Emotion recognition from dynamic emotional displays
following anterior cingulotomy and anterior capsulotomy for
chronic depression.

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Abstract

Four patients that had received an anterior cingulotomy (ACING) and five patients that had received both an ACING and an anterior capsulotomy (ACAPS) as an intervention for chronic, treatment refractory depression were presented with a series of dynamic emotional stimuli and invited to identify the emotion portrayed. Their performance was compared with that of a group of non-surgically treated patients with major depression (n=17) and with a group of matched, never-depressed controls (n=22). At the time of testing, 4 of the 9 neurosurgery patients had recovered from their depressive episode, whereas 5 remained depressed. Analysis of emotion recognition accuracy revealed no significant differences between depressed and non-depressed neurosurgically treated patients. Similarly, no significant differences were observed between the patients treated with ACING alone and those treated with both ACING and ACAPS. Comparison of the emotion recognition accuracy of the neurosurgically treated patients and the depressed & healthy control groups revealed that the surgically treated patients exhibited a general impairment in their recognition accuracy compared to healthy controls. Regression analysis revealed that participants’ emotion recognition accuracy was predicted by the number of errors they made on the Stroop colour-naming task. It is plausible that the observed deficit in emotion recognition accuracy was a consequence of impaired attentional control, which may have been a result of the surgical lesions to the anterior cingulate cortex.
1. Introduction

The ability to accurately recognise the emotional expressions of others is fundamental for successful social interaction (Ekman, 1982). In line with this notion, it has been suggested that impaired emotion recognition might underlie the social dysfunction that is associated with a number of psychiatric conditions, notably clinical depression (Persad & Polivy, 1992; Surguladze et al, 2004) and schizophrenia (Mueser et al, 1996). This is important as such a deficit could undermine the patients’ social support, which has been identified as a significant factor in ongoing psychological distress and the onset of mental illness (Klineberg et al, 2006; Hipkins, Whitworth, Tarrier & Jayson, 2004; Lee et al, 2006; Vaananen, Vahtera, Pentti & Kivimaki, 2005). With this in mind, the increased research effort dedicated to understanding the neural underpinnings of emotional processing that has been evident in recent years represents a vitally important avenue of clinically relevant investigation (see reviews by Adolphs, 2002; Phillips, Drevets, Rauch & Lane, 2003).

Much of this expansion of research effort has involved the use of functional neuroimaging techniques which have enabled considerable advances in understanding event-related changes in brain activity in response to emotional stimuli (Davidson & Irwin, 1999; Dolan, 2002; Phan, Wager, Taylor, & Liberzon, 2002). However, these studies cannot provide information concerning the necessity of different regions for intact emotion processing. By contrast, investigation of patients with acquired brain lesions can help to identify regions that are critical for normal emotion processing. For example, numerous studies (Adolphs, Tranel, Damasio, & Damasio, 1994; Adolphs et al, 1999;
Broks et al, 1998; Calder et al, 1996; Phelps & Anderson, 1997; and Scott et al, 1997) have reported impaired recognition of negative emotions (especially fear) in patients with damage to the amygdala. Similarly, other studies have reported impaired emotional processing in patients with damage to the insula (Calder et al, 2001), caudate nucleus (Sprengelmeyer, 1996), orbitofrontal cortex (Hornak, Rolls, & Wade, 1996; Hornack et al, 2003) and anterior cingulate cortex (Hornak et al, 2003, Wang et al, 2002).

It is difficult to draw firm conclusions from the studies conducted by Hornak et al (2003) and Wang et al (2002), as the patients studied had additional damage to brain regions (orbitofrontal cortex & amygdala) outside of the ACC that have also been implicated in changes to emotional processing. Evidence from patients with circumscribed lesions to the ACC alone would provide more compelling evidence of a critical involvement of this region in emotion recognition. With this in mind, it is notable that there has been a recent resurgence of interest in the use of anterior cingulotomy (ACING) as an intervention for chronic treatment-refractory psychiatric disorder, particularly depression (Matthews & Eljamel, 2003). ACING involves the placement of bilateral lesions within the anterior cingulate gyri with relatively little disturbance of other brain tissue. Another neurosurgical intervention that might plausibly be associated with changes in emotion processing is anterior capsulotomy (ACAPS). ACAPS involves the placement of bilateral lesions to the anterior limbs of the internal capsule, with the intention of disconnecting OFC from midline thalamic and caudate nuclei; regions that have been implicated in emotion processing (Hornak et al, 1996; 2003; Sprengelmeyer et al, 1996). In addition to gaining key information concerning the possible involvement of
these regions in emotional processing, it is important to assess if these neurosurgical procedures are associated with changes in cognitive and/or affective processing, particularly since they involve making permanent changes to the structure (and plausibly function) of the patients’ brains.

