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Facial Expression and Emotional Face Recognition in Schizophrenia and Depression

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Summary. Twenty-three acute schizophrenics, 21 acute major depressives (Research Diagnostic Criteria), and 15 normal controls participated in a study on facial expression and emotional face recognition. Under clinical conditions, spontaneous facial expression was assessed according to the affective flattening section of the Scale for the Assessment of Negative Symptoms. Under experimental laboratory conditions involuntary (emotion-eliciting interview) and voluntary facial expression (imitation and simulation of six basic emotions) were recorded on videotape, from which a raterbased analysis of intensity or correctness of facial activity was obtained. Emotional face recognition was also assessed under experimental conditions using the same stimulus material. All subjects were assessed twice (within 4 weeks), controlling for change of the psychopathological status in the patient groups. In schizophrenics, neuroleptic drug influence was controlled by random allocation to treatment with either haloperidol or perazine. The main findings were that schizophrenics and depressives are characterized by different quantitative, qualitative, and temporal patterns of affect-related dysfunctions. In particular, schizophrenics demonstrated a trait-like deficit in affect recognition and in their spontaneous and voluntary facial activity, irrespective of medication, drug type and dosage, or extrapyramidal side-effects. In depressives a stable deficit could be demonstrated only in their involuntary expression under emotion-eliciting interview conditions, whereas in the postacute phase a reduction in their voluntary expression became apparent. Differences in patterns of affect-related behavioral deficits may reflect dysfunctions in different underlying psychobiological systems.

Key words: Schizophrenia – Depression – Facial expression – Emotional face recognition

Introduction

In both schizophrenia and depression disturbances of affect and/or mood are an important component of the clinical syndrome. According to DSM-III-R (APA 1987), affect is defined as "a pattern of observable behaviors that is the expression of a subjectively experienced feeling state (emotion)", whereas mood is "a pervasive and sustained emotion". In particular, the definition of expression includes "facial action, pitch of voice, and hand and body movements". A limited number of "basic emotions" seems to be linked to innate facial reaction patterns, explaining why individuals even from different cultures share a common set of feelings and expressions and hence can judge one another's emotions (Ekman et al. 1972). From social psychology and clinical neurology it is known, however, that facial expression is not necessarily related to a corresponding emotion. Additionally, because of their different neuroanatomical organization a "double dissociation" (Teuber 1955) between voluntary and emotional facial movements can be demonstrated, i.e. either can be interrupted or disturbed by neurological damage while the other remains intact (Rinn 1984). Hence, the three compounds of the psychophysiological triad (Lolas 1988) – subjective feeling, expressive motor activity, and their physiological correlates – should be assessed separately and objectively to identify illness-related patterns of affective/emotional dysfunction, particularly in schizophrenia (Holzman 1988).

Reduced facial expression is one sign of affective flattening (Andreasen 1982), a core characteristic of negative symptoms in schizophrenia (Crow 1985). However, schizophrenics' facial expressivity differs from normals' not only quantitatively but also qualitatively (Heimann and Spoerri 1957, Spoerri and Heimann 1957, Pitman et al. 1987, Steimer et al. 1988, Schneider et al. 1990). It is still unknown whether schizophrenics' deviations in facial muscular activity are due to an emotional deficit, an intentional/motor deficit, or both. Since the facial expression of retarded depressives may look very much alike (Kulhara and Chadda 1987, De Bosset and Shaul 1989), the question of nosological specificity has also to be raised. Moreover, the contribution of (post-) psychotic depression and drug-induced parkinsonism to reduced facial expressivity in schizophrenics has to be taken into account (Prosser et al. 1987, Siris et al. 1988).

In addition to an "encoding" deficit a "decoding" deficit concerning facial expression has been demon-

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strated in schizophrenia (for an overview see Morrison et al. 1988). Although it has been assumed that both of these deficits may contribute to the social skills deficits in schizophrenic patients (Bellack et al. 1989), their co-occurrence and interrelationship have not yet been assessed. Decoding of inner emotion-related cues and their integration into the subjective experience of emotional deficits (Liddle and Barnes 1988), is a related subject to be considered.

