Eur Arch Psychiatry Clin Neurosci (1996) 246:165–170

ORIGINAL PAPER

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Facial affect recognition in the course of schizophrenia

Received: 6 July 1995 / Accepted: 6 December 1995

Abstract Deficits in facial affect recognition have been shown repeatedly in schizophrenia. However, the stability of this deficit over time remains to be clarified. A total of 36 remitted, 32 acutely ill schizophrenic patients and 21 healthy volunteers participated in a cross-sectional and longitudinal study. All subjects were assessed twice within 4 weeks (acute schizophrenics and normal controls), or 12 weeks, respectively (remitted schizophrenics). Subjects had to identify six basic emotions from corresponding facial expressions shown as photographs on a video screen. Both acute and remitted schizophrenics demonstrated a stable deficit over time in facial affect recognition unrelated to psychopathology and medication. This suggests that deficits in facial affect recognition in schizophrenia reflect a trait-like, rather than a state-dependent, characteristic.

Key words Facial affect recognition · Trait marker · Schizophrenia

Introduction

There have been many studies investigating facial affect recognition in psychiatric patients during the past three decades. In schizophrenics, especially those with negative symptoms, this interest resulted at least partially from the hypothesis that difficulties in *decoding* facial affect might contribute to schizophrenics' known deficit in *encoding* emotional expression (Borod et al. 1993). Whereas several studies employing different stimuli and various task requirements have demonstrated that schizophrenic patients have deficits in facial affect recognition (e.g. Borod et al. 1993; Heimberg et al. 1992; Gaebel et al. 1989; Gaebel and Wölwer 1992; Schneider et al. 1992; for an overview on methods and results up to 1988 see Morrison et al. 1988), the nosological specificity, task specificity and especially the temporal stability of this deficit have remained less clear.

With respect to nosological specificity, it has been shown that major depressives either have no comparable deficit (Archer et al. 1992; Cutting 1981; Gaebel and Wölwer 1992; Gessler et al. 1989; Schneider et al. 1992; Walker et al. 1984) or range between schizophrenic patients and normal controls (Feinberg et al. 1986). Only one study showed that depressives perform as poorly as schizophrenics (Zuroff and Colussy 1986). Because anxiety neurotics also showed no recognition deficit (Mandal and Palchoudhury 1989; Mandal and Rai 1987), this led to the conclusion that deficits in facial affect recognition are limited to schizophrenics.

Functional models of face processing (e.g. Bruce and Young 1986) postulate that three are several separable functional components involved in face processing, such as structural encoding, physical analysis, facial speech analysis and expression analysis. It is still controversial whether schizophrenics' deficit in facial affect recognition specifically concerns the process of expression analysis, or reflects a more generalised deficit in face processing, or is due to cognitive dysfunctions not specially related to face processing. Whereas some authors conclude from their results that there is a specific deficit in facial affect recognition (Berndl et al. 1986a, Cutting 1981; Walker et al. 1984), others have found evidence for a generalised deficit (Archer et al. 1992; Gessler et al. 1989; Kerr and Neale 1993), or for a prominent affect-specific deficit in the context of a generalized information processing deficit (Borod et al. 1993; Feinberg et al. 1986; Heimberg et al. 1992).

The stability of the deficit over time has not been adequately investigated to date. Cross-sectional studies by Cutting (1981) and Gessler et al. (1989) only found facial affect recognition deficits only in acute, but not in remitted, schizophrenics. On the other hand, two longitudinal studies by Gaebel et al. (1989) and Gaebel and Wölwer

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(1992) revealed stable deficits in acute schizophrenics over the period of their hospital stay or over a period of 4 weeks, respectively, despite clinical improvement. However, in both studies there were also (nonsignificant) trends of a greater improvement over time in schizophrenics compared with normal controls. This suggested perhaps that the recognition deficit might have disappeared over a longer time period, i.e. after full remission. In the present study we therefore compared the performance of remitted schizophrenics assessed twice within 3 months with that of acute schizophrenics and normal controls. The two main questions to be answered were: (a) Do remitted schiozophrenics show a facial affect recognition deficit like acute schizophrenics (interindividual cross-sectional comparison), and (b) Do these deficits, if confirmed, persist in the course of the illness (intraindividual longitudinal comparison)?