It has been suggested (Albert, Cohen, & Koff, 1991) that emotion recognition deficits associated with brain damage might be a consequence of more general neuropsychological deficits. This notion is consistent with a body of work conducted by Pessoa and colleagues demonstrating that emotional processing is significantly modulated by attentional control processes, even when this processing is apparently occurring below the level of conscious awareness (Pessoa, Kastner, & Ungerleider, 2002; Pessoa, 2005; see also Holmes, Kiss, & Eimer, 2006; Vuilleumier, 2002). It is plausible that emotion recognition deficits observed in patients with ACC damage might be a consequence of impaired attentional control, as this region has consistently been implicated in such processing (Botvinick et al, 1999; Pardo, Pardo, Janer, & Raichle, 1990; Swick & Jovanovic, 2002; van Veen & Carter, 2002). Furthermore, there is some evidence that the ACING procedure itself may lead to impaired attentional processing (Cohen et al, 1999; Janer & Pardo, 1991; Ochsner et al, 2001; Turken & Swick, 1999).

The present study involved assessing two groups of neurosurgically treated patients (ACING alone and combined ACING & ACAPS), a clinical control group (non-neurosurgically treated patients with chronic treatment refractory depression) and a group of healthy matched never-depressed controls on a battery of executive tasks and a
‘dynamic’ emotion recognition task. This task has been used in previous studies to assess the consequences of traumatic brain injury (TBI) on emotion recognition accuracy (McDonald et al, 2002; 2003) and was chosen for present study due to its strong ecological validity. It was predicted that both groups of neurosurgically treated patients would exhibit significantly impaired emotion recognition accuracy in comparison to the healthy controls. It was also predicted that the emotion recognition accuracy of the clinical control group would differ from that of the healthy controls. Also of interest were potential differences in the emotion recognition accuracy of the neurosurgery patients compared with clinical controls. It was predicted that successful emotion recognition would be related to performance on tasks tapping central executive function.

2. Method

2.1. Participants

Nine patients (2 males, 7 females) that had received neurosurgical treatment for chronic, treatment refractory, depression (NS group) took part in the present study. Five of the 9 had been treated with two neurosurgical procedures (both ACING & ACAPS) the remaining four with ACING alone. ACING involved placing two thermal lesions bilaterally within the dorsal ACC. Mean co-ordinates (in Talairach space; Talairach & Tournoux, 1988) of the centre of left ACING lesion were $x=10.34\text{mm (SD}=2.2), y=17.08\text{mm (SD}=3.4) \& z=27.71\text{mm (SD}=4.9) \& z=27.71\text{mm (SD}=4.9)$ and the mean volume of this lesion was $166.4 \text{ mm}^3 (SD=95.6)$. Mean co-ordinates of the centre of the right ACING lesion were $x=-9.24\text{mm (SD}=3.1), y=14.18 \text{ (SD}=7.6) \& z=28.27 \text{ (SD}=4.6)$ and the mean volume of this lesion was $228.9 \text{ mm}^3 (SD=163.4)$. ACAPS involved placing two thermal lesions
bilateral in order to interrupt the fibres running within the anterior limb of the internal
capsule. Mean co-ordinates of the centre of the left ACAPS lesion were $x=21.19\text{mm}
(\text{SD}=1.13)$, $y=14.18\text{mm} (\text{SD}=7.6)$ & $z=3.61\text{mm} (6.1)$ and the mean volume of this lesion
was $189.9\text{mm}^3 (\text{SD}=54.6)$. Mean co-ordinates of the centre of the right ACAPS lesion
were $x=-21.78\text{mm} (\text{SD}=1.4)$, $y=16.97\text{mm} (\text{SD}=2.6)$ & $z=5.15\text{mm} (\text{SD}=6.1)$ and the mean
volume of this lesion was $166.8\text{mm}^3 (\text{SD}=47.6)$. The position of the characteristic
lesions for ACING and ACAPS are presented in figures 1 and 2. These figures (created
using version 1.39 of MIRcro software) feature the mean location of the lesions (based on
the neurosurgical patients’ data) illustrated on spatially normalised MRI scans. It should
be noted that lesion markers do not indicate size. All lesions were created by standard
methods under stereotactic control, with bilateral radiofrequency-induced
thermocoagulation stimuli ($70^\circ \text{C}$ for 90s) delivered by 6mm tip monopolar electrodes.
The median length of time since the patients’ most recent operation was 14 months
(range 12 to 46 months). At the time of testing, 4 of the NS patients had recovered from
their depressive episode (i.e. no longer met diagnostic criteria for clinical depression
(ICD-10; World Health Organisation, 1993)) and whose depression severity was rated
scored below the clinical range ($\text{Mean}=4.0, \text{SD}=1.4$) on the Hamilton Rating Scale for
Depression (HRSD; Hamilton, 1960) the residual five NS patients remained significantly
depressed ($\text{Mean HRSD}=25.6, \text{SD}=2.5$). It should be noted that one of these patients had
a secondary ICD-10 diagnosis of obsessive-compulsive disorder. Importantly, this is the
only patient in the neurosurgically treated group with a comorbid diagnosis.
The clinical control group (patients with MD) consisted of seventeen individuals (4 males & 13 females) who were currently experiencing a chronic depressive episode (according to ICD-10 criteria) of at least moderate severity according to the HRSD (Mean=21.9, SD=2.9). It should be noted that the clinicians also performed a computerised diagnosis on the case notes of the patients using version 4 of OPCRIT for Windows (2003) in order to provide an additional diagnosis according to the DSM-IV criteria. OPCRIT has been shown to produce valid and reliable diagnoses based on the DSM-IV criteria (Craddock et al, 1996; Williams et al, 1996). From this procedure it was evident that the clinical control group also met DSM-IV criteria for a major depressive episode. The median duration of the patients’ depressive episodes was 5 years (range 2 to 10 years) and the median number of previous major depressive episodes was 2 (range 1 to 3). The duration of these episodes are categorised in accordance with the guidelines set out in a recent report by a task force of the American College of Psychopharmacology (ACNP) concerning the definition of recovery and remission (Rush et al, 2006). To be considered recovered (out of episode) the patient requires at least 4 months where minimal symptom status (absence of both sadness and anhedonia and fewer than 3 of the remaining 7 symptoms listed in the DSM-IV-TR) is maintained. It should be noted that four of the patients in the clinical control group had evidence of co-morbid disorders; two met ICD-10 criteria for generalised anxiety disorder, one for dysthymia and one for obsessive-compulsive disorder. However, it should be noted that these conditions were all secondary to the ongoing depressive episode. The remaining patients in the clinical control group exhibited no evidence of comorbid conditions. None of the patients in this group had been treated with electro-convulsive therapy (ECT) within the previous 12
months. However, all patients were being treated with antidepressants at the time of testing. Seven of the patients were taking Selective Serotonin Reuptake Inhibitors (SSRIs), four were being treated with Monoamine Oxidase Inhibitors (MAOIs) and the remainder were taking combined Noradrenaline and Serotonin reuptake inhibitors. It should be noted that 3 were also being treated with atypical antipsychotic medication.