These different findings have not yet been brought together in a systematic fashion. According to contemporary perceptual motor theories of emotion, these findings might be interpreted as dysfunctions of interacting perceptive, voluntary and involuntary motor processes ("schematic", "conceptual", and "expressive" mechanism; Leventhal 1980). In order to generate hypotheses concerning the psychobiological basis of affective/emotional disturbances in schizophrenia and depression, both involuntary and voluntary motor aspects of facial activity as well as facial affect recognition and subjective experience of deficits have been assessed in the present exploratory study, which is part of a larger research project on emotional dysfunctions in schizophrenia (Gaebel and Renfordt 1988). Illness course and medication have been taken into account, to control for state/trait dependence and drug influence.

Methods

Subjects

Subjects included in the study were 23 schizophrenics (S; 17 male, 6 female; mean age: 31.3 years) and 21 patients with major depressive disorder (D; 12 male, 9 female; mean age : 39.0 years) diagnosed according to Research Diagnostic Criteria (Spitzer et al. 1978). Exclusion criteria were organic brain disease, drug abuse, and subnormal intelligence. Fifteen healthy volunteers (N; 9 male, 6 female; mean age: 31.0 years) were included as controls.

All psychiatric subjects were inpatients after acute clinical admission. Assessments took place within 3 days after admission (T0) and 4 weeks thereafter (T1). Normal controls were also assessed twice. Eleven out of 23 S and 9 out of 21 D were never medicated before, 3 S and 3 D were not medicated for at least 3 months, 9 S and 9 D had drug washout periods ranging from 3 to 60 days. Schizophrenics were randomly allocated at T0 to open oral treatment with either perazine or haloperidol for four weeks. Mean daily and cumulative dosages were calculated in milligram chlorpromazine-equivalents (mg CPZ). Depressives were treated with various antidepressants; depressives on neuroleptics were excluded.

Procedure

After informed consent a clinical interview was accomplished in psychiatric patients. During the experimental session all subjects participated in the same standardized tasks. First a semistructured (experimental) interview took place, focusing on present or past good and bad experiences to elicit corresponding positive and negative emotions. This interview was conducted via a videolink, i.e. both interviewer and interviewee were located in different rooms, facing and hearing each other via an audiovisual link. Thereafter a prerecorded video was presented with the following tasks:

– Emotional face recognition:
All subjects were exposed for 8 s each to 2 × 6 still videos of pictures of facial affect of a male and a female poser (Ekman and Friesen 1976), each expressing six basic emotions (happiness, sadness, surprise, fear, disgust, anger). 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. After 8 s the picture was supplemented at its right margin for another 10 s by a list containing the names of these six emotions and an emotional neutral state, from which the subjects had to choose the appropriate one by naming.
– Imitation:
In order to assess an externally guided voluntary motor response, the same 2 × 6 emotional faces were exposed again for 8 s each, during which the subjects were asked to imitate the shown facial expression as correct as possible.
– Simulation:
According to the names of the six basic emotions, which were shown on the video screen for 8 s each, the subjects were requested to simulate the corresponding facial expression as if they were in the respective emotional state. This task should assess a more internally guided voluntary motor response.

Assessment

– Behavioral recording setup:
At T0 and T1 experimental assessments took place in a special laboratory for basic behavioral research (Gaebel 1990). Subjects were comfortably seated in front of a videomonitor, on which the tasks described above were displayed. While performing the tasks, the subjects' facial as well as hand/body movements were recorded separately by two video cameras. Both visual informations together with the task displayed were stored simultaneously on videotape for subsequent analysis using a split videoscreen (findings of simultaneously recorded eye movements and EEG will be reported elsewhere).
– Clinical assessment:
For clinical assessment at T0 and T1, which was accomplished on the ward by a single trained rater, the following rating scales were used in S and D respectively:
- Brief Psychiatric Rating Scale (BPRS, Overall and Gorham 1962)
- Scale for the Assessment of Negative Symptoms (Sans, Andreasen 1982)
- Hamilton Depression Ratingscale (HAM-D, 21-item version, Hamilton 1967)
- Extrapyramidal Side Effects Ratingscale (EPS, Simpson et al. 1970)

Data analysis

– Experimental data:
The number of correct answers (ranging from 0–12) in the face recognition task was subjected to further analysis. Facial reactivity during the emotion inducing interview was rated for intensity on a 7-point LIKERT-scale (0 = inactive, 6 = maximum activity) by a trained rater.

