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Deficits in facial emotion perception in adults with recent traumatic brain injury

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Abstract

We examined whether facial emotion perception was compromised in adults with recent traumatic brain injury (TBI). Few studies have examined emotion perception in TBI; those that have, examined *chronic* patients only. Recent and chronic TBI populations differ according to degree of functional reorganization of the brain, use of compensatory strategies, and severity of cognitive impairments—any of which might differentially affect presentation of emotion perception deficits. A secondary aim of the study was to utilize the TBI population—in whom diffuse axonal injury (DAI) is a cardinal neurological feature—to examine the suggestion of Adolphs et al. [*Journal of Neuroscience 20*(7) (2000) 2683] that damage to white matter tracts should give rise to emotion perception deficits. *Methods*: Thirty TBI participants and 30 age-matched controls were tested. A 2 × 3 mixed design was employed. The dependent variable was accuracy on neutral and emotional face perception tests. *Results*: (1) The TBI group performed significantly less accurately than the matched controls on the facial emotion perception tasks, whereas the groups performed equivalently on a non-emotional face perception control task. (2) A sub-group of TBI participants *without* evidence of focal injury to areas of the brain most commonly implicated in facial emotion perception was as impaired on the emotion perception tasks as a second sub-group who had sustained focal lesions to these areas. This suggests an alternative neurological mechanism for deficits in the first sub-group, such as DAI. *Conclusions*: Patients with recently acquired TBI are impaired in their ability to perceive emotions in faces. DAI alone may cause facial emotion perception deficits.

Keywords: Affect; Neuropsychology; Diffuse axonal injury; TBI; Right hemisphere

1. Introduction

The ability to read emotion in other people's faces can be selectively impaired as a result of neurological disease or injury; this impairment has been investigated for several decades in a number of neurological populations (Borod et al., 1998; Heilman & Gilmore, 1998). Researchers have studied facial emotion perception in patients with unilateral focal lesions (usually due to cerebro-vascular accident and tumors; Blonder, Bowers, & Heilman, 1991; Borod et al., 1998; Starkstein & Robinson, 1988) schizophrenia, (Borod et al., 1990), Parkinson's Disease (Rao, Huber, & Bornstein, 1992; Raskin, Borod, & Tweedy, 1990) and traumatic brain-injury (TBI, a.k.a. closed head injury; Jackson & Moffat, 1987; Spell & Frank, 2000), although studies of the TBI population are few.

Many studies of emotion perception in faces have been concerned with identifying those regions of the brain which, when damaged, give rise to emotion perception deficits (see Adolphs, 2002). A large number of focal lesion studies over the past several decades have implicated the posterior regions of the right hemisphere (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod, 1996; Strauss & Moscovitch, 1981). In particular, recent evidence has pointed towards a critical role for the right somatosensory-related cortices (including S-I, S-II, insula, and anterior supramarginal gyrus; Adolphs, Damasio, Tranel, Cooper, & Damasio, 2000) as well as the right mesial anterior infracalcarine cortex (Adolphs et al., 1996). Several studies have suggested a role for the amygdala (Adolphs, Tranel, Damasio, & Damasio, 1994; Calder et al., 1996) and the basal ganglia (Cancelliere & Kertesz, 1990) in facial emotion perception, and studies have produced equivocal findings with respect to a role for the prefrontal cortex (e.g. Hornak et al., 2003). When prefrontal regions have been shown to play a role in facial emotion perception, it has been on tasks that have explicit verbal

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requirements (e.g. labeling; Adolphs et al., 2000; Hornak, Rolls, & Wade, 1996; Keane, Calder, Hodges, & Young, 2002; Kawasaki et al., 2001); the frontal lobes do not appear to play a role in so-called "conceptual" tests of facial emotion, that is, tests with minimal lexical demands, such as discrimination and sorting paradigms (see Adolphs et al., 2000 for an illustration of this dissociation). In studies of patients with frontal lobe lesions where facial emotion perception impairments were observed, the impairments were associated with lesions to the orbitofrontal and frontal opercular regions as well as the superior medial and anterior cingulate cortex (Adolphs et al., 2000; Hornak et al., 1996; Kawasaki et al., 2001; Keane et al., 2002). In addition to the regions mentioned above, Adolphs et al. (Adolphs et al., 2000; Adolphs et al., 1996) have hypothesized a role for white matter structures in emotion perception, suggesting that facial emotion perception requires the white matter structures that surround and connect the occipital and somatosensory related cortices involved in facial emotion perception. Based on this hypothesis, one would expect facial emotion perception to be disrupted in TBI patients because TBI causes widespread damage to white matter tracts (Meythaler, Peduzzi, Eleftheriou, & Novack, 2001; Teasdale & Graham, 1998).

