe
9	M	46	CVA	CT	Right basal ganglion and right striatum
10	M	67	CVA	CT	Right side of pons
11	F	52	CVA	CT	Left internal capsule haematoma

Ventral frontal: Patients with damage to the ventral part of the frontal lobes (see text). A further indication of where in the ventral part of the frontal lobe the lesion was located is included under lesion site.

Non-ventral: Patients with damage in brain regions other than the ventral parts of the frontal lobes. (Cases no. non-ventral 5 and 6 had some frontal damage but this was confined to the dorso-lateral surface, and case 8 non-ventral had some damage to the posterior lateral surface of the frontal lobe (see text).)

CVA: Cerebrovascular accident; HI: head injury; Cyst: colloid cyst with acute hydrocephalus.

MCA: Middle cerebral artery.

ACA: Anterior cerebral artery.

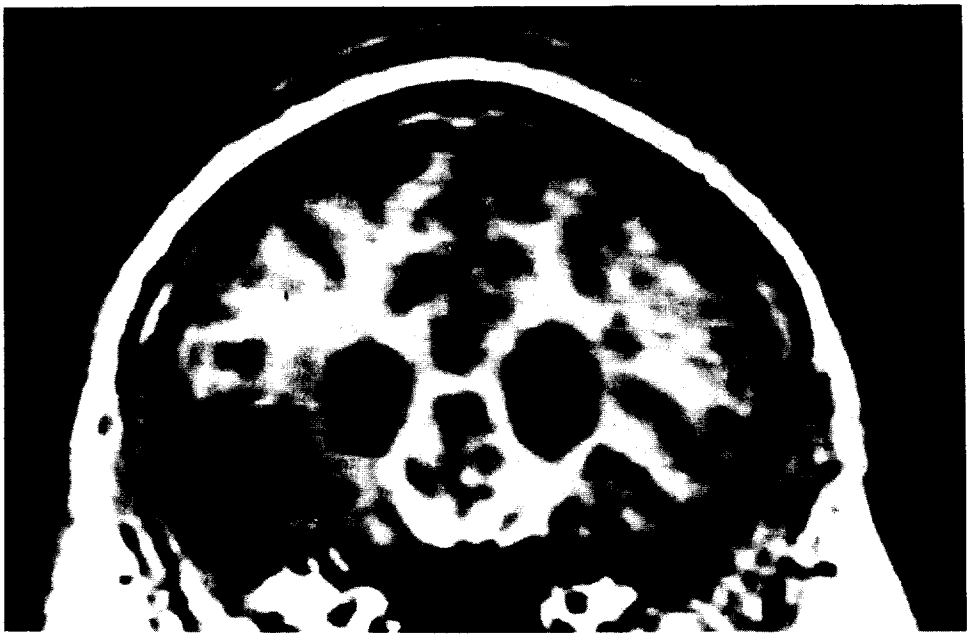

Fig. 1. An MRI scan showing the lesion in one of the patients, case 11 of the ventral group. The lesion (dark patch on the left of the figure) was centred at 43 mm anterior in the right orbitofrontal region (see text).

their language comprehension was adequate for understanding the instructions for the tests and their expressive language was adequate for answering questions about their emotions and behaviour. For this reason there was a preponderance of patients with right-sided damage. All the patients included in this study had severe disability as evidenced by the need to be at the Rivermead Rehabilitation Centre. The etiology of the brain damage was varied, as were the disabilities of the patients. Being disabled itself might potentially account for some emotional and behavioural changes, and this issue is addressed when the results are described. Nevertheless, none of the patients in either group suffered from any general cognitive deterioration.

Evidence on the neuropsychological profile was available for the majority of the patients. Verbal IQ for all patients in this study was in the average range. Additionally, all three patients (cases 1, 8 and 9) in the ventral frontal group tested on the Tower of London [33] performed normally. In addition, verbal fluency was normal in four of the five patients with ventral frontal damage tested (cases 2, 5, 6 and 9), and was impaired in one (case 1). Paired associate learning was average or above average in most patients in both groups, and there was no significant difference between the two groups (for further details see Rolls *et al.* [29]). The investigation had local ethical committee approval and patients consent was obtained.

Results

Facial expression and vocal expression identification

The percentage correct in the facial and vocal expression identification tests are shown for the patients in the two groups in Table 2. The values in parentheses show the number of standard deviations above or below the mean of the normal subjects in these tests, which are provided in the Methods section. (In the following 'impaired' is used to refer to performance more than 1.64 standard deviations below the mean [i.e. $P<0.05$, the 5th centile], and 'severely impaired' refers to performance more than 1.96 standard deviations below the mean [$P<0.01$].) It is shown that for the facial expression identification test, nine of the 12 patients in the ventral frontal group were severly impaired. In contrast, only one of the 11 patients in the non-ventral group was severely impaired, with one other impaired at the 0.05 level. A statistical comparison of the two patient groups and the 11 normal subjects showed significant group differences [Kruskal–Wallis analysis of variance, 2 d.f., $H=14.1$, $P<0.01$]. A subsequent comparison of the different groups on the facial expression identification test showed that the ventral group was more impaired than the non-ventral group [Mann–Whitney $U=22$, $P<0.01$]. (The ventral frontal group was also significantly impaired relative to the normal group [$U=11$, $P<0.001$], and the non-ventral group was not significantly different from the normal [$U=39.5$, $P=0.16$].) Some of the patients with face expression identification deficits had unilateral damage (see Tables 1 and 2, e.g. cases 1 and 12).