The second control group (HC) consisted of twenty-two healthy, never-depressed, individuals (5 males & 17 females) that were similar in age and educational background to the individuals in the patient groups. The characteristics of the individuals making up the four participant groups are reported in table 1. It should be noted that an attempt was made to match the groups in terms of age, sex, educational background, and pre-morbid intellectual ability. The NS patients took part in the present study as part of their long-term follow up at the Advanced Interventions Service at Ninewells Hospital, in Dundee. Clinical controls (MD) were recruited from referrals to the same clinical service. Healthy controls (HC) were recruited face-to-face by the principal author and by electronic and poster advertisements. Prior to their participation in the study, the clinical status of all NS and MD patients was confirmed by detailed psychiatric examination. Diagnostic status was confirmed according to ICD-10 and severity of their depression estimated by the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). The study was approved by the Tayside Committee for Medical Research Ethics.
2.2. Materials and Apparatus

2.2.1. ‘Dynamic’ displays of emotion: The stimuli presented to the participants during the emotion recognition phase of the study consisted of two sets (A & B) of twenty-eight video clips (each lasting 15 – 60 seconds) featuring either a single individual (speaking on the telephone or directly addressing the viewer), or two different individuals engaged in conversation. In the majority of these scenes the central protagonist portrayed (via their facial expression, body posture & tone of voice) one of the six primary emotions (happiness, sadness, surprise, anger, anxiety/fear & disgust). Within each set of stimuli, the six primary emotions were portrayed in four different video clips. However, in four of the scenes in each set, the featured character(s) portrayed no strong emotion (neutral affect). Analysis of the healthy controls’ performance in the present study revealed that the recognition of emotion from set A (Mean=87.7%, SD=6.5) did not differ significantly from that of set B (M=92.2%, SD=6.6), t(20)=1.6, p>0.05. A further video clip, featuring an individual portraying happiness, was included as a practice stimulus for both sets of stimuli. Four printed response cards were provided; each featuring the six emotional descriptors and the word ‘neutral’ in a different order (a fifth response card was also provided for use with the practice clip). All of the video clips included in the present study were drawn from the Emotion Evaluation phase of the Awareness of Social Inference Test (TASIT; McDonald et al, 2002).

2.2.2. Measures: The 21-item Beck Depression Inventory (BDI; Beck et al, 1961) was used to quantify the self-perceived severity of depressive symptoms being experienced by the patients with MD and the NS groups and also to screen for the
presence of depressive symptoms in the HC group. The 17-item version of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) was used to rate objectively the severity of the patients’ ongoing depressive symptoms. The 14-item Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was also used to screen for the presence and severity of anxiety symptoms in all participant groups and to enable the effects of anxiety on emotion recognition to be controlled for statistically. The National Adult Reading Task (NART; Nelson & Williamson, 1991) was used to estimate general intellectual ability. Importantly, performance on the NART has been shown to be unaffected by depression (e.g. Crawford et al, 1987) and also less sensitive to cerebral damage than other methods of assessing premorbid intelligence (see Spreen & Strauss, 1998), thus it provides a reasonably accurate way of estimating premorbid intellectual ability in patients with depression and/or sub-cortical lesions.

2.2.3. ‘Executive’ tasks: Three additional tasks, the traditional Stroop colour-naming task (adapted from Trenerry, Crossen, DeBoe & Leber, 1989), the Hayling sentence completion task (Burgess & Shallice, 1997) and the F-A-S verbal fluency task (drawn from Spreen & Strauss, 1998) were included to test whether emotion recognition accuracy was related to general measures of ‘executive’ neuropsychological functioning.