The small number of studies that have investigated facial emotion perception in groups of TBI patients have indeed found evidence of impairments in emotion perception: Prigatano and Pribram (1982) examined perception (and memory) of facial emotion in a group of patients with closed head injury, CVA and tumors. When they divided the group into those with closed head injury ($N = 10$) and those with CVA or tumor ($N = 10$), they found that the former performed less accurately than the latter on the facial emotion perception (and memory) tasks. Jackson and Moffat (1987) compared 15 patients with severe closed head injury to a group of age and IQ-matched controls on tests measuring perception of facial expressions and postures. They screened out all prospective participants who scored less than 100% on a neutral face perception task. Using photographs and line drawings to measure emotion perception, they found that the closed head injury group performed worse than the controls on all emotion perception tasks. Spell and Frank (2000) recently examined the recognition of facial emotion (and vocal prosody) in a group of 24 TBI patients and 24 matched controls. They too found evidence of facial emotion perception deficits in their group of TBI participants. These studies did not match TBI and control group performances on a neutral facial perception task. So, it is possible that impairments observed in the respective TBI groups were attributable to task difficulty or to other cognitive deficits (e.g. attention)—and not to emotion perception deficits per se. However, in the Jackson and Moffat (1987) study, this alternative explanation is unlikely: first, participants were screened on a non-emotional face perception task, and second, test items that controls found difficult were removed, and then an items analysis was performed to assess whether the TBI group performed worse on those remaining items that the control group found to be relatively difficult. They found no significant relationship between test performance and item difficulty.

The above studies provide initial evidence that patients with chronic TBI suffer facial emotion perception deficits. They thereby provide indirect support for the hypothesis of Adolphs et al. that white matter damage should give rise to emotion perception deficits (Adolphs et al., 2000). In the present study, we tested this hypothesis more directly by examining the facial emotion perception performances of two sub-groups of TBI patients. Patients were assigned to one sub-group if they showed CT evidence of focal injury to any of those areas that have been most implicated in facial emotion perception deficits (i.e. the posterior right hemisphere, the basal ganglia and the amygdala). They were assigned to the second sub-group if they showed *no* CT evidence of focal injury to these areas. If the second sub-group were to show facial emotion perception deficits, this would suggest an alternative mechanism of impairment, such as prefrontal lobe damage (Adolphs et al., 2000; Hornak et al., 1996; Rolls, 2002) or diffuse axonal injury (DAI), as suggested by Adolphs et al. (2000). If the second sub-group were to show impairment on "conceptual" facial emotion perception tasks (tasks for which the prefrontal lobes appear to play no role), this would implicate DAI specifically.

The TBI population is not only of interest scientifically, but it is also of clinical interest because patients with TBI are well known to experience significant psychosocial adjustment problems following injury: the inability to read the emotions of others could play a role in this disability. This possibility has been previously suggested (Morton & Wehman, 1995; Prigatano & Pribram, 1982; Spell & Frank, 2000) and is bolstered by findings of positive correlations between emotion perception and social adjustment/competence in both healthy children (Leppanen & Hietanen, 2001) and brain-injured adults (Hornak et al., 1996).

We examined facial emotion perception in *recently* traumatically brain-injured patients. The previous studies described above examined patients with *chronic* lesions: the mean number of years post-injury was 6.5 in the Jackson and Moffat (1987) study and 6.8 in the Spell and Frank (2000) study. (Mean time post-injury was not provided for the closed head injury group in the Prigatano and Pribram study.) Examining patients early post-injury allowed us to examine emotion perception deficits before a fully functionally reorganized brain or adaptive compensatory mechanisms altered manifestation of the deficit (Luria, 1963; Luria, Naydin, Tsvetkova, & Vinarskaya, 1975; Robertson & Murre, 1999). Clinically, the recent TBI group is of particular importance because emotion perception deficits might interact deleteriously with other deficits that dominate clinical presentation in the first months after injury, such as emotional dysregulation, disinhibition and compromised judgement (Feinstein, 1999).