Subjects and methods

A total of 36 remitted schizophrenic patients from a psychiatric day hospital (-S/r-, 17 females and 19 males; mean age ± SD 35.9 ± 8.8 years), 32 acute schizophrenic inpatients (-S/a-, 10 females and 22 males; mean age ± SD 31.7 ± 10.6 years) and 21 healthy volunteers (-N-, 6 females and 15 males; mean age ± SD 34.2 ± 10.0 years) participated in the study after informed consent.

All subjects were assessed twice: in S/a assessment took place within 3 days after admission (T0) and after 4 weeks of neuroleptic treatment (T1). N were assessed twice within 4 weeks as well. According to the assumption that any changes in performance of remitted patients – if at all – would occur only after a time period longer than 4 weeks, remitted patients were assessed at least 4 weeks after discharge from an actue ward (T0´) and three months later (T1´).

All patients were diagnosed according to Research Diagnostic Criteria (Spitzer et al. 1978). Subjects with organic brain damage, drug abuse and subnormal intelligence were excluded from the study. In addition, remitted schizophrenics relapsing during the observation period were also excluded.

Assuming that patients with marked negative symptoms, especially affective flattening, have the poorest facial affect recognition (Borod et al. 1993), a subgroup of the acute schizophrenics with persisting affective flattening at T1 (7 patients: 5 males and 2 females; mean age ± SD 27.7 ± 9.9 years) were assessed a third time 8 weeks after admission (T2). Inclusion criteria for this group were a minimum score of 4 in at least one item of the SANS-subscale "affective flattening" and a minimum sumscore of 16 in the five items reflecting reduced "affective expression" ("unchanging facial expression", "decreased spontaneous movements", "paucity of expressive gestures", "affective nonresponsivity" and "lack of vocal inflections").

Fourteen S/a and three S/r were first-episode patients. The remaining patients had mean illness durations of 6.7 ± 6.9 years and 8.9 ± 7.8 years, respectively, with a mean number of 5.0 ± 4.4 and 5.3 ± 3.4 previous schizophrenic episodes.

The S/a were orally treated with either perazine (n = 20) or haloperidol (n = 12). The average daily dosage in chlorpromazine equivalents (CPZE) in the T0–T1 interval did not differ significantly (perazine: 436 ± 217 mg CPZE; haloperidol: 531 ± 313 mg CPZE). Among S/r 10 patients were treated with clozapine (mean daily dosage = 426 ± 144 mg CPZE), 21 received typical neuroleptic drugs either orally or as depot (mean daily dosage = 477 ± 430 mg CPZE) and 5 patients were drug-free in the T0´–T1´ interval. Five S/a, but none of the S/r, received anticholinergic medication.

Procedure

Facial affect recognition performance was assessed using videotaped photographs of the "pictures of facial affect" (Ekman and Friesen 1976). Twelve (2 × 6) photographs depicting 1 male and 1 female poser, each displaying one of six basic emotions (fear, anger, surprise, happiness, disgust and sadness) were presented to the subjects. The pictures were chosen from the series according to their reported high interrater reliability (88–100%) for emotion decoding on each emotion displayed by a single poser. Each face was presented for 8 s. Subjects then had to select the appropriate emotion from a list containing the six emotion labels and a neutral state inserted beside the face for an additional 10 s. Display time was chosen from the upper range of Kirouac and Doré's (1983) data, in which normal subjects required 3–7 s for emotion recognition.

The "number of correct answers" (NC) summed over all 12 pictures was used as a measure of recognition performance.

Clinical assessment

The Brief Psychiatric Rating Scale (BPRS; Overall and Gorham 1962), the Scale for the Assessment of Negative Symptoms (SANS; Andreasen 1982) and the Extrapyramidal Side Effects Rating Scale (EPS; Simpson et al. 1970) were used to assess clinical course in schizophrenic patients. The assessment was performed by two trained raters.