The Stroop task utilised in the present study involved presenting participants with two printed A4-sized sheets (control & experimental conditions), each featuring an array of 112 stimuli. The stimuli presented during the control condition were a series of rows of capital Xs, each printed in one of four colours (blue, green, red & brown). The stimuli
presented during the experimental condition were a random repeated series of four colour names \textit{(green, blue, red & brown)} printed in one of four colours (note: all words were printed in incongruent colours, e.g. the word RED printed in GREEN ink). The participants’ task was to read out the colour of ink that the stimuli were presented in. The participants were given a maximum of 90 seconds to read out as many colours as they could on each sheet. The order that the participants completed the two lists was counterbalanced.

The Hayling sentence completion task involved presenting the participants with two sets of 15 sentences where the final, predictable, word was missing (e.g. “It’s hard to admit when one is…”). The participants’ task was to provide a word to complete each sentence. In the control condition (the first set of 15 sentences), a sensible word was required to complete the sentences (e.g. for the example shown the word ‘wrong’ would be acceptable). The total response time (in seconds) to complete all 15 sentences was taken as the performance measure in this phase. In the experimental condition (the second 15 sentences) the participant was required to inhibit the predictable response and provide a word that was unrelated to the meaning of the sentence. Two performance measures were collected during this phase of the task; the total response time (in seconds) and the number of errors (providing a word that was related to the sentence).

The F-A-S task was conducted according to standard procedure (outlined in Spreen & Strauss, 1998). Participants were presented with the letters F, A and S (one at a time) and were asked to provide as many words as they could that began with each letter.
The participants were informed that they should not respond with proper nouns (e.g. France), the same word with a different suffix (eat, eating, eaten) or profanities. The participants were allowed 60 seconds for each letter.

2.3. Procedure

2.3.1. General Assessment: At the outset of the test session, once general demographic information had been collected, participants were asked to complete the BDI and HADS questionnaires. Participants were then assessed on the F-A-S Verbal Fluency, Hayling sentence completion and Stroop colour-naming tasks. Finally, each participant completed the NART (using the standard procedure) before moving on to the emotion recognition testing.

2.3.2. Emotion Recognition Task: During the Emotion Recognition phase, participants were presented with one of the two sets of video clips (either set A or B) one at a time in a fixed, pseudorandom order and were asked to watch the clips to the end. Once each stimulus had been presented, the participants were asked to identify, by indicating the appropriate word on the response card, the emotion they considered was being displayed by the central character in the video clip they had just viewed. If participants considered that more than one emotion was present they were asked to indicate which of the present emotions they considered to be stronger. The experimenter marked down the participants’ response on a printed scoring sheet. Following each response, the participants were asked to place the response card they had just used to the back of the pack of four (in order to change the order that the emotion descriptors
appeared to the participants). Once participants had viewed and responded to all of the 28 video clips they were thanked for their participation and fully debriefed as to the rationale for the study.

3. Results

3.1. Participant characteristics

Analysis of the demographic variables (illustrated in Table 1) was conducted using MANOVA with participants’ age, educational background (years of full-time education completed) and IQ (estimated from the participants error score on the NART), entered as dependent variables and participant group entered as a between subjects factor. The results of this analysis (Pillai’s Trace) revealed that the four groups did not differ significantly on the demographic variables; F(9, 132)=1.5, p>0.05. Analysis of the participant’s mood (presented in Table 1) was also conducted using MANOVA with participants’ depression (BDI & HADS_D) and anxiety (HADS_A) scores entered as the dependent variables and participant group entered as the between subjects factor. The results of this analysis (Pillai’s Trace) revealed that the mood of the four groups differed significantly; F(9, 132)=13.0, p<0.001. Separate univariate ANOVA revealed that the groups differed significantly on all mood measures; the Beck Depression Inventory (BDI), the depression subscale of the Hospital Anxiety and Depression Scale (HADS_D) and the anxiety subscale of the HADS (HADS_A); F(3, 44)=161, p<0.001, F(3, 44)=132, p<0.001 and F(3, 44)=61, P<0.001 respectively. Subsequent analyses, using Least Significant Difference tests, revealed that depressed NS and MD participants scored significantly higher on the BDI, HADS_D and HADS_A than did the non-depressed NS
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patients or healthy controls, all tests $p<0.05$. Depressed NS patients also scored significantly higher on the BDI than the MD participants, $p<0.05$. However, these two groups did not differ on either subscale of the HADS, both tests $p>0.05$. Furthermore, non-depressed NS patients did not differ significantly from healthy controls on any of the mood measures, all tests $p>0.05$. Analysis, using univariate ANOVA, of clinician-rated depression severity (indexed by scores on the Hamilton rating scale for depression) revealed that the patient groups differed significantly; $F(2, 23)=88; p<0.001$. Subsequent analysis, using Least Significant Difference tests, revealed that depressed neurosurgery patients were rated as significantly more depressed than the patients with MD and the non-depressed neurosurgery patients; $p<0.05$ and $p<0.001$ respectively. Further, patients with MD were rated as significantly more depressed than were non-depressed neurosurgery patients, $p<0.001$.