It is shown that for the vocal expression identification test, eight of the 11 patients in the ventral frontal group who performed the test were severely impaired [i.e. at the $P<0.01$ level]. In contrast, none of the eight patients in the non-ventral group was severely impaired (although four of these eight patients showed a milder impairment significant at the 0.05 level). A statistical comparison of the two patient groups and the 18 normal subjects showed significant group differences [Kruskal–Wallis analysis of variance, 2 d.f., $H=19.8$, $P<0.0001$]. A subsequent comparison of the different groups on the vocal expression identification test showed that the

Table 2. Vocal and facial expression identification in the ventral frontal and non-ventral groups

Case No.	Behav.	Subj. emot. change	Facial expr. % Correct (S.D.)	Vocal expr. % Correct (S.D.)
Ventral frontal				
1	6.0	—	29 (−6.5)†	42 (−3.7)†
2	4.0	2.0	84 (−0.4)	30 (−4.8)†
3	6.0	7.5	60 (−3.1)†	36 (−4.9)†
4	7.5	4.5	60 (−3.0)†	54 (−2.5)†
5	8.5	7.0	58 (−3.2)†	39 (−4.0)†
6	5.0	1.5	75 (−1.3)	67 (−1.3)
7	6.0	5.0	67 (−2.3)†	58 (−2.1)†
8	7.0	2.5	54 (−3.7)†	—
9	4.0	1.5	83 (−0.4)	81 (+0.1)
10	5.0	4.0	67 (−2.2)†	60 (−1.9)*
11	4.5	6.5	40 (−5.3)†	53 (−2.6)†
12	3.0		38 (−5.6)†	43 (−3.5)†
Medians	5.5	4.3	60	53
Non-ventral				
1	0.0	0.5	79 (−0.9)	
2	2.5	1.0	83 (−0.4)	
3	0.5	0.0	83 (−0.4)	61 (−1.8)*
4	0.0	2.0	75 (−1.4)	61 (−1.8)*
5	0.0	1.5	71 (−1.8)*	67 (−1.2)
6	2.0	1.0	92 (+0.6)	75 (−0.5)
7	2.5	1.0	75 (−1.4)	61 (−1.8)*
8	0.0	2.5	96 (+0.1)	78 (−0.2)
9	0.5	1.0	67 (−2.3)†	
10	1.0	1.5	79 (−0.9)	72 (−0.7)
11	0.5	1.0	83 (−0.4)	61 (−1.8)*
Medians	0.5	1.0	79	64

*Scores which fall below the 5th centile of the normal distribution, i.e. S.D. < -1.64 (impaired).
†Scores which fall below the 1st centile of the normal distribution, i.e. S.D. < -1.96 (severely impaired).
Bahav: Behaviour questionnaire; Subj. emot. change: Subjective emotional change questionnaire; Facial expr: Facial expression identification test; Vocal expr: Vocal expression identification test; S.D.: number of standard deviations above (+) or below (−) the means for normal subjects.

ventral group was more impaired than the non-ventral group [Mann–Whitney $U = 12$, $P < 0.01$]. (The ventral frontal group was also very significantly impaired relative to the normal group, $P < 0.001$, with the non-ventral group showing some difference from the normal group, $U = 22$, $P < 0.01$.)

It is shown that eight of the 12 patients in the ventral frontal group were impaired on *both* the facial and the vocal expression tests, whereas in the non-ventral group, none of the 11 patients was impaired on both tests. A notable finding was that patients impaired on *both* expression identification tests had high subjective emotional change scores (range 4–7) (see later and Table 2). By contrast, patients impaired at only one expression identification test had much lower subjective emotional change scores (range 0–2).

Although most patients who performed poorly at the one of the expression identification tests (facial and vocal) also performed poorly at the other test (see Table 2), there were some exceptions. For example, patient 2 in the ventral frontal group performed at 4.8 standard deviations below the mean at the vocal expression identification test, but performed normally at the face expression identification test. (As described below, this patient could imitate accurately the actual sounds from the vocal expression test, even those he could not identify.) Further examples of patients who performed differently on the vocal and face expression identification tests are shown in Table 2.

Comparison tests on face and voice discrimination

Some patients (ventral frontal cases 3, 4 and 5) who were impaired at facial expression identification were also impaired at a test which required discrimination between faces, the Warrington facial recognition test. However, there were some interesting exceptions. Cases 1 and 7 from the ventral frontal group performed normally on the face recognition test (with scores at the 25th and 50th centiles, respectively), but were severely impaired at the face expression identification test (with results 6.5 and 2.3 standard deviations respectively below the normal mean). Conversely, cases 2 and 6 from the ventral frontal group and case 2 from the non-ventral group were impaired at the face recognition test

(i.e. below the 5th centile), but were unimpaired at the face expression identification test (see Table 2). This was very striking for example with case 2 of the non-ventral group, who was 3.2 standard deviations below the mean ($P<0.002$) on the face recognition test, but only 0.4 standard deviations (n.s.) below the mean on the face expression identification test. His difficulty with face recognition was not part of a general cognitive impairment, in that his score on the PALT test was 0.9 standard deviations above the mean. Thus, in line with previous studies [13, 38], the ability to identify facial expression and to recognise the identity of faces may be doubly dissociable.