The aim of the present study was to build upon past research as follows: (1) to confirm the finding that emotion perception deficits are present in TBI, matching patient and control performance on a neutral facial perception task in order to rule out alternative interpretations; (2) to extend the finding to recently traumatically brain-injured patients; and (3) to investigate preliminarily the hypothesis that damage to white matter tracts causes impaired facial emotion perception.

In order to achieve these objectives, we measured facial emotion perception using three tasks from *The Florida Affect Battery*, Revised (FAB; Bowers, Blonder, & Heilman, 1989; 1998), an emotion labeling task, an emotion discrimination task and a control task involving non-emotional face perception. As mentioned above, Adolphs (2002) demonstrated a dissociation between the brain regions involved in performance of a "lexical" emotion perception task (labeling) versus a non-lexical or "conceptual" task (sorting). We employed both types of tests: a labeling task with direct lexical demands, and a discrimination task ("same–different" judgements) without direct lexical demands. We compared the performance of 30 recently traumatically brain-injured participants (an average of 2.6 months post-injury) to that of an age matched control group. We also divided our TBI group into two sub-groups (those with vs. those without evidence of focal lesions to the right posterior cortex, amygdala or basal ganglia) and we examined their respective performances.

Our predictions were as follows: (1) as compared to the control group, the TBI group would show disproportionate impairments on facial emotion perception tasks relative to a neutral face perception control task and (2) even patients *without* focal lesions to the right posterior hemisphere, the basal ganglia or amygdala would show facial emotion perception deficits because of disruption to white matter connections between these critical areas.

2. Method

2.1. Participants

2.1.1. TBI group

The TBI patient group ($N = 30$) was recruited from the Acquired Brain Injury, In-patient service of the Neurorehabilitation Program at the Toronto Rehabilitation Institute. Patients were included in the study if they had sustained a TBI as evidenced by: (1) a clinical diagnosis of TBI during acute-care hospitalization; and (2) positive neuroimaging findings and/or a lowest Glasgow Coma Scale (GCS) score (Teasdale & Jennett, 1974) of 12 or less. All patients had positive neuroimaging findings except for one, and his lowest GCS score was three. Exclusion criteria were as follows: (1) the presence of neurological disease (including any known dementias) or neuroradiological evidence of past brain-injury; (2) cause of TBI secondary to another neurological insult (e.g. a fall, which occurred as a result of a stroke); (3) English below semi-fluent (either due to English as a second language or to aphasia) as judged by the treating speech language pathologist; (4) engaged in alcohol or substance abuse within 2 months of testing; or (5) active psychotic illness as diagnosed by the treating neuropsychiatrist. The contribution of depression to test performance, as measured by the Beck Depression Inventory (BDI; Beck & Steer, 1984) was controlled for statistically with analysis of co-variance (ANCOVA).

The mean GCS score for the group was 7.41 (S.D. = 3.7) and the mean number of months post-injury was 2.6 (S.D. = 1.3). The mechanisms of injury included the following: fall ($N = 10$), driver or passenger in a moving vehicle accident ($N = 10$), pedestrian struck by a moving vehicle ($N = 3$), assault ($N = 2$), blow to head during sport ($N = 2$). Mechanism was unspecified in three patients. All patients were participating in in-patient neurorehabilitation at the time of the study. The mean age of the patients was 40.4 (S.D. = 14.8), mean years of education was 13.3 (S.D. = 2.6) and the male:female ratio was 4:1. Mean BDI was 9.3 (S.D. = 6.5), which is within the normal range.

Ethnic group of participants was characterized because there is some evidence that there are neural differences in the processing of face stimuli depending on whether the stimuli are of the same or different ethnic group as the observer (Hart et al., 2000). Most patients were Caucasian ($N = 22$), two were East-Indian, two were Hispanic, one was African Canadian, one was Asian and one was Aboriginal. Finally, 23 participants were right-handed and seven were left-handed or displayed mixed handedness.