The BPRS subscores "activation", "hostility" and "thought disorder" were summed (BPRS-S) to monitor the degree of positive symptoms. As measures of negative symptoms the SANS "summary score" (SANS-SS, sum of global ratings; Andreasen 1982) and the BPRS subscale "anergia" (BPRS-ANER) were used. Symptoms of anxiety and depression were assessed using the BPRS subscale "anxiety/depression" (BPRS-ANDP).

Data analysis

The analysis of recognition performance was performed in two steps:

1. A cross-sectional comparison of the three groups at their first time of assessment (T0/T0´) was performed using a one-way ANOVA.

2. The time course of recognition performance over the 4-week T0–T1 interval was examined by a 2 × 2 MANOVA (group × time) for S/a and N, and separately for S/r during the longer T0´–T1´ interval by paired t-test.

When necessary, Tukey's Honestly Significant Differences (HSD) test was used for post hoc comparisons of group means. In order to adjust the nominal error probability of $\alpha = 0.05$ for the five F- and t-tests, each of these tests was carried out with an adjusted error probability of $\alpha = 0.05/5 = 0.01$. All further comparisons were only computed for descriptive and exploratory purposes without α-adjustment.

The data analysis for the 7 patients with persisting affective flattening at T1 was performed in two steps as well: In order to get an idea of how representative the small subgroup was for the larger sample of S/a, the data of these 7 patients were compared firstly with the data of the rest of the S/a group with between subject t-test, and secondly, the time course of clinical and performance data during the 4-week T1–T2 interval was examined using paired t-test.

Results

Clinical course

After 4 weeks of neuroleptic treatment, S/a were significantly improved in positive symptoms (BPRS-S), in BPRS-

Table 1 Psychopathological ratings (group means ± SD) for acute schizophrenics at T0 and T1 and remitted schizophrenics at T0′ and T1′, respectively, and results of separate paired *t*-tests (T0 vs T1 and T0′ vs T1′). BPRS-S sumscore of subscales "activation", "hostility", and "thougth disorder" (range 10–70); BPRS-ANDP subscale "anxiety/depression" (range 4–28); BPRS-ANER subscale "anergia" (range 4–28); SANS-SS summary score (sum of global ratings of "affective flattening", "alogia", "avolition", anhedonia", and "attentional impairment"; range 0–25); EPS sumscore of all nine items (range 0–27)

	Acute schizophrenics			Remitted schizophrenics		
	T0	T1	t/p	T0′	T1′	t/p
BPRS-S	25.72 ± 8.13	17.61 ± 4.77	5.27***	12.29 ± 3.21	13.63 ± 4.44	–2.40*
BPRS-ANDP	7.69 ± 3.14	6.65 ± 2.47	2.38*	6.58 ± 3.09	6.44 ± 3.33	0.28
BPRS-ANER	10.84 ± 3.97	9.53 ± 3.42	2.01(*)	10.11 ± 3.08	10.02 ± 3.08	0.15
SANS-SS	13.63 ± 5.00	11.56 ± 4.52	2.53*	9.31 ± 4.15	8.69 ± 5.00	0.94
EPS	0.72 ± 1.65	3.00 ± 4.16	–3.18**	0.94 ± 1.88	1.08 ± 2.22	–0.33

***$p < 0.001$
**$p < 0.01$
*$p < 0.05$
(*) $p < 0.10$

Table 2 Number of correct answers in facial affect recognition (group mens ± SD) for acute schizophrenics (S/a) and normal controls (N) at T0 and T1 and remitted schizophrenics (S/r) at T0′ and T1′, respectively