Interrater reliability, computed by means of an ANOVA intraclass correlation coefficient (ICC, Bartko 1966), was ICC = 0.82, retest reliability was ICC = 0.92. Videoanalysis of the imitation and simulation task was performed by the same rater (blind for diagnosis, treatment, and time of assessment) using a rating scale, which consists of a global judgement (based on the Facial Action Coding System – FACS, Ekman and Friesen 1978) of the adequacy of correspondence between the subjects' and either the poser's (imitation) or a prototypical emotional facial expression (simulation). Corresponding to the recognition task, for both tasks the number of "correct" reactions (ranging from 0–12 and 0–6, respectively) went into the analysis. Interrater reliability for this categorial rating was Kappa = 0.65, retest reliability Kappa = 0.89.

During the recognition, imitation, and simulation tasks some patients (but no normal controls) gave sometimes no answer or did not engage in facial activity. In particular, this phenomenon occurred in S at T0 during the recognition and imitation task, and in D at T1 in the imitation and simulation task. This behavior was rated as "non-reaction". It may reflect task related problems in input and/or output processes, whereas a general motivational deficit seems unlikely because of its inconstant appearance across tasks. For data analysis, both "non-reaction" and "false" reactions were assigned as "incorrect" in contrast to "correct" reactions. To rule out the possibility, that patients' poorer performance was merely due to the phenomenon of non-reaction, additional analyses were run excluding non-reacting patients. However, none of these analyses altered the original findings substantially. Therefore, the reported results are based on the complete patient sample.

– Clinical data:
The items "unchanging facial expression" and "inappropriate affect" of the SANS-subscale "affective flattening" (SANS-A), were used as indicators of altered spontaneous facial expression during the clinical interview in schizophrenics and depressives. As a more comprehensive parameter of affective flattening a sumscore of the SANS-A-items "poor eye contact", "decreased spontaneous movements", "paucity of expressive gestures", "unchanging facial expression", "affective nonresponsivity", and "lack of vocal inflections" was computed. The SANS-A-items "inappropriate affect" and "subjective rating of affective flattening" were not included because of their low item-subscale-intercorrelation reported by Andreasen (1982). The subjective rating of affective flattening was used as a measure of the patient's awareness of his/her expressive deficit.

BPRS-subscores "activation", "hostility", and "thought disturbance" were summed (BPRS-S) to monitor the schizophrenics' course of positive symptoms.

– Statistics:
All statistics were done by 3 × 2 MANOVA (group × time), if not indicated otherwise. Tukey's Honestly Significant Difference test (TUKEY-HSD) was used for post hoc comparisons. In view of the exploratory character of the study, all computed error probabilities will only be used in a descriptive manner, i.e. as an estimation of the magnitude of mean differences, without α-adjustments. Error probabilities will be reported whenever $\alpha \leq 0.10$.

Results

Clinical course

After 4 weeks of inpatient drug treatment, S and D were significantly improved in their BPRS-S (T0: $x = 25.6$ $s = 8.7$; T1: $x = 18.4$ $s = 5.1$; paired t test $t = 3.8$ $p < 0.001$) or HAM-D score respectively (T0: $x = 22.6$ $s = 9.2$; T1: $x = 10.1$ $s = 7.3$; paired t test $t = 7.3$, $p < 0.001$). Concerning the SANS-A sumscore S and D did not differ significantly at T0 (S: $x = 14.6$ $s = 7.2$; D: $x = 14.9$ $s = 6.5$). At T1, however, only D were significantly improved (S: $x = 13.9$ $s = 7.9$; D: $x = 9.6$ $s = 7.2$; 2 × 2 – MANOVA diagnosis × time: $F = 5.1$ $p < 0.05$). The complete SANS-A item profile, from which the SANS-A sumscore was derived is given in Fig. 1.

Subjective rating of affective flattening

Despite their similarity in the clinical observer rating of affective flattening at T0, in their subjective rating S complained less about affective nonresponsivity than D (see Fig. 1). In accordance with their clinical improvement in the SANS-A sumscore, however, D's complaints were significantly decreased at T1 (2 × 2 – MANOVA diagnosis × time: $F = 8.9$ $p < 0.01$), whereas those of S remained stable. Hence, although S and D differed in the subjective evaluation of their affective flattening at T0, both seemed to evaluate its time course correctly.