Similarly, some of the patients in the ventral group (cases 3, 4 and 11) who performed very poorly on the vocal expression identification test (as shown in Table 2) were nevertheless unimpaired on the voice discrimination test, with scores above or just below the mean (with standard deviations respectively 0.3, 1.1 and −0.5 from the mean). (Since the voice discrimination test was performed some months after the vocal expression identification test, the latter was repeated on the same day as the voice discrimination test. The results for this repeated test on the vocal expression identification test (expressed in S.D.) were now case 3: −2.7, case 4: −1.8, and case 11: −2.1, which represents some improvement, perhaps due to practice effects, for all of the three patients. However cases 3 and 11 still scored below the 5th centile cutoff. Also, although case 4's score (−1.8) was now just above this cutoff, his new score should be contrasted with his superior performance on the voice discrimination test, in which his score was 1.1 above the mean.)

Additionally, some of the patients who were impaired at the vocal expression identification test were also not impaired at the environmental sound identification test (cases 3, 4, 10 and 11 in the ventral frontal group). Particularly striking was the performance of case 3 in the ventral frontal group, who scored 4.9 standard deviations below the mean on the vocal expression identification test, but just above the mean on the environmental sounds recognition test.

Imitation and production of vocal emotion sounds

The nature of the patients' deficit on the vocal expression test was further investigated by determining whether patients could imitate sounds that they could not identify. (The rationale was to analyse where in information processing the impairment in these patients might operate.) For cases 2 and 3 of the ventral frontal group (who performed at 30 and 36% correct on the vocal expression identification test), the imitations these patients made of those sounds *they could not identify* were sufficiently good that three normal subjects could identify 42 and 60% of them correctly (chance was 14% correct).

The patients were generally well able in clinical testing to produce, on request, sounds expressing the emotions from the voice expression identification test. This was true even of those sounds they had particular difficulty in identifying. Case 2, for example, never identified 'sad'. He imitated the sound repeatedly so well, however, that in formal tests all three normal subjects correctly identified all of these imitations as sad, even though case 2 was still unable to identify the sound he had imitated. In a later session, when asked to produce 'sad' sounds, he then made sounds which, as judged by four normal subjects, were readily recognised as 'sad', and were indistinguishable from his accurate imitations. These excellent productions were made by case 2 in spite of never having been given feedback in the vocal expression identification test.

Behavioural changes

On the behavioural changes questionnaire the commonest types of abnormality, present in various combinations, in at least nine of the 12 patients, were:

(1) Disinhibited or socially inappropriate behaviour (extreme for five of the patients).
(2) Misinterpretation of other people's moods.
(3) Impulsiveness.
(4) Unconcern or underestimation of the seriousness of his or her condition.
(5) Lack of initiative.

None of the patients in the non-frontal group was disinhibited or socially inappropriate. Their total scores were significantly lower than in the ventral frontal group, with most patients scoring zero (see Table 2; $U=0$, $P<0.0001$). There was in fact no overlap between scores in the two groups. (Further details are given by Rolls *et al.* [29]. Each of 12 questions could receive a score of 0–1.5.)

All of the patients who were disinhibited or socially inappropriate nevertheless had at least some insight into their behavioural problems. All who were questioned agreed that they were impulsive or that they had problems in controlling their behaviour. All were able to give good examples to illustrate this, which had not necessarily been witnessed by staff. The patients demonstrated a surprising degree of insight into the social repercussions of their behaviour, and in some cases, a considerable degree of regret. In particular, patients were aware of being too outspoken or too hasty in their response to what others said to them. Examples are provided in Appendix 1c.

Subjective emotional change questionnaire

Patients in the ventral frontal group scored far higher on the subjective emotional change questionnaire than did patients in the non-ventral group, as can be seen in Table 2 [$U=8$, $P<0.001$]. As shown in Table 3, 10 of

Table 3. Subjective emotional change

Case No.	Sad	Angry	Frightened	Disgusted	Excited (enjoyment)	Total change
Ventral frontal						
2	0	0	−1.0	0	−1.0	2.0
3	ABS	−1.5	−1.5	ABS	1.5	7.5
4	ABS	1.5	−1.5	0	0	4.5
5	1.5	1.5	ABS	ABS	−1.0	7.0
6	1.0	—	0	0	−0.5	1.5
7	1.5	0.5	−1.5	0	1.5	5.0
8	1.5	0.5	−0.5	0	0	2.5
9	0	ABS	0	0	0	1.5
10	ABS	−1.0	−1.5	—	0	4.0
11	−1.0	−1.5	−1.0	ABS	1.5	6.5
Non-ventral						
1	0	0	0.5	0	0	0.5
2	0	1.0	0	0	0	1.0
3	0	0	0	0	0	0.0
4	0.5	0	0.5	0	1.0	2.0
5	0	1.0	0.5	0	0	1.5
6	0	0.5	0.5	0	0	1.0
7	0	0.5	−0.5	0	0	1.0
8	1.0	−0.5	0.5	0	0.5	2.5
9	0.5	0.5	0	0	0	1.0
10	0	0	0.5	0	1	1.5
11	0	−0.5	0.5	0	0	1.0

—: Not questioned; 0: no change; 0.5: slightly increased; 1.0: increased; 1.5: greatly increased; −0.5: slightly decreased; −1.0 decreased; −1.5: greatly decreased.
ABS: Absent (this emotion was no longer experienced, scored as −1.5).
Total: The sum of the absolute values of the emotional changes.