	T0/T0′			T1/T1′		
	S/a	S/r	N	S/a	S/r	N
Total (NC, 12 pictures)	6.41 ± 4.15	7.35 ± 3.14	9.43 ± 1.86	8.56 ± 1.66	7.97 ± 2.88	9.76 ± 1.14
Happiness (2 pictures)	1.42 ± 0.88	1.83 ± 0.50	2.00 ± 0.00	2.00 ± 0.00	1.83 ± 0.50	2.00 ± 0.00
Surprise (2 pictures)	1.34 ± 0.86	1.53 ± 0.69	1.86 ± 0.48	1.87 ± 0.33	1.55 ± 0.73	1.90 ± 0.30
Fear (2 pictures)	1.03 ± 0.86	1.08 ± 0.87	1.38 ± 0.74	1.06 ± 0.84	1.08 ± 0.87	1.47 ± 0.68
Anger (2 pictures)	0.81 ± 0.82	1.00 ± 0.86	1.52 ± 0.60	1.22 ± 0.70	1.25 ± 0.77	1.62 ± 0.60
Disgust (2 pictures)	1.15 ± 0.92	1.30 ± 0.78	1.76 ± 0.44	1.62 ± 0.55	1.50 ± 0.81	1.95 ± 0.22
Sadness (2 pictures)	0.62 ± 0.61	0.78 ± 0.68	0.90 ± 0.70	0.78 ± 0.55	0.75 ± 0.60	0.81 ± 0.60

ANDP and in negative symptoms (SANS-SS and BPRS-ANER; see Table 1). On the other hand, extrapyramidal symptoms (EPS) increased significantly during the T0–T1 interval.

The S/r group turned out to be very stable in terms of psychopathology throughout the observation period of 3 months. Although they almost did not show any positive symptoms, symptoms of anxiety or depression, and no notable extrapyramidal symptoms at T0′ and at T1′, respectively, they exhibited negative symptoms comparable to the T1-level of the acute sample. Due to the very low symptom level, a statistically significant increase in BPRS-S from T0′ to T1′ appears to be clinically irrelevant.

The 7 patients of the S/a group with persisting symptoms of affective flattening at T1 not only showed more negative symptoms (SANS-SS: $t_{30} = -2.90$, $p = 0.007$; BPRS-ANER: $t_{30} = -2.47$, $p = 0.019$) compared with the rest of the S/a group at T1, but were more anxious-depressive as well (BPRS-ANDP: $t_{30} = -3.25$, $p = 0.003$). On the other hand, there was no difference in the extent of positive symptoms between the two subgroups of S/a at T1. The subgroup with flattened affect at T1 improved significantly during the T1–T2 interval regarding positive (BPRS-S: $t_6 = 3.35$, $p = 0.015$) as well as negative symptoms (SANS-SS: $t_6 = 5.0$, $p = 0.002$).

Facial affect recognition

With respect to performance in the facial affect recognition task, the cross-sectional group comparison of NC at T0 revealed a significantly poorer performance for S/a as well as S/r compared with N (see Table 2; main effect group: $F_{2,86} = 5.26$, $p = 0.007$). The longitudinal analysis for the T0–T1 interval on average showed an improvement over time for S/a and N (main effect time: $F_{1,51} = 9.22$, $p = 0.004$), pointing to a practice effect. This general improvement appeared to be slightly more pronounced in S/a than in N (interaction group × time: $F_{1,51} = 4.94$, $p = 0.031$), but failed to reach statistical significance compared with the adjusted $\alpha = 0.01$. However, irrespective of this improvement single comparisons of group means at T1 confirmed a significantly poorer performance of S/a compared with N, even under partly remitted conditions. For S/r no significant change in recognition performance during the 3-month period of the T0′–T1′ interval could be shown ($t_{35} = -1.27$, $p = 0.21$).

A highly significant retest reliability of $r_{T0', T1'} = 0.76$ ($p < 0.001$) further confirmed the high stability of the recognition performance in S/r. Reliability scores for S/a and N were also significant, but lower in value (S/a; $r_{T0, T1} = 0.56$, $p = 0.001$; N: $r_{T0' T1} = 0.48$, $p = 0.029$). A data inspection revealed that in S/a this lower reliability was due mainly to a very heterogeneous improvement in facial affect recognition of those 9 patients showing the poorest

performance at T0 (NC at T0: 0–2; NC at T1: 5–10). In N the lower reliability score mainly resulted from their comparably small performance range both at T0 and at T1 (NC at T0: 7–12; NC at T1: 8–12).

The 7 S/a patients with marked affective flattening showed a T1 performance not significantly different from that of the rest of the S/a group ($t_{30} = 0.99, p = 0.72$). During the T1–T2 interval no further improvement in facial affect recognition ($t_6 = -0.35, p = 0.74$) could be shown.