Spontaneous facial activity

Clinical interview:
According to Fig. 1, the facial expression of S and D was rated as similarly "unchanging" during the clinical interview at T0. With ongoing remission facial activity became slightly more expressive in D (2 × 2 – MANOVA diagnosis × time: $F = 3.0$ $p < 0.10$), whereas S exhibited a stable deficit in facial expressivity. "Inappropriate"

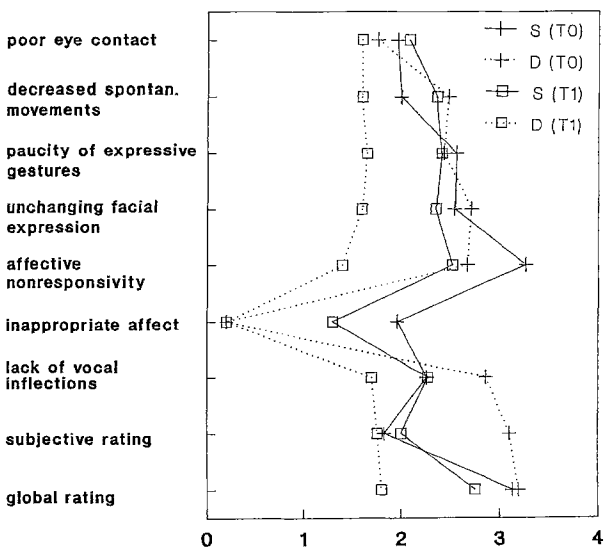

Fig. 1. Profile of SANS-subscale "affective flattening" in schizophrenics (S) and depressives (D) at T0 and T1, 0 = no decrease, 4 = marked decrease

affect however, which refers to situationally improper, though not necessarily restricted facial expression, was a nosologically specific (and relatively stable) characteristic of S (2×2 – MANOVA diagnosis: $F = 17.0$ $p < 0.001$).

Experimental interview:
Table 1 summarizes the main results of the experimental tasks. For the facial activity rating during the emotion eliciting interview a significant group effect was due to reduced facial activity in S and D compared to N both at T0 and T1 (Fig. 2a). No change over time or interaction group × time occurred.

Emotional face recognition

As can be seen in Fig. 2b, only S showed a significant and timestable recognition deficit. All three groups im-

Table 1. Results from 3 × 2-MANOVA (group × time) and post hoc comparisons (Turkey's HSD) for the experimental tasks

	Main effect group		Main effect time		Interaction group × time	
	F	*p*	*F*	*p*	*F*	*p*
Exp. Interview (expressivity rating)	17.1 S, D ≪ N	<0.001	1.5	>0.10	1.6	>0.10
Recognition (no. correct answers)	8.1 S < N	0.001	8.0 T0 < T1	0.006	2.0	>0.10
Imitation (no. correct reactions)	10.5 S ≪ N, D < N	<0.001	3.2 T0 < T1	0.08	6.4 T0: S ≪ N T1: S, D ≪ N	0.003
Simulation (no. correct reactions)	11.0 S ≪ N, D < N	<0.001	0.1	>0.10	2.7 T0: S ≪ N T1: S, D ≪ N	0.08