3.2. Assessment of executive function

The participants’ performance on the different executive tasks (illustrated in Table 2) was assessed using MANOVA with F-A-S verbal fluency score (total number of words produced to the three different letters), Hayling’s error score (scaled score based on the number of semantically related words given during the nonsense element of the task), Stroop interference score (% increase in colour-naming time from control to experimental task) and Stroop error score (number of errors made in the experimental, incongruent-colour condition) entered as the dependent variables and group as the between subjects variable. The results of this analysis (Pillai’s Trace) revealed that the four groups differed significantly in terms of their executive function; $F(12, 129)=5.2,$
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Separate univariate ANOVA revealed that the groups differed on all measures of executive function; the F-A-S, $F(3, 44)=3.7$, $p<0.05$; Hayling’s, $F(3, 44)=14.9$, $p<0.001$; Stroop interference, $F(3, 44)=7.2$, $p<0.001$ and Stroop errors, $F(3, 44)=6.9$, $p<0.01$. Subsequent analyses, using Least Significant Difference tests, revealed that depressed NS patients produced fewer words on the F-A-S verbal fluency task, more errors on the Hayling’s and more errors on the Stroop than did the non-depressed NS patients or healthy controls, all tests $p<0.05$. Patients with MD produced significantly more words on the F-A-S and fewer errors on the Stroop than did the depressed NS patients, both tests $p<0.05$. However, patients with depression produced more errors on the Hayling’s and exhibited greater interference on the Stroop than did the healthy controls or non-depressed NS patients, all tests $p<0.05$). Furthermore, they produced significantly more errors on the Stroop than did the healthy controls, $p<0.05$. It is notable that non-depressed NS patients did not differ from the healthy controls on any of the measures of executive function, all tests $p>0.05$.

3.3. Emotion recognition accuracy

Initial inspection of the emotion recognition accuracy of the subgroups of the neurosurgically treated patients revealed that individuals that remained depressed following surgery and those that had subsequently recovered from their depressive episode exhibited equivalent levels of emotion recognition accuracy (depressed Mean=81.4%, SE=6.7; recovered Mean=81.3%, SE=5.3). Similarly, the emotion recognition accuracy of the patients treated with ACING (M=83.9%, SE=4.5) did not differ significantly from patients that had been treated with both ACING and ACAPS;
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(Mean=79.3%, SE=6.9), t(7)=0.6, p>0.05. With these findings in mind, the emotion recognition scores of neurosurgically treated patients were combined and compared with those of the MD participants and healthy controls.

Analysis of the participants’ emotion recognition accuracy (illustrated in table 3), using a 3 (group) x 7 (emotion) ANCOVA with participant age entered as a covariate, revealed no significant main effects of emotion or age, both tests F<1. There were also no significant age x emotion or group x emotion interactions; F<1 and F(6, 12)=1.3, p>0.05 respectively. However, there was a significant difference between the three groups in terms of their emotion recognition accuracy; F(2,44)=3.4, p<0.05. Subsequent analyses, using Least Significant Difference tests, revealed that neurosurgically treated patients identified significantly fewer emotional expressions correctly (Mean=81.3, Standard Error=4.2) than did the controls (M=89.9, SE=1.4), p<0.05. However, the percentage of emotional displays correctly identified by the patients with major depression (M=86.1, SE=2.3) did not differ from the controls or from the neurosurgically treated patients, both tests p>0.05.

4.4. Relationships between demographic, mood & executive variables and emotion recognition accuracy (total score on the TASIT emotion recognition test)

Inspection of the correlation matrix (illustrated in table 4) revealed that participants’ Stroop error scores were significantly negatively related to their emotion recognition accuracy; r(48)=-0.37, p<0.05. No other variables were significantly associated with emotion recognition accuracy. As the groups differed significantly on
emotion recognition accuracy and Stroop error scores it is difficult to draw any conclusions from this relationship. Therefore a hierarchical linear regression analysis was conducted in order to establish if emotion recognition accuracy could be predicted from Stroop error score, once the differences between the groups had been controlled for. Stroop error score was entered in the first level of the regression and this analysis resulted in a significant model that accounted for around 14% of the variance; $R^2=0.14$, $R^2_{\text{adjusted}}=0.12$; $F(1, 46)=7.4$, $p<0.01$. This analysis revealed that Stroop error score was a significant predictor of emotion recognition accuracy; $\text{Beta}=-0.37$, $p<0.01$. In order to establish if group membership predicted emotion recognition accuracy over and above the participants’ Stroop error scores participant group was entered into the second level of the hierarchical regression. However, as group was not a dichotomous variable three dummy variables were created, according to the procedure outlined in Field (2005), and it was these variables were entered into the regression. The result of this analysis revealed a non-significant model that accounted for around 18% of the variance; $R^2=0.18$, $R^2_{\text{adjusted}}=0.10$; $F(4, 43)=2.4$, $p=0.067$. The only variable that remained in the model was Stroop error score; $\text{Beta}=-0.3$, $p=0.077$. The change in $R^2$ was not significant; $F_{\text{change}}(3, 43)=0.72$, $p>0.05$.