2.1.2. TBI sub-groups

We divided TBI patients into two sub-groups, one with and one without evidence of injury to those areas that have been most commonly implicated in facial emotion perception deficits, namely the right posterior hemisphere, the basal ganglia and the amygdala. No patients displayed basal ganglia or amygdalar focal lesions. Sixteen patients showed evidence of right posterior focal lesions (amongst other lesions). These patients comprised the right posterior focal lesion sub-group (+RPF). The second sub-group ($N = 6$) displayed no evidence of right posterior lesions. This group was called the −RPF sub-group.[1]

The sub-groups were very similar. For the +RPF and −RPF sub-groups, respectively: mean ages were 40.6 (S.D. = 15.2) and 41.7 (S.D. = 14.9); mean years of edu-

[1] Non-right-handed patients were excluded from the TBI sub-group analyses because of the increased probability of reversed hemispheric dominance in such individuals and the increased theoretical possibility, therefore, that a left hemisphere lesion could affect an emotion perception region (Szaflarski et al., 2002). Patients were also excluded if neuroimaging was unavailable. Eight patients were excluded from these analyses on the basis of these criteria.

cation were 12.9 (S.D. = 2.3) and 13.3 (S.D. = 2.2); mean numbers of months post-injury were 2.5 (S.D. = 1.2) and 2.3 (S.D. = 1.5); and mean GCS scores were 7.6 and 6.2. For all comparisons, $t < 1.0$, n.s.

2.1.3. Age matched control group

Control subjects ($N = 30$) were recruited from the local community and matched to the full TBI group on age. Exclusion criteria included the following: active psychotic illness, active clinical depression (based on self-report), neurological disease or past history of brain-injury requiring hospitalization. The mean age of the control subjects was 37.5 (S.D. = 14.6), which was not significantly different from that of the TBI group, $t = -0.764$, n.s. The mean number of years of education of the control group was 15.6 (S.D. = 2.7), which was significantly different from that of the TBI group, $t = 3.35$, $P < 0.05$. The normal control group comprised 28 Caucasians and one Asian and one East-Indian participant, which reflected a different ethnic make-up of the two groups. (Differences between the TBI and control groups in years of education and ethnic group differences were controlled for statistically.)

With regard to the control group and the two TBI subgroups (+RPF and −RPF), there were no significant differences between the three groups on age, $F = 0.35$, n.s., but a significant difference was present for education $F(2,49) = 6.85$, $P < 0.005$.

2.2. Materials

We used the control task and two experimental tasks from the facial affect section of the FAB (Bowers et al., 1989; Bowers et al., 1998), which was kindly provided by the authors. The stimuli used in these tasks included four different women, each expressing one of five different emotions: happy, sad, angry, frightened and neutral. All stimuli used in the battery were derived from a larger set that was rated by 50 college students and 20 normal elderly who were asked to judge the emotion that was depicted on the face. This larger set comprised black and white photographs of actors and actresses, who had been asked to produce the five target emotions. To be included in the battery, each emotional face had to exceed 80% agreement among the raters as to its emotional content, and *all* the emotional expressions made by an *individual* actor/actress had to meet the 80% agreement criterion. (Of the initial 33 actor/actress participants, only females met the above criteria, which is why the stimuli in the FAB battery are all women.) Test–retest reliability was examined in college students and adults in their early 50s (tested 2 weeks apart) and ranged from 0.89 to 0.97 (Bowers, Blonder, & Hellman, 1999). The battery has been validated as a measure of emotion perception deficits in a variety of brain-disordered patients, including stroke and Parkinson's patients (Bowers, Bauer, & Heilman, 1993; Bowers, Blonder, Feinberg, & Heilman, 1991; Bowers et al., 1989).

For two of the tasks, participants were asked to make same–different judgments, and here, a card was provided containing the words "same" and "different". For one task, participants were asked to label the emotion displayed in the photograph, and for this task, a card containing the names of all emotions represented (i.e. happy, sad, angry, frightened and neutral), was provided as a reference for participants.

2.3. Design and procedures

A 2 × 3 mixed design was employed in the main study. Group (TBI versus matched control) and task (two emotion perception tasks and one control task) were the independent variables, and number correct (on the FAB tasks) was the dependent measure. All subjects performed all tasks, and the order of tasks was counterbalanced. The instructions were given verbally by the experimenter. The number of trials in each task was 20. Practice was given for each task for each response type (e.g. same or different for *discrimination*; happy, sad, angry, frightened, neutral for *labeling*). Participants were given unlimited time to complete each of the practice and experimental trials.