the 11 patients in the non-ventral group reported an increase of the negative emotions sadness, anger and fear—changes that might be expected as a result of the limitations and difficulties imposed by their handicap [9]. Table 3 also shows that in the ventral frontal group by contrast, many of the changes were in the opposite direction to what might be expected. In particular, with the exception of case 6, all patients in the ventral frontal group reported a reduction or complete absence of the ability to feel one or more of the negative emotions. Examples of the decrease in the ability to feel sadness and fear, in some cases accompanied by increased aggression, described by some of the patients in the ventral frontal group are provided in Appendix 1a. Table 3 reveals that the two groups did in fact differ most in their experience of fear. Whereas eight of the 10 patients with ventral frontal damage who answered the questionnaire reported a diminution, usually marked, or a complete absence of fear since their illness or injury, this emotion was reported as slightly increased in most of the patients in the non-ventral group. Three of those patients in the ventral frontal group who reported an absence of or reduction in fear also reported an inability to feel disgust (in the sense of physical revulsion). They claimed that they would feel nothing at all if they found, for example, maggots wriggling in food they were about to eat, but would just throw it away. Disgust was unaltered in all patients in the non-ventral group. Some of the patients in the ventral frontal group also either described, or displayed, an inability to empathise with the sufferings of others which would result from actions they were planning. They also described themselves as far less concerned about what other people thought of them (Appendix 1b).

In general the patients in the ventral frontal group were surprisingly definite and clear about which particular emotional changes they had experienced. They spontaneously gave examples and were well able to contrast how they felt now with how they had felt before injury or illness. Case 3, for example, volunteered that, of late, the emotions of anger and love had been beginning to "come back a little". They also showed insight into the repercussions of their behaviour (Appendix 1c).

Four of the patients in the ventral frontal group with reduced or absent experience of one or more of the negative emotions additionally reported some reduction in positive emotion (and/or an increase in sadness), as shown in Table 3 (cases 2, 5, 7 and 8). Three of the patients in the ventral frontal group showed extreme increases (+ 1.5) in positive emotion (see Table 3). (These patients often reported feeling happy all the time.)

Relation between the findings from the emotional and behavioural changes questionnaires

A significant positive correlation was obtained between the scores on the behavioural questionnaire and scores on the subjective emotional change questionnaire

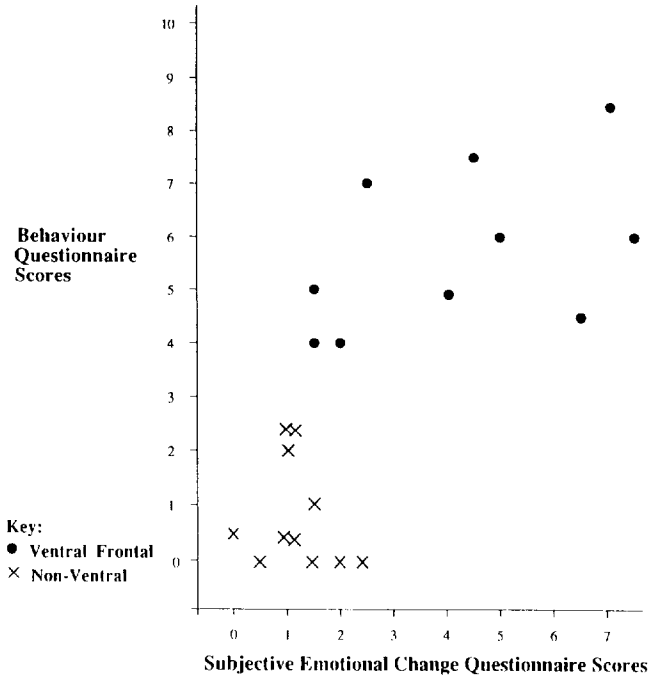

Fig. 2. Relationship between scores on the behaviour questionnaire and the scores on the emotional change questionnaire.

(Spearman rank correlation [rho: 0.66, $P=0.005$] (see Fig. 2)).

Relation between face and voice expression identification, and the emotional and behavioural changes

Using the mean score obtained by each individual on both tests, there was a significant negative correlation between the mean on the expression identification tests, and scores on the subjective emotional change questionnaire (see Fig. 3) [Spearman's rho $(7)=-0.882$, $P<0.05$, within the ventral frontal group]. Taken separately a significant negative correlation was obtained between scores on the subjective emotional change questionnaire and percentage correct on the facial expression test [Spearman's rho $(21)=-0.61$, $P<0.01$].

A significant negative correlation was also obtained between scores on the subjective emotional change questionnaire and percentage correct on the vocal expression test [Spearman's rho $(16)=-0.62$, $P<0.05$]. (This did not reflect a general cognitive deterioration in the ventral frontal group, in that neither the verbal IQ scores nor the paired associate learning test scores were correlated with the performance on the expression identification tests.)

Nine of the 11 patients in the ventral frontal group shown in Table 4 were judged as part of the behavioural questionnaire to misinterpret other peoples' moods and feelings. Eight of these nine patients *were* impaired on both face and voice expression identification. There is therefore a good correspondence between staff judgement in the behaviour questionnaire and the expression identification test results.

Only one of the nine patients in the ventral frontal group who were judged to misinterpret peoples' feelings (case 12, who was also impaired at both expression identification tests) was aware of any such difficulty.