4. Discussion

The aim of the present study was to examine whether therapeutic neurosurgical interventions for chronic, treatment refractory depression (ACING and combined ACING with ACAPS) were associated with subsequent deficits in emotion recognition. To this end, a group of neurosurgically treated patients were identified, as part of their long-term
clinical follow up, and tested on their ability to identify the primary emotions from a
series of videotaped social interactions. The performance of these patients was compared
with that of a group of non-neurosurgically treated patients with chronic unipolar
depression and a matched group of healthy, never-depressed, controls.

In line with predictions, patients that had received psychiatric neurosurgery
exhibited significantly impaired emotion recognition accuracy relative to healthy
controls. This result is consistent with Hornak et al (2003) who reported that patients with
circumscribed lesions to the anterior cingulate and orbitofrontal cortex exhibited marked
deficits in their ability to identify the primary emotions from faces and recorded voices.
The findings of the present study and those of Hornak et al (2003) are convergent with
the findings of a number of functional imaging studies implicating the ACC in successful
emotion recognition (Blair et al, 1999; Kilgore & Yurgelun-Todd, 2004).

Although there was no significant difference in the emotion recognition accuracy
of the patients treated with combined ACING & ACAPS and those treated with ACING
alone, inspection of the means revealed that the patients that received both interventions
correctly identified nearly 5% fewer emotional expressions than those that received
ACING only. Given the small numbers in these groups, it would be unwise to conclude
that ACAPS was not associated with additional impairment of emotion recognition.
Indeed, this remains plausible given the previous evidence from lesion (Hornak et al,
1996; 2003) and neuroimaging studies (Blair et al, 1999; Narumoto et al, 2000;
Vuilleumier et al, 2001) implicating OFC in successful emotion recognition. Further
research is required to assess the emotion recognition accuracy of patients that have
received an ACAPS procedure alone in order to provide more conclusive evidence
concerning the impact of ACAPS on emotion processing.

The current findings of a general impairment of emotion recognition in the neurosurgery
patients differ from the specific deficits in the recognition of fear and disgust that were
exhibited by the patient with ACC lesions reported by Wang et al (2002). However, the
specific impairment of fear recognition in their study could relate to the concomitant
damage to the amygdala that was also present in this patient. Evidence for this proposal
comes from numerous studies that have demonstrated that this region is critically
important in the recognition of fear (Adolphs et al, 1994; Adolphs et al, 1999; Broks et al,
1998; Calder et al, 1996; Phelps & Anderson, 1997; and Scott et al, 1997).

Contrary to predictions, there was no significant difference in emotion recognition
accuracy between the non-neurosurgically treated patients with chronic depression and
the healthy controls. This finding is inconsistent with a number of previous studies that
have reported a general deficit in emotion recognition accuracy in depressed participants
(Feinberg, Rifkin, Schaffer & Walker, 1986; Mikhailova et al, 1996; Persad & Polivy,
1993). However, it is convergent with several other studies that have reported no
evidence of emotion recognition deficits in depressed individuals (Archer et al, 1992;
Gaebel & Wölwer, 1992; Ridout et al, 2003). One factor that may account for the failure
to observe a significant difference between the depressed and healthy control samples is
the use of highly ecologically valid stimuli in the present study. All participants attained
above 80% recognition accuracy on the emotion identification task used in the present study, which suggests that it may have been an easier task than those used in previous studies. For example, some studies (Mikhailova et al, 1996; Gur et al, 1992) utilised schematic drawings of emotional faces rather than photographic images. Furthermore, a recent study demonstrating impaired emotion recognition in depressed patients presented the stimuli rapidly (Surguladze et al, 2004), which would have increased the task difficulty and thus increased recognition differences between the groups.

The emotion recognition performance of the neurosurgically treated patients did not differ significantly from that of the non-neurosurgically treated patients with depression, suggesting that the neurosurgery patients’ performance was not impaired relative to their probable pre-surgical ability. However, inspection of performance means revealed that the surgery patients correctly recognised around 5% fewer emotion expressions than did the depressed patients, suggesting that the surgery patients were not as accurate as the depressed patients at identifying the emotional displays. The lack of a significant difference between these two groups is likely to be a consequence of limited statistical power caused by the relatively small number of patients in the neurosurgery group. Future research should utilise a longitudinal design, testing patients both prior to and post their surgery in order to confirm the effects of neurosurgery on emotion recognition accuracy.