(<) and (>): $p < 0.10$; < and >: $p < 0.05$; ≪ and ≫: $p < 0.01$

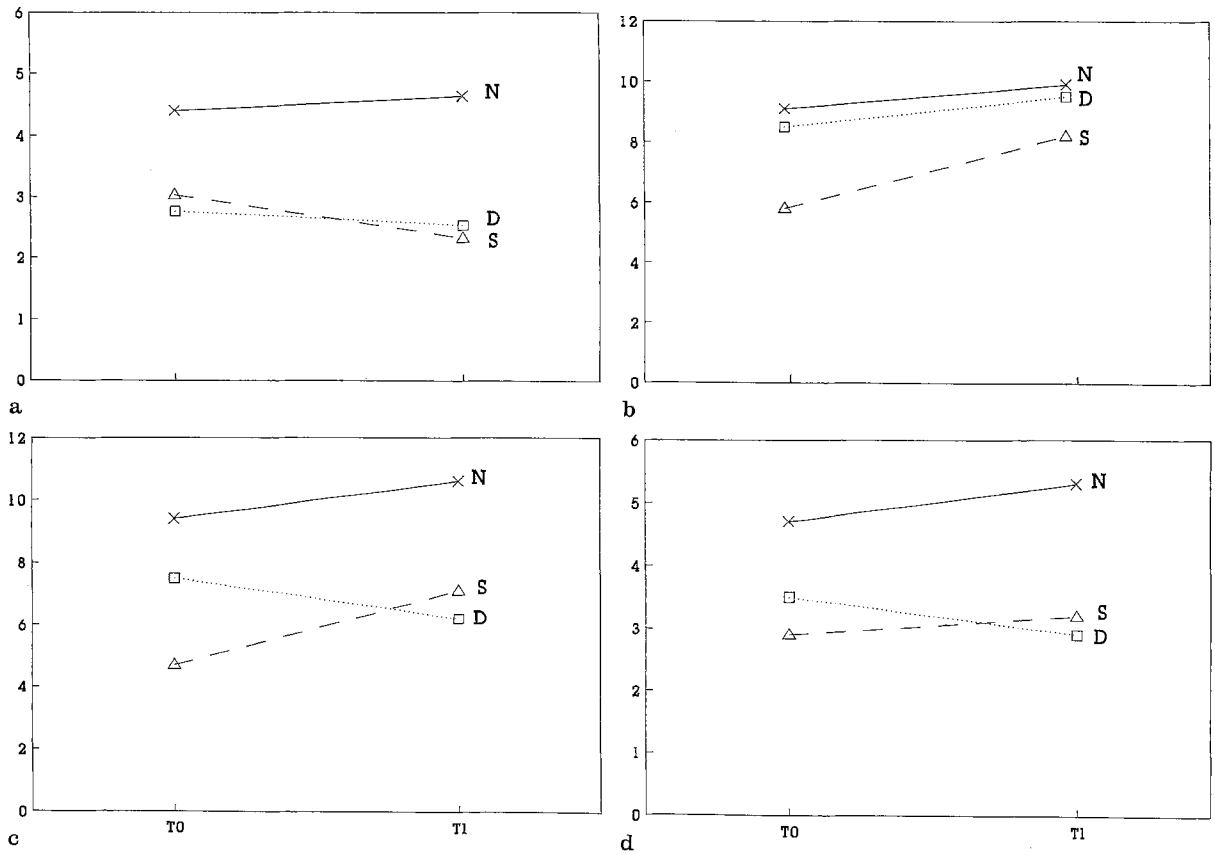

Fig. 2. a Spontaneous facial activity (experimental interview). Facial activity during the experimental interview in schizophrenics (S), depressives (D), and normals (N) at T0 and T1 (see also Table 1) 0 = inactive, 6 = maximum activity. **b** Recognition performance. Number of correct answers in the recognition task in schizophrenics (S), depressives (D), and normals (N) at T0 and T1 (see also Table 1) 12 = maximum number of answers. **c** Imitation performance. Number of correct actions in the imitation task in schizophrenics (S), depressives (D), and normals (N) at T0 and T1 (see also Table 1) 12 = maximum number of actions. **d** Simulation performance. Number of correct actions in the simulation task in schizophrenics (S), depressives (D), and normals (N) at T0 and T1 (see also Table 1) 6 = maximum number of actions

proved in their performance over time. There was no interaction group × time.

Voluntary facial activity

Imitation:
On the average of the two assessments (T0 and T1) S and D performed significantly less often correctly than N (Table 1, Fig. 2c). However, whereas S and N improved in their performance from T0 and T1, D's performance slightly decreased, resulting in a significant group × time interaction.

Simulation:
Similarly to the imitation performance, both groups of patients showed – on the average of T0 and T1 – less often a correct simulation of the required facial expression compared to normals (Fig. 2d). As for imitation, whereas D's performance slightly decreased between T0 and T1, S and N tended towards a better performance at T1, resulting in a slightly significant group × time interaction.

Intervening variables

Taskspecific (basic) emotions:
Considering the possible influence of the respective emotions in the recognition, imitation and simulation task by means of separate 3×6 – MANOVA (group × emotion) for T0 and T1, for correct emotional face recognition an additional interaction effect with the group membership emerged at T1 (group × emotion $F = 3.32$, $p < 0.01$, Greenhouse-Geisser epsilon-corrected because of nonsphericity). This effect was particularly due to a poorer performance of S compared with D in the correct recognition of fearful faces ($p < 0.05$). All three groups' stable recognition deficit of sad faces was obviously due to an insufficient facial representation of sadness.