Of the 11 patients in Table 4 with ventral frontal damage, eight of the 10 who were asked about this claimed that other people misinterpreted *their* moods or feelings.

In the non-ventral group staff opinion and test performance also correspond: no patient was considered by staff to misinterpret other peoples' moods and none was impaired on both of the expression identification tests. (Only case 4 in the non-ventral group, who was impaired at the vocal expression identification test, felt that he had a problem, although he was not judged by staff to have one.)

Discussion

In this study, the hypothesis developed from studies on non-human primates that damage to the ventral parts of the frontal lobes may be involved in face processing relevant to emotion was tested, and deficits were found in some patients with ventral frontal damage in face and also voice expression decoding. Decoding face and voice expression is likely to be involved in normal social and emotional responses to other individuals [21–24, 26, 27, 29]. Face and voice expression can convey reinforcing signals, and the orbitofrontal cortex may be involved in rapid learning of the reinforcing signals given by different individuals [21–24, 26, 29]. The results of this investigation also indicate that the face and voice decoding deficits are strongly associated with the subjective emotional changes and the observable changes in behaviour. These aspects of the results are discussed next.

Relationship between identification of emotional expression and subjective emotional change

The present study is the first to demonstrate a relationship between performance on tests of facial and vocal expression identification and alterations, since the lesion, in the patients' experience of those emotions featured in the tests. The greater the total alteration in emotional experience (increase or decrease in both positive and negative emotions) as reflected in the subjective emotional changes questionnaire, the worse the performance on each of the tests of expression identification (see Table 2, and as reflected in Fig. 3). This correlation suggests that for some patients with frontal damage at least, the expression identification

Table 4. Interpretation of moods and feelings

Patient Case no.	Staff judgement: Does the patient misinterpret other people's moods/feelings? Yes/No	Patient's opinions Questions asked of patient: Do you misinterpret people's moods/feelings? Yes/No	Do others misinterpret your moods/feelings? Yes/No	Patient's comments
Ventral frontal				
2*	No	No	Yes	My wife gets me wrong
3†	Yes	—	Yes	People think I'm angry or sad when I'm not—they think I'm harsh and hard
4†	Yes	—	Yes	People think I'm more aggressive than I am
5†	Yes	DK	Yes	They haven't got a clue how I feel
6	No	No	No	
7†	Yes	No	Yes	People think I'm OK when I'm low, depressed
8†	Yes	No	—	
9	Yes	Yes	Yes	People think I'm happy when I'm not and that I'm grumpy when I'm not
10†	Yes	No	Yes	People think I'm much angrier than I am
11†	Yes	No	No	
12†	Yes	Yes I can't tell what they're feeling—they seem flat	Yes	My wife thinks I'm grumpy when I'm not
Non-ventral				
1	No	No	No	
2	No	No	No	
3*	No	No	No	
4*	No	Yes People seem happy all the time	Yes	People think I'm happier than I am
5*	No	No	No	
6	Sometimes	No	No	
7*	No	No	No	
8	No	No	No	
9*	No	No	Yes	My voice is monotonous
10	No	—	—	
11*	No	No	No	

*Impaired at one of the expression identification tests at $P<0.05$.
†Impaired at both of the expression identification tests at $P<0.05$.
—: Not asked; DK: don't know.

deficit may be part of a wider emotional disturbance (see below).

The present study is also the first to test the same patients on both voice expression identification and voice discrimination and to demonstrate a dissociation between performance on these two tasks. The fact that cases 3, 4 and 11 performed so poorly on the voice expression test but had no difficulty in discriminating successively presented voices (in the voice discrimination test) indicates that their difficulty in the interpretation of the emotional meaning of the sounds in the expression test need not be due to any low-level auditory problem, nor even to any special difficulty with the analysis of the information contained in voices. It suggests instead that the impaired identification of vocal expression in patients with ventral frontal damage genuinely reflects what could be called 'auditory affective agnosia'. This is further supported by the fact that many patients who performed poorly on the vocal expression identification test were unimpaired on the environmental sounds test and/or were well able to imitate the emotional sounds they could not identify. Cases 2 and 3 did this so well that a high proportion of their imitations were correctly identified by normal subjects who subsequently listened to recordings of them. The ability to imitate convincingly what they cannot interpret had parallels with visual agnosia, in which patients can copy drawings recognisably without recognising either what the drawing or their copy represents. Further research will determine whether patients are more impaired at identifying or producing, on request, convincing sounds (or facial expressions) corresponding ot those emotions they can no longer experience.

The demonstration that the identification of facial expression may be impaired independently of any difficulty in recognising the identity of faces is also consistent with what has been found in other studies [13, 38]. It demonstrates that the facial expression identification deficit, like that for vocal expression, need not be