The results of the regression analysis revealed that overall emotion recognition accuracy could be predicted from the participants’ error score on the Stroop colour-
naming task. This finding is consistent with the proposal that emotion recognition deficits might be a consequence of general neuropsychological impairment (Albert et al, 1991). However, as emotion recognition accuracy was only significantly related to Stroop error score and not with any of the other neuropsychological tests, the current findings are not entirely consistent with this proposal. As the Stroop is considered to be the classic measure of controlled attention, then the present findings are consistent with previous work (Holmes et al, 2006; Pessoa et al, 2002; Pessoa, 2005; Vuilleumier, 2002) suggesting that intact emotional processing is dependent upon attentional control. For example, Pessoa et al (2002) demonstrated that brain regions involved in processing emotional faces, including the amygdala, were not activated automatically, but rather were selectively activated only when there were sufficient attentional resources available to process the faces. Thus, in the present study it is plausible, due to impaired attentional control, that sufficient resources were not made available to adequately process the faces. This, in turn, may have resulted in impaired recognition of the portrayed emotion. With this in mind, it is possible that the emotion recognition deficit exhibited by the surgery patients was a consequence of impaired cognitive control associated with lesions to the ACC. In line with this notion, there is consistent evidence that the anterior cingulate cortex (ACC) is critically involved in attentional control (Botvinick et al, 1999; Pardo, Pardo, Janer, & Raichle, 1990; Swick & Jovanovic, 2002; van Veen & Carter, 2002). Furthermore, previous studies have reported impaired attentional processes in patients that have received ACING (Cohen et al, 1999; Janer & Pardo, 1991; Ochsner et al, 2001; Turken & Swick, 1999). These findings must be interpreted with caution due to the small number of surgery patients and the fact that, after controlling for group membership, the
significance of Stroop error score as a predictor of emotion recognition accuracy dropped below conventional significance (p= 0.077). However, it is notable that it was the Stroop error score and not group membership that remained in the regression model.

The central finding of the present study - that patients treated with ACING or both ACING & ACAPS exhibit impaired emotion recognition - is noteworthy, as this deficit could have important implications for these patients’ social functioning. For example, Mueser et al (1996) reported that impaired emotion recognition in schizophrenic patients was associated with poorer social competence. Furthermore, Surguladze et al (2004) suggested a similar relationship exists in depressed patients. Impaired social competence could plausibly undermine important social bonds and thus represent a risk factor for future depressive episodes. Investigation of the relationship between emotion recognition, social competence and subsequent relapse represents an important avenue for future study.

Limitations of the present study include the fact that the performance of the surgery patients on the neuropsychological and emotion recognition tasks was not assessed prior to surgery. Additionally, we were only able to test a small number of participants, particularly in the surgery group. It is also not possible to discount the possibility of medication effects within the depressed and neurosurgically treated groups. It should be emphasised, however, that great care was taken in recruiting the clinical control group to resemble as closely as possible the neurosurgery patients. Furthermore, considerable effort was taken to ensure that the healthy controls were matched to the
clinical patients on all important demographic variables. It would also have been valuable to have been able to test patients that had received ACAPS alone. Future work to develop the findings of the present study should include the assessment of patients pre, as well as post, surgery; testing patients treated with ACAPS alone, and testing patients with lesions in other regions to compare the neuropsychological profile and emotion recognition accuracy of the different patient groups.

In summary, patients treated with neurosurgery, but not clinically depressed patients, were significantly impaired relative to the healthy controls in their ability to recognise the primary emotions from dynamic social displays. There was some evidence to suggest that this impairment was related to a deficit in attentional control, which in turn may have been a consequence of the surgical lesions to the anterior cingulate cortex.

References


http://people.cas.sc.edu/rorden/mricro.html.


Emotion recognition following neurosurgery


Figure 1.

Location (identified by Talairach coordinates) of the characteristic lesions for the Anterior Cingulotomy (ACING) procedure (Standard deviations are presented in parentheses)
Figure 2.

Location (identified by Talairach coordinates) of the characteristic lesions for the Anterior Capsulotomy (ACAPS) procedure (Standard deviations are presented in parentheses)
Table 1. Participant characteristics: mean age, sex ratio, mean years of education completed, IQ (estimated from the participants NART error score), depression (BDI & HADS_D) and anxiety scores (HADS_A) of the participants in the different groups (standard deviations are reported in parentheses).

<table>
<thead>
<tr>
<th></th>
<th>Depressed (MD) (N=18)</th>
<th>Controls (HC) (N=22)</th>
<th>NS (Depressed) (N=5)</th>
<th>NS (Recovered) (N=4)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>45.5 (7.6)</td>
<td>40.1 (7.6)</td>
<td>39.4 (4.3)</td>
<td>40.0 (6.0)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>4:13</td>
<td>5:17</td>
<td>2:3</td>
<td>0:4</td>
<td>n.s.</td>
</tr>
<tr>
<td>Y of FTE</td>
<td>12.4 (2.4)</td>
<td>13.8 (2.4)</td>
<td>13.0 (2.1)</td>
<td>15.0 (2.4)</td>
<td>n.s.</td>
</tr>
<tr>
<td>IQ</td>
<td>114 (4.8)</td>
<td>113 (5.3)</td>
<td>116 (2.0)</td>
<td>116 (3.2)</td>
<td>n.s.</td>
</tr>
<tr>
<td>HRSD</td>
<td>21.9 (2.9)</td>
<td>N/A</td>
<td>25.6 (2.5)</td>
<td>4.0 (1.4)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BDI</td>
<td>32.2 (6.2)</td>
<td>2.6 (2.5)</td>
<td>46.6 (9.8)</td>
<td>5.0 (6.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HADS_D</td>
<td>15.3 (3.1)</td>
<td>1.0 (1.6)</td>
<td>16.8 (3.8)</td>
<td>2.0 (1.8)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HADS_A</td>
<td>14.3 (2.8)</td>
<td>3.0 (2.8)</td>
<td>13.0 (2.0)</td>
<td>3.5 (3.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: NS = neurosurgically treated patients; HC = healthy controls; MD = patients with major depression; Y of FTE = years of full-time education completed; HRSD = Hamilton Rating Scale for Depression; BDI = 21-item Beck Depression Inventory; HADS_D = Hospital Anxiety and Depression Scale (Depression Subscale), HAD_A = Hospital Anxiety and Depression Scale (Anxiety Subscale)