2.3.1. Neutral face discrimination

Participants were presented pairs of photographs; each pair contained either two female faces representing the same woman or two female faces representing two different women. They were asked to decide whether photographs represented the same or different people.

2.3.2. Emotional face labeling

Participants were presented photographs of faces of different women, each expressing one of five different emotions (happiness, sadness, anger, fear or neutral). They were asked to name the emotion represented in each picture.

2.3.3. Emotional face discrimination

Participants were presented pairs of photographs displaying two different female faces, expressing either the same or different emotions. They were asked to decide whether the emotions expressed were the same or different.

3. Results

We first conducted a 2 × 3 mixed-design analysis of variance (ANOVA) with group (TBI and matched control subjects) and task (the control task and the two emotion perception tasks) as independent variables. The analysis yielded a significant main effect of group, $F(1, 58) = 25.17$, $P < 0.001$, a significant main effect of task, $F(2, 116) = 48.48$, $P < 0.001$, and a significant group-by-task interaction, $F(2, 116) = 9.79$, $P < 0.001$. TBI patients and matched controls

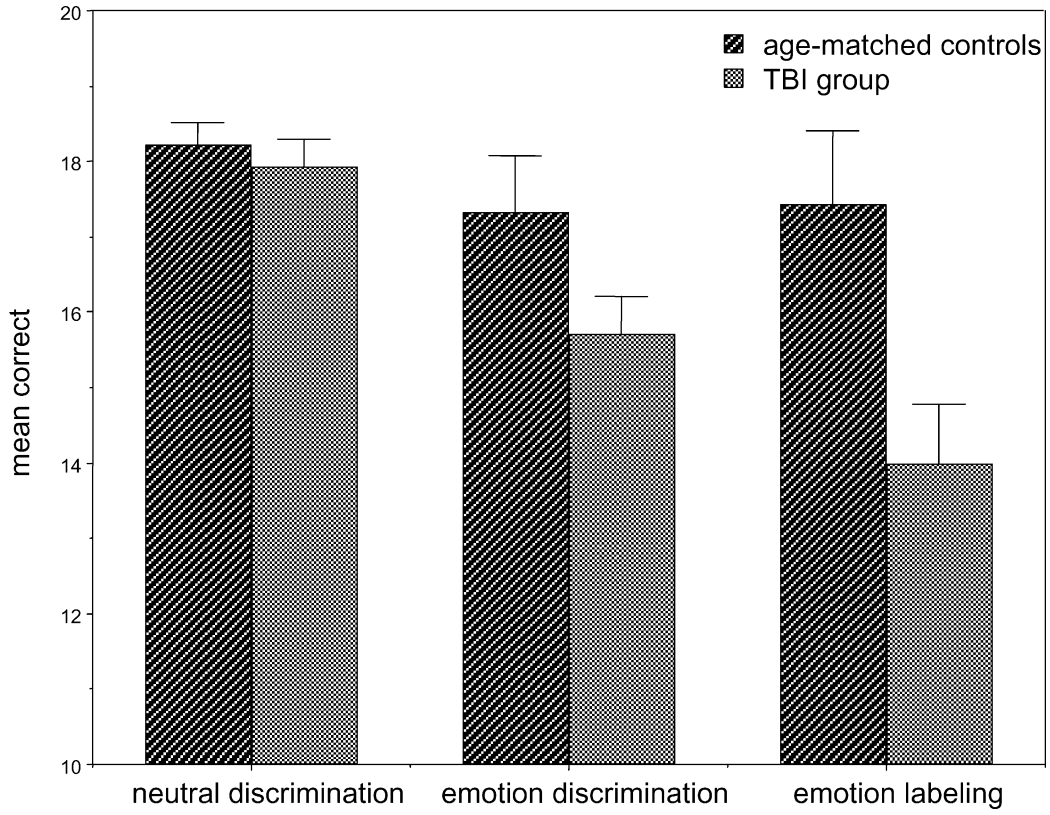

Fig. 1. Off-the-ceiling TBI vs. age-matched control group performances on neutral and emotion perception tasks.

performed at similar levels on the control task (neutral face discrimination), but patients performed worse than normal controls on both emotion perception tasks. Table 1 presents the means for both groups on all three tasks.