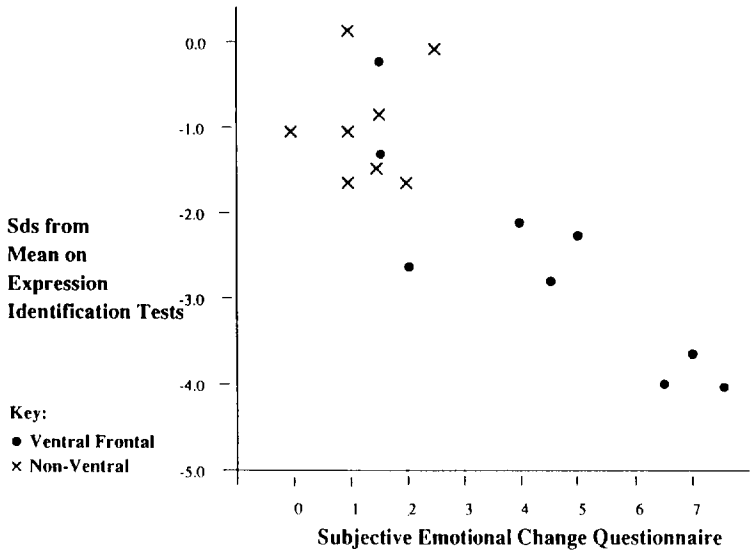

Fig. 3. Relation between subjective emotional change and expression identification. (The greater the emotional change reported, the higher the score.) The expression identification measure is the mean over the face and the vocal expression identification tests for each subject of the number of standard deviations their score is away from the mean scores of normal subjects. Circles—patients in the ventral frontal group. Crosses—patients in the non-ventral group.

secondary to perceptual difficulties, but relates specifically to the emotional nature of the stimuli.

Consistent with what has previously been reported, patients impaired at expression identification in one modality were generally also impaired in the other [37]. However, exceptions were found in the present investigation as can be seen in Table 2 and described in the Results. The dissociations found in some patients are consistent with the possibility of separate brain regions responsible for the perceptual aspects of facial and vocal expression identification, each of which may project to an area which is responsible for emotional experience. Impaired performance on expression identification could result either from damage to one or both of the modality-specific analyzers or from damage to or disconnexion of these from the higher areas concerned with experience. Particular difficulty in the identification of emotions whose experience had been affected would be consistent with such a model. The fact that minimal emotional change was reported in the patients who were impaired at only one test, whereas quite drastic changes were reported by patients impaired at both tests (see Table 2), is also consistent with this hypothesis. Anatomical and physiological evidence is also consistent with separate (although overlapping) systems for the analysis of emotion and expression-related stimuli in the two modalities, in the ventral frontal cortex of macaques is known to receive separate visual and auditory inputs and contains neurons in these regions which can respond to faces, or in other regions to auditory stimuli [1, 19, 21, 27, 28, 36, Rolls and Critchley, unpublished observations).

The damage in the patients in the ventral frontal group in the present study arose in some cases as a result of head injury, and in others as a result of CVA (or in one case a cyst). What was common to these patients was clearly identifiable pathology in the ventral frontal region, and it was on this basis that the patients were included in the ventral frontal group. The fact that similar results were found in the patients with head injury and with other types of pathology within the ventral frontal group, and that the results were different from the patients in the non-frontal group, provides evidence that the critical site of brain damage for the findings described here is the ventral part of the frontal lobe.

It is of interest that although some of the patients in the ventral frontal group with expression identification deficits had bilateral damage, some had unilateral damage (see Table 1). In particular, when the damage was caused by a CVA, we can be quite confident that the damage evident in the brain scan was restricted to one hemisphere. Thus the present study suggests that unilateral ventral frontal damage may be sufficient to cause the expression identification deficits described here. It was notable that although one of the most prototypical patients in this study (case 4) had left ventral frontal damage, this patient was left-handed.

Relationship between the behavioural and subjective emotional changes

The present study has demonstrated a strong positive relationship between scores on the behaviour questionnaire, completed by staff, which was designed to reflect the abnormal behaviour (especially social) associated with frontal damage, and scores on the subjective emotional change questionnaire completed by the patients, which was designed to reveal how they felt their ability to experience emotion had changed since their illness or injury.

The best correlation in the present study between

scores on the behaviour questionnaire and scores on the subjective emotional change questionnaire was obtained by using the sum of the changes in either direction of both positive and negative emotions. Further research would be needed to establish clearer links between changes in particular emotions and particular behavioural abnormalities. The present group of patients had mostly suffered right or bilateral damage and the pattern of disinhibited behaviour, combined with a reduction or absence of negative emotion (accompanied in some cases by a marked increase in positive emotions) present in many patients, is in fact consistent with the views of Starkstein and Robinson [34], according to whom right frontal damage produces both these effects. The present results are also consistent with the study by Damasio *et al.* [7], in which patients with sociopathic behaviour who repeatedly behaved in a way that led to negative consequences were shown to lack normal autonomic responses to emotionally-charged stimuli. As well as being disinhibited in daily life, the present group of patients, in tests of reversal and extinction, repeatedly responded in a way which lost them points—by touching a once rewarded stimulus long after doing so had become incorrect [29]. Like Damasio *et al.*'s sociopathic patients, they also suffered from reduced emotional responsiveness, especially in the present study in the ability to feel fear or anxiety. This was in fact the emotional change which best distinguished the ventral frontal from the non-ventral groups of patients. Some of the patients in the ventral frontal group even gave reduced fear and anxiety as the reason for some of their impulsive and disinhibited behaviour.

However, although most of the disinhibited patients (all but case 11 in the ventral frontal group Table 3 were disinhibited) did have reductions in negative emotions, especially in fear, many also had some reduction in positive emotion and/or an increase in sadness. This observation is in fact consistent with Damasio *et al.*'s study of sociopathic patients who failed to show autonomic responses to positive as well as negative emotionally-charged pictures.