* MANOVA; † univariate ANOVA

p<0.001 a,b,d–p; p<0.05 c
Table 2. Mean F-A-S verbal fluency, Hayling’s error, Stroop interference and Stroop error scores of the participants in the four groups (standard errors are reported in parentheses).

<table>
<thead>
<tr>
<th></th>
<th>Depressed (MD) (N=18)</th>
<th>Controls (HC) (N=22)</th>
<th>NS (Depressed) (N=5)</th>
<th>NS (Recovered) (N=4)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FAS verbal fluency</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>41.8 (3.1)a</td>
<td>44.6 (2.2)b</td>
<td>28.8 (5.4)abc</td>
<td>51.8 (3.6)c</td>
<td>P&lt;0.05</td>
</tr>
<tr>
<td><strong>Hayling’s Error Score</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2.8 (0.5)d</td>
<td>6.3 (0.2)def</td>
<td>3.4 (1.0)f</td>
<td>3.5 (1.0)f</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td><strong>Stroop Interference</strong></td>
<td>132.4% (16.3)gh</td>
<td>62.6% (4.1)i</td>
<td>96.7% (27.1)</td>
<td>77.7% (15.5)h</td>
<td>P&lt;0.001</td>
</tr>
<tr>
<td><strong>Stroop Error score</strong></td>
<td>4.5 (1.0)jk</td>
<td>2.1 (0.5)ij</td>
<td>9.2 (1.4)ijkl</td>
<td>4.3 (1.6)jk</td>
<td>P&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: NS = neurosurgically treated patients; HC = healthy controls; MD = patients with major depression; Y of FTE = years of full-time education completed; HRSD = Hamilton Rating Scale for Depression; BDI = 21-item Beck Depression Inventory; HADS_D = Hospital Anxiety and Depression Scale (Depression Subscale), HAD_A = Hospital Anxiety and Depression Scale (Anxiety Subscale)

P<0.05 a, b, i, k; P<0.01 b, c, e, f; P<0.001 d, g, j
Table 3. Percentage of each type of emotional expression correctly identified as a function of participant group

<table>
<thead>
<tr>
<th>Type of emotional expression</th>
<th>Depressed (MD) (N=17)</th>
<th>Controls (HC) (N=22)</th>
<th>Neurosurgery (NS) (N=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Happiness</td>
<td>Mean: 77.9% ± 6.7 SE</td>
<td>Mean: 96.6% ± 1.9 SE</td>
<td>Mean: 75.0% ± 7.2 SE</td>
</tr>
<tr>
<td>Surprise</td>
<td>Mean: 88.2% ± 4.8 SE</td>
<td>Mean: 93.2% ± 2.4 SE</td>
<td>Mean: 81.8% ± 5.0 SE</td>
</tr>
<tr>
<td>Neutral affect</td>
<td>Mean: 75.0% ± 7.1 SE</td>
<td>Mean: 81.8% ± 5.0 SE</td>
<td>Mean: 80.6% ± 9.1 SE</td>
</tr>
<tr>
<td>Sadness</td>
<td>Mean: 85.3% ± 3.7 SE</td>
<td>Mean: 87.5% ± 3.6 SE</td>
<td>Mean: 77.8% ± 7.7 SE</td>
</tr>
<tr>
<td>Anger</td>
<td>Mean: 88.2% ± 4.4 SE</td>
<td>Mean: 81.8% ± 4.4 SE</td>
<td>Mean: 80.6% ± 6.9 SE</td>
</tr>
<tr>
<td>Anxiety</td>
<td>Mean: 91.2% ± 4.8 SE</td>
<td>Mean: 92.0% ± 3.8 SE</td>
<td>Mean: 75.0% ± 7.2 SE</td>
</tr>
<tr>
<td>Disgust</td>
<td>Mean: 97.1% ± 2.1 SE</td>
<td>Mean: 96.6% ± 1.9 SE</td>
<td>Mean: 91.7% ± 4.2 SE</td>
</tr>
<tr>
<td>TOTAL*</td>
<td>Mean: 86.1% ± 2.3 SE</td>
<td>Mean: 89.9% ± 1.4 SE</td>
<td>Mean: 81.3% ± 4.2 SE</td>
</tr>
</tbody>
</table>

Abbreviations: SE=standard error, MD=major depression, HC=healthy controls, NS=Neurosurgery

* Significant effect of group on emotion recognition accuracy; F(2,44)=3.4, p<0.05

a p<0.05