Intervening variables

Regarding the *type of expressed emotion* as a possible intervening variable in recognition performance, exploratory analyses (one-way ANOVA at T0/T0´) revealed group differences between normals and schizophrenics for all emotions, except "fear" and "sadness" (happiness: $F_{2,86} = 6.09, p = 0.003$; surprise: $F_{2,86} = 3.22, p = 0.045$; anger; $F_{2,86} = 5.25, p = 0.007$; disgust: $F_{2,86} = 4.02, p = 0.022$; fear: $F_{2,86} = 1.21, p = 0.30$; sadness; $F_{2,86} = 1.18; p = 0.31$). Although all pictures used were reported to have high interrater reliability in normals (Ekman and Friesen 1976), the pictures showing these two emotions were found to be only poorly recognized in general, even by normals (see Table 2). Therefore, to the lower discriminatory power of these emotions may at least be due partly to bottom effects.

Pearson correlations of recognition performance with *psychopathology* were inconsistent across time and groups. In particular, no stable relationship with negative symptoms could be shown (correlation between NC and SANS-SS at T0/T0´: S/a $r = -0.49, p = 0.005$; S/r: $r = -0.08, p = 0.66$; at T1/T1´; S/a: $r = -0.26, p = 0.15$; S/r: $r = -0.36, p = 0.03$).

In order to get an idea of the effect of *medication* on facial affect recognition, pearson correlations of the recognition performance with the neuroleptic dosage at the day of assessment, with the mean daily dosage during the observation interval, and with extrapyramidal side effects (EPS), were calculated. No coefficient exceeded $r = |0.2|$ and no correlation proved to be statistically significant. Furthermore, no effect of the kind of neuroleptic drug received during the observation interval (S/a: perazine vs haloperidol; S/r: clozapine vs typical neuroleptics vs no medication) could be found. Finally, S/a without premedication at T0 did not differ from premedicated patients regarding recognition performance at T0 and the time course of recognition performance during the T0–T1 interval.

Discussion

The present study confirms previous findings on facial affect recognition deficits in schizophrenia. Moreover, this deficit proved to be stable over time and at different stages of the illness. In accordance with our previous results (Gaebel et al. 1989; Gaebel and Wölwer 1992) deficits in facial affect recognition in acute schizophrenics persisted during the short-term course of their hospital stay, despite a significant improvement in psychopathology and a slight improvement in performance. Remitted schizophrenics exhibited comparable impairments, which remained stable over the 3-month period of observation. This confirms a recent report by Addington et al. (1994) who found facial affect recognition deficits in schizophrenics to be relatively stable from the inpatient phase of the illness to a period of relative remission 12–16 weeks after discharge. On the other hand, the results are in contradiction to cross-sectional findings of Cutting (1981) and Gessler et al. (1989) who found deficits only in acute, but not in remitted, schizophrenics. Besides that the interpretation of Cutting's results is difficult because he had not included a nonpsychiatric control group, this discrepancy might be due to differences in the kinds of stimuli and task conditions used: in both studies subjects had only to distinguish between happy and sad (Gessler et al. 1989) or more or less friendly faces (Cutting 1981), respectively. However, differences between schizophrenics and normals have been reported to be smaller for the recognition of positive emotions than of negative emotions, or are even limited to negative emotions (Borod et al. 1993; Dougherty et al. 1974; Garfield et al. 1987; Kline et al. 1992; Mandal and Rai 1987; Muzekari and Bates 1977). Only few studies have not found this effect of emotional valence (Heimberg et al. 1992; Walker et al. 1980; Zuroff and Colussy 1986), which possibly reflects the less complexity and the more frequent occurrence in everyday life of positive compared with negative facial expression (Ekman et al. 1972; Zuckerman et al. 1975). Moreover, in our study facial expressions of "sadness" were most poorly recognized, demonstrating that not even all kinds of negative emotions necessarily contribute to schizophrenics' overall recognition deficit. Accordingly, facial expressions of friendliness or happiness and sadness as used by Cutting (1981) and Gessler et al. (1989) may have less discriminatory power than other emotions (Novic et al. 1984), because happiness is often almost perfectly recognized and sadness is often poorly recognized, in general.