Planned comparisons revealed that there was no significant difference in performance between patients and matched controls on the control task, $t(58) = 1.41$, n.s., but patients performed significantly worse than the matched controls on both emotion tasks: discrimination, $t(58) = 4.30$, $P < 0.001$, and labeling, $t(58) = 4.68$, $P < 0.001$. Moreover, the patient group performed significantly worse on the two emotion tasks than they did on the control task: discrimination, $t(29) = 6.30$, $P < 0.001$, and labeling, $t(29) = 9.38$, $P < 0.001$. These results confirmed our first prediction: that the TBI group would show disproportionate impairments on facial emotion tasks relative to the neutral control task, as compared to the normal controls.[2]

Some participants obtained perfect scores on the control task. We considered, therefore, the possibility that a ceiling effect was masking a true difference between patients and control subjects on the control task, and creating a spurious group by task interaction. To evaluate this possibility, we re-analyzed the data using only participants whose performance was off the ceiling on the control task ($N = 23$). The analysis yielded a significant group by task interaction, $F(2, 42) = 4.53$, $P < 0.02$, illustrated in Fig. 1, confirming that the effect was not an artifact of a ceiling effect. Moreover, when off-ceiling TBI and control groups were analyzed separately, we observed no main effect of task for the control subjects, $F(2, 16) < 1.0$, n.s., but a significant main effect of task for the TBI patients, $F(2, 26) = 17.64$, $P < 0.001$.

Table 1
Mean accuracy (out of 20) and standard deviations for TBI full group vs. age-matched controls on emotion perception and neutral control tasks

	Neutral discrimination (S.D.)	Emotion face discrimination (S.D.)	Emotion face labeling (S.D.)
TBI group ($N = 30$)	19.03 (1.38)	15.9 (2.41)	15.00 (2.65)
Matched controls ($N = 30$)	19.47 (0.97)	18.22 (1.67)	17.97 (2.24)

[2] The mean score on the BDI in the TBI group was within the normal range. Nevertheless, a within-subjects ANCOVA was undertaken to examine whether depression in some patients could account for the pattern of performance of the TBI group. This was not the case: there was no interaction of emotion by task, $F = 0.40$, n.s., and an effect of task remained, $F(2,17) = 9.116$, $P < 0.01$. Simple contrasts revealed that there were no significant differences between either of the emotion tasks $F = 0.12$, n.s., but there were significantly worse performances on the emotion discrimination and labeling tasks than on the neutral task: $F(1,18) = 11.43$, $P < 0.005$ and $F(1,18) = 13.56$, $P < 0.005$. BDI scores could not explain the pattern of performance in the TBI group.

Planned comparisons also confirmed that off-ceiling TBI patients performed significantly better on the control task than on both emotion tasks: discrimination, $t(13) = 3.51$, $P < 0.01$, and labeling, $t(13) = 5.44$, $P < 0.001$. Off-ceiling TBI patients also performed better on the emotion discrimination task than on the emotion labeling task, $t(13)=2.71$, $P < 0.05$.

3.1. Involvement of right posterior hemisphere

We next compared the performances of the −RPF sub-group, the +RPF sub-group, and the control group. We suspected that patients without evidence of focal injury to the right posterior hemisphere would still display deficits in facial emotion perception, owing to neural mechanisms of injury other than right posterior focal injury, namely diffuse axonal injury and/or prefrontal lesions (Adolphs, 2002; Adolphs et al., 2000). We predicted, therefore, that emotion perception performance in *both* the +RPF and −RPF sub-groups would be worse than performance in the control group.

A 3 × 3 mixed design ANOVA comparing performance by three groups on the three tasks revealed a main effect of group, $F(2, 49) = 10.95$, $P < 0.001$, a main effect of task, $F(2, 98) = 29.57$, $P < 0.001$, and a group by task interaction, $F(4, 98) = 2.83$, $P < 0.05$. This interaction is displayed in Fig. 2.