In considering the pattern of emotional change found in the ventral frontal group, it should also be noted that four of the five emotions about which patients were questioned were negative, reflecting the emotions used in the two tests of emotional expression identification (which are also those generally used in other studies). Although patients were questioned in some detail about various ways in which positive emotion might have altered, responses to these questions were combined and are represented in just one of the five columns in Table 3. A differently designed questionnaire might have given more weight to changes in positive emotions. Nevertheless the reduction in positive emotion (and/or increased sadness) described by some of the patients in the ventral frontal group in the present study was related by them to the difficulties imposed by their disabilities—including a sense of social isolation and uselessness. It was therefore an appropriate psychological response to their situation. As such it was in contrast to the loss or reduction of negative emotion (and/or marked increase in positive) which, because of its inappropriateness, seems more likely to be a direct result of damage to the brain, and more likely therefore to be relevant to the behavioural abnormalities also displayed by the patients.

Although some of the patients in the ventral frontal group (e.g. cases 4 and 5) who were disinihibted or socially inappropriate clearly had a reduced ability to empathise with the feelings of others, and to this extent conform to the definition of sociopath, this did not appear to be true in all cases. Case 7, for example suffered considerable remorse if he upset people and case 8 was very concerned about the possible future effects of her behaviour. Even cases 4 and 5 (both of whom were ambulant) found the sight of other patients in wheelchairs, with severe disabilities, distressing.

Further research is necessary to determine how lesion site or pre-morbid personality might contribute to the difference between patients with disinhibited or inappropriate behaviour who do or do not empathise with the suffering of others or express concern about the effects of their behaviour on others. Further research would also be necessary to determine whether the underlying impairment in what Damasio *et al.* [7] refer to as 'sociopathic' patients, lies in a failure to experience the negative consequences of their behaviour as genuinely punishing, or whether perhaps in some cases it lies more in forming associations between behaviour and those consequences which are experienced as negative. Greater understanding of this could be important when considering how best to motivate and retrain behaviourally abnormal and emotionally impaired patients.

Awareness of emotional change and of problems in controlling behaviour

As shown in the answers given on the subjective emotional change questionnaire, patients were both articulate about and showed considerable awareness of emotional changes since illness or injury (see Appendix 1). They were surprisingly well able to recall how they had experienced the different emotions before the brain damage and to compare this with their current experience, even though in some cases the damage had occurred many years earlier. It would appear that the patients in the present group preserved some representation of the subjective experience of emotions they no longer experienced, as well as the linguistic ability to convey both this and their present contrasting experience. As such these observations are consistent with those made by Blonder *et al.* [2] who found that the understanding of emotional words was unimpaired in patients with expression identification deficits (although that study did not look at subjective emotional change).

They are also consistent with Damasio *et al.* [7], whose sociopathic patients failed to respond autonomically to emotionally-charged pictures, but who did in fact respond normally when asked to describe the same pictures.

According to Prigitano [20], frontal damage is likely specifically to affect awareness of socially inappropriate behaviour. It is true that the present group of patients did seem unaware of the inappropriateness of their behaviour while they were engaged in it. Nevertheless, as documented in Appendix 1, most patients were also able to appreciate to some extent at least that they were generally impulsive or had difficulty in controlling their behaviour, and they were aware of some of the problems this could cause. Although Saver and Damasio [32] did not question their patient E.V.R. about his feelings or behaviour, the present observations are consistent with the descriptions of him as having well preserved social knowledge in the context of a social conduct disorder. They are also consistent with the fact that the present group of patients could all explain what should be done or avoided in the reversal and extinction tests referred to earlier, but failed to act accordingly.

Finally, the fact that patients were generally *unaware* of their problems in recognising emotional expression in others, but felt instead that others misinterpreted them, is consistent with their more general unconcern or underestimation of the seriousness of their condition, as reported by staff. This is one way in which the present findings could have implications for rehabilitation. Given the patients' tendency towards reduced spontaneous expressiveness, as also reported by Borod *et al.* [4, 5] and Blonder *et al.* [2, 3], their complaints that others misinterpreted *their* feelings may well have been justified.

Implications for rehabilitation

The present results have other important implications for rehabilitation. Difficulty in relating to others, and especially to their families, causes considerable difficulty when patients return home, and frequently leads to marital breakdown. Explanations of the patient's underlying difficulties to the patient's family should be given. Patients such as case 2, for example, whose identification of vocal emotion is so impaired but whose facial expression identification is normal, could be encouraged to look at people whenever they speak to him, and his wife could also be advised on how best to communicate with him. Analysis of systematic errors in interpreting expression (for example, falsely recognising 'sad' as 'puzzled' would also be beneficial, and could help to target efforts at rehabilitation and retraining most efficiently. Patients who complain that others misinterpret them in particular ways could be helped to gain awareness of their own facial and vocal expressions, and especially to try to avoid appearing angry when they are not. Their spouses could be counselled about the ways in which they misinterpret the patients.

Conclusion

In summary, a correlation has been demonstrated between abnormal behaviour and subjective changes in emotional experience in a group of patients with ventral frontal lobe damage who also suffered from expression identification deficits. It was also shown that the greater the reported emotional change, the worse the performance in each of the tests. It can be concluded that the expression identification deficits, although they may exist in patients with relatively intact subjective emotional experience, are, in frontal patients, generally part of a wider disturbance of emotional experience, which in turn is related to the severity of behavioural change. In the present study, an impairment on just one of the two expression identification tests was found only in patients with minimal alteration in their emotional experience. By contrast, all of the patients who were impaired on both tests (in all cases patients in the ventral frontal group) reported dramatic alterations in the experience of the emotions which had featured in the tests. Even patients with extreme alterations in subjective emotional experience retained, however, an understanding of emotional terms, along with the ability to recall prelesion emotion and to describe how things had changed. They were also surprisingly well able to comment on the problems they had in controlling their behaviour, relating it sometimes to emotional changes, especially to a reduction in fear or anxiety. The ability to speak about what they should or should not do is consistent with the performance of the same patients on reversal and extinction tests and provides further illustration of the dissociation between what frontal patients know and what they do.