Further evidence for a high temporal stability of the facial affect recongition deficit in schizophrenia has been reported by Walker et al. (1980): They have shown that schizophrenic children (8–12 years) and adolescents (13–19 years) already perform as poorly as adult schizophrenics (20–50 years) in identifying facial expressions compared with age-matched normal controls. Similar results have also been found by Berndl et al. (1986b) for schizophrenic adolescents and adults. Taken together with the current results, facial affect recognition deficits in schizophrenia may thus reflect a vulnerability linked trait marker, rather than a state-dependent characteristic. However, investigations of relatives of patients with schizophrenia are needed to verify this judgement.

In support of this conclusion no stable relationship to psychopathology could be found. In particular, our data do not confirm the hypothesis that the deficit in facial affect recognition is related to negative symptoms or con-

fined to a subgroup of schizophrenics with flattened affect (Borod et al. 1993): The group of acute schizophrenics with marked symptoms affective flattening at T1 demonstrated a performance level comparable to the rest of the S/a sample, and in accordance with the findings of Novic et al. (1984) there was no correlation of facial affect recognition with clinical ratings of affective flattening. A dissociation between skills in affect recognition and affect expression has been reported from studies in normals (Fridlund et al. 1987) and brain-damage subjects (Borod et al. 1986) as well, indicating that "these processing models involve minimally overlapping or even separate systems" (Borod et al. 1993; see also Feyereisen 1986).

Little is known of the neural basis of facial affect recognition deficits in schizophrenia. As already indicated in the discussion of the functional specificity of the deficit, different systems participating in a distributed neural network involved in facial affect recognition, face perception and visual scanning may possibly be disturbed: There is convincing evidence that the inferior temporal cortex plays an important role in the recognition of faces in humans (Allison et al. 1994; Sergent et al. 1992) and in nonhuman primates (Perrett 1985; Desimone 1991). Moreover, research with patients with neurological disorders has indicated "that the frontal lobe does indeed have a role in facial recognition, at least under some social constraints" (Kolb 1989). Kolb postulates a neural system regulating emotional behaviour in which the orbitofrontal cortex and the amygdala are crucial parts. The amygdala serves as a node in the network where socially relevant visual information is processed (Kolb 1990; LeDoux 1994). Given that facial affect is socially relevant visual information, some empirical support for Kolb's assumption is provided by Leonard (1985) who found face-selective cells in the amygdala of monkeys. With respect to visual scanning processes, both Goldman-Rakic (1988) and Mesulam (1990) focus on the prefrontal cortex and the posterior parietal cortex. Mesulam (1990) integrates the neural processing of planning and guidance of exploratory eye movements by these cortical structures into the concept of a large-scale neural network for the distribution of directed attention.

Of the neural systems probably involved in decoding of facial affect, the one which is primarily disturbed in schizophrenia remains unclear to date. Further studies using functional neuroimaging methods should help to elucidate this.

Although the relationship between face recognition, facial affect recognition and social skills has never been examined explicitly in schizophrenia (Ennis and Whelton 1994), facial affect recognition is obviously important in social communication. Schizophrenics' limited capability concerning these skills may thus be a contributing factor to the poor social performance of schizophrenic patients (Bellack at al. 1989). Because the deficit appears to be unrelated to clinical course and neuroleptic medication, a specific training in facial affect recognition, which could be incorporated into existing behaviour therapy programs, such as social skills training (Liberman et al. 1982) or integrated psychological therapy (Brenner et al. 1992), might be beneficial.

Acknowledgements This research project was granted in part by the German Ministry for Science and Technology (BMFT grant no. 701636). The study was conducted at the Department of Psychiatry, Free University of Berlin, the department all authors except M. Streit were affiliated with at that time. The authors thank Prof. Dr. H. Helmchen (Head of the Department of Psychiatry) and PD Dr. St. Priebe (Head of the Department of Social Psychiatry) for their support in studying patients from their departments.

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