Planned comparisons revealed that the −RPF sub-group and the matched controls performed at similar levels on the neutral face discrimination task, $t(34) = -1.3$, n.s., but the −RPF sub-group performed worse than the matched controls on both emotion discrimination, $t(34) = -2.34$, $P < 0.05$, and emotion labeling, $t(34) = 2.52$, $P < 0.05$. Thus, emotion perception deficits among TBI patients were not contingent on the presence of right focal posterior lesions. The +RPF sub-group also performed at similar levels to the matched controls on the neutral face discrimination task, $t(44) = -1.94$, n.s., and worse than the matched controls on both emotion tasks: discrimination, $t(44) = -3.66$, $P < 0.005$, and labeling, $t(44) = 3.42$, $P < 0.005$.

Mean performance levels for the +RPF and −RPF sub-groups were very similar, and planned comparisons revealed no significant differences between these two groups on any of the three tasks, $t(20) < 1.0$, n.s. for all three comparisons.

4. Discussion

We tested whether patients with recently acquired TBI are impaired at perceiving emotions conveyed through facial expressions. Patients with recent TBI were significantly less accurate than matched control participants at discriminating and labeling emotions. A group by task interaction indicated

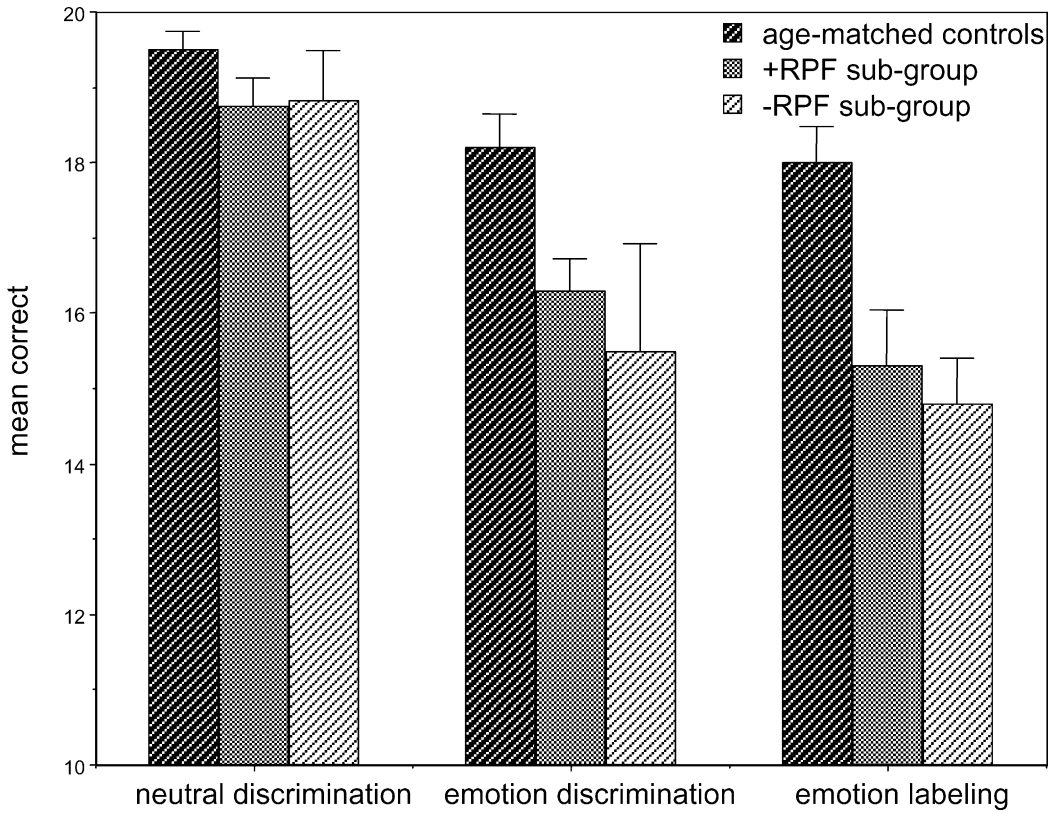

Fig. 2. +RPF sub-group, −RPF sub-group and age-matched control group performances on neutral and emotion perception tasks.

Table 2
Mean accuracy (out of 20) for patients with and without focal frontal lesions. All patients are from the −RPF group

	Neutral discrimination (S.D.)	Emotion face discrimination (S.D.)	Emotion face labeling (S.D.)
Without focal frontal lesions ($N = 1$)	20	18	17
With focal frontal lesions ($N = 5$)	18.6 (1.67)	15 (3.67)	14 (1.14)

that TBI patients performed less accurately than the controls on the emotion perception tasks, but not on the neutral task. The same findings were also observed in a sub-group of TBI and control participants—whose respective performances on the neutral task were off the ceiling.