The impairments in expression identification, and the changes in subjective emotional experience described in this paper were not a simple consequence of a general emotional change produced by brain damage or disability, in that similar changes were *not* found in the non-ventral group (see results section on subjective emotional changes). Instead, the changes were found to be associated in this study with damage to the ventral part of the frontal lobe. Further investigations of the brain regions crucial for the changes described here will be important.

Acknowledgements—This research was supported by Oxfordshire Regional Health Authority NHS Locally Organised Research Grant No. 90/01, MR/CC 94.94671; and by a grant from the Oxford McDonnell-Pew Centre in Cognitive Neuroscience. We thank Dr P. Anslow and the staff of the Magnetic Resonance Imaging Unit at the John Radcliffe Hospital for their help and guidance with the MR imaging; the patients and others who participated in the research; the staff of the

Rivermead Rehabilitation Centre; J. McGrath, Clinical Psychologist at the RRC, and Dr J. Cockburn; and M. Freedman, who participated in preliminary studies.

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Appendix 1

(a) Examples of the decrease in the ability to feel sadness and fear described by some of the frontal patients

Case 4: "Emotion, tears, that's all gone out of the window. If I saw someone cry I'd just laugh—people look silly getting upset."

Case 3: "I have no feelings. . . . I can't tell if I have emotions—I can't tell if I am saddened. . . . I don't get really sad—just soppy and sentimental."

Case 10: "I don't really get sad." According to Case 10, the nearest he gets to sadness is disappointment—"If I'm expecting something nice which doesn't happen."

Case 5 and 4 both described an increase in aggression, accompanied by a decrease in fear. Case 5 said that anger and irritability "had increased dramatically," while fear and anxiety were no longer part of his experience. Case 4: "I'm much more aggressive and I feel much less fear. I go fighting for no reason: I don't get anxious."

(b) Examples of lack of empathy (or indifference to the opinion of others)

Case 5 wanted to kill the driver who had knocked him down, and even tried to enlist help to do so. He was deterred by thoughts of ending up in "an asylum," rather than by ethical considerations. He looked blank and said nothing when asked about the effects such a murder would have on the family of the driver, and said he found that hard to think about. He also claimed to be a lot less concerned about other people's opinion of him since the accident. "I don't give a monkey's about it."

Case 4 also expressed murderous intentions towards someone who owed him money, but who was refusing to pay him back. He claimed that killing someone wouldn't "bother" him now (i.e. since his head injury); that it would "mean nothing." "I've no emotions since the accident. Years ago I had lots of feeling." As another example he described how upset he had once become after seeing his girlfriend with another man. Now, he said, he wouldn't care: "There are plenty more fish in the sea."

Case 7 explained how he felt his personality had altered since his injury. "I used to be shy and quiet. Now I'm happy-go-lucky. I'm always telling jokes. I used to be frightened people would look at me (dancing for example). Now I have a go at most things." Later he said: "I ain't scared of nobody. I'm not frightened of opening my mouth and speaking my mind. In the old days (i.e. before his head injury) I wouldn't put someone in their place, but now, if I think someone's in the wrong, I'll tell them and not give a monkey's what they think of me."

(c) Examples of patients' insight into the social repercussions of their behaviour

Case 2: "I've become impulsive since the accident. If I have something to say I can't wait and have to say it straight away. I am trying to teach myself to be more patient."

Case 4 explained that, because he was so impulsive: 'Someone might wind me up with the first sentence they say. I'm over-anxious to fight and I'll not wait to hear what else they have to say." Case 4 readily provided additional examples of impulsiveness (for example, overspending), and admitted that "Since I've taken up body-building I tend to show off a bit." (One of his most salient disinhibited behaviours was to practice martial arts in inappropriate circumstances.)

Case 7, referring to his tendency to speak his mind (referred to below), said; "Afterwards, I think to myself 'Ah, well, I should have kept my bloody big mouth shut . . . if only I'd approached it in a different way'—and it preys on my mind, and I feel sick and nervy." He also felt very bad about an incident in which he had, in a fit of exuberance, swept a member of staff off her feet and planted a kiss on her cheek, wrenching her neck painfully.

Case 5 described the difficulty he had in controlling his temper and his urge for revenge. He asked whether his MRI scan would reveal "which part of my brain controls my temper" and whether doctors knew "what chemicals are released into the bloodstream which might trigger it off?"

Case 10 described how he sometimes swears, shouts and bangs the table if he misses a shot playing snooker "and people think 'What's going on, why did he swear like that?' and I didn't mean it (i.e. to upset people)."

Case 8 claimed that she does and say things she wouldn't have before her illness. She deplored her greatly increased sex drive, and reported that her husband complained that she was far more demanding "sex-wise"—that "I'm not the woman he married." She also said that she was "much more outspoken" and feared that it would get her into trouble one day. She said she was trying hard to control it, and agreed it was part of a general impulsiveness.