In a small number of previous studies, evidence of emotion perception deficits in TBI patients has been obtained. These studies did not include a neutral facial perception control task on which patients and controls were matched, and so other interpretations of the findings were possible. The present results corroborate the conclusions in these previous studies and they extend research to a *recently* traumatically brain-injured population. Previous studies examining facial emotion perception in TBI included only patients who were many years post-injury.

We also explored preliminarily whether facial emotion perception deficits would be observed in patients *without* evidence of injury to those areas of the brain that have been most often implicated in facial emotion perception. Extensive research involving focal lesion patients has revealed the importance of structures in the posterior regions of the right hemisphere for emotion perception (Adolphs et al., 1996; Borod et al., 1998); the importance of basal ganglia and amygdala has also been highlighted in a number of studies (Calder et al., 1996; Cancelliere & Kertesz, 1990). We found that a sub-group of patients without lesions in any of these areas (i.e. the −RPF sub-group) performed like the larger TBI group, that is, less accurately on the emotion perception tasks than the normal controls, but comparably to the controls on the neutral facial perception task. Moreover, when this sub-group's performance was compared to that of a matched TBI sub-group who had sustained right posterior focal lesions (the +RPF sub-group), the findings in the two groups were the same: significantly less accurate performance on the emotion tasks than in the neutral task. These findings suggest that right posterior focal injury is not a *necessary* condition for emotion perception deficits and indicate that a different mechanism of injury must have caused facial emotion perception deficits in the −RPF group. One explanation is provided by the hypothesis of Adolphs et al. (2000), that DAI disconnects critical areas of emotion perception, such as the visual cortices, somatosensory-related cortices, and amygdala. DAI is a cardinal feature of TBI; it is very likely that DAI caused the emotion perception deficits in these patients. To our knowledge, the present study is the first to provide data that bear directly on this hypothesis.

A second possible explanation is that impaired emotion perception in the −RPF sub-group resulted from prefrontal cortex lesions: some studies have found evidence of impairment to the lexical aspects of facial emotion perception in patients with prefrontal lesions (Adolphs et al., 2000; Hornak et al., 2003; Hornak et al., 1996). As there was CT evidence of focal injury to the frontal lobes in five of our six patients in the −RPF group (either contusions or over-lying extra-parenchymal injury), this explanation must be considered. Two factors mitigate against this explanation, however. First, the one patient without evidence of focal frontal lobe injury showed deficits on the emotion perception tasks and sparing of performance on the neutral task (see Table 2, and note that a ceiling effect on the neutral task may mask an *even greater* emotion perception deficit in this patient).

As well, patients in the −RPF sub-group displayed deficits on *both* the lexical and non-lexical ("conceptual") facial emotion perception tasks. As discussed in Section 1, Adolphs et al. found that frontal lobe lesions were associated with facial emotion deficits on a lexical (i.e. labeling) but not a "conceptual", non-lexical task. Therefore, frontal lobe lesions are not likely to account for the deficits observed in the −RPF group on the non-lexical task. Quite possibly, DAI disrupted facial emotion perception on the discrimination and labeling tasks, and prefrontal cortex lesions, when present, further compromised performance on the labeling task. This interpretation is consistent with our finding that labeling was more disrupted than discrimination (see Fig. 1).

In conclusion, these results corroborate previous findings of emotion perception deficits in TBI. We have extended previous research through the inclusion of a recently brain-injured group. Our results may warrant particular clinical attention: facial emotion perception deficits may interact in a deleterious manner with other impairments commonly observed in recent TBI, such as compromised social judgement and emotional regulation (Feinstein, 1999). The findings from our study also extend previous research by providing evidence for the interesting possibility that DAI can cause emotion perception deficits. Further research examining TBI patients who have sustained neither right posterior hemisphere, basal ganglia, amygdala nor focal frontal lesions would provide more conclusive evidence for a role of DAI in facial emotion perception deficits. The question could also be addressed using quantitative MRI methods that correlate

degree of axonal injury with degree of emotion perception deficit.

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