For imitation, an additional interaction ($F = 2.07$, $p < 0.05$) occurred at T0, originating from poorer performance of S compared to N in fearful faces ($p < 0.05$). for simulation finally, an additional interaction at T0 ($F = 2.86$, $p < 0.05$, epsilon-corrected) was based on a slightly better performance of depressives in sad and angry facial expression and poorer performance in other emotions compared with normals, but none of the single comparisons of means proved to be significant.

Psychopathology:
As parameters for the relationship between task performance and psychopathological characteristics (SANS-subscale sumscores, SANS-A-item "unchanging facial expression", BPRS-S, HAM-D) Pearson correlations were calculated. In S facial reactivity in the experimental interview was inversely correlated with the SANS-A sumscore at T0 ($r = -0.55$, $p < 0.05$) and T1 ($r = -0.66$, $p < 0.01$). Recognition performance was inversely correlated with SANS-alogia sumscore (T0: $r = -0.58$, T1: $r = -0.51$, both $p < 0.05$). Imitation and simulation performance were not significantly related to psychopathology. In D there were no significant correlations with psychopathology at all.

Medication:
Comparison of the 14 S who were unmedicated for at least 3 months, and the 9 S who had drug washout periods ranging from 3–60 days before admission, did not yield any difference in task performance either at T0 or T1. During the 4-weeks study period, 13 S were treated with perazine, 10 with haloperidol. The mean daily/cumulative dosages were 376/10160 mg CPZ and 445/16400 mg CPZ respectively. For the total group of S there was no significant relationship between daily/cumulative CPZ-dosage and any of the experimental measures. Concerning drug type, there was no significant treatment interaction with either the BPRS-S or SANS-A sumscore. "Unchanging facial expression" in the clinical interview, however, improved in the perazine group, but became even worse in the haloperidol group (drug × time $F = 4.3$, $p < 0.05$). Although the EPS-sumscore significantly increased in the haloperidol group (drug × time $F = 4.8$, $p < 0.05$), correlation coefficients between the EPS-sumscore at T1 and facial activity both under spontaneous and voluntary conditions were all insignificant, ranging from $r = -0.22$ to $r = 0.27$. Neither spontaneous nor voluntary facial expression (imitation and simulation) under experimental conditions, nor emotional face recognition were affected by drug type.

Discussion

According to these findings schizophrenics and depressives seem to be different from each other as well as from normals in their affect-related behaviors. Obviously, the behavioral pattern in major depression is in accordance with its conceptualization as an "affective" disorder. Involuntary facial expression is acutely reduced, but improves significantly with clinical remission. However, in an emotion-eliciting interview facial activity remains reduced, contrary to the subjective evaluation of improved affective responsivity.

The discrepancy between the two ratings of spontaneous facial activity may reflect the different situational demands of the clinical and experimental interview: the more emotion-stimulating character of the experimental interview could have led the depressives to withdraw. On the other hand, it cannot be ruled out that the clinical rating has been biased towards improvement because of non-blind rating conditions, whereas the video-based rating during the experimental interview was conducted blindly regarding time of assessment (and diagnosis).

Voluntary facial expression in depressives is relatively intact in the acute depressed state, whereas with ongoing remission correct performance in imitation and simulation seems to decrease. Facial affect recognition is at no time impaired. The latter finding is in contrast to the results of Walker (1981), and Walker et al. (1984), who reported deficits in facial emotion judgement both for depressed and schizophrenic patients. Similarly, Feinberg et al. (1986) reported an emotion-labeling deficit also in major depression, although less pronounced compared with schizophrenia. However, our results confirm the findings of Gessler et al. (1989), which showed no difference between depressed and normal subjects in emotional face recognition.

On the contrary, schizophrenics under acute as well as partly remitted conditions are characterized by a stable reduction of involuntary facial activity under clinical and experimental conditions, which is not adequately reflected in their subjective evaluation of improving affective responsivity. At the same time their voluntary facial activity remains disturbed as indicated by their poor imitation and simulation performance. In accordance with our previous findings (Gaebel et al. 1989), facial affect recognition seems to be persistently impaired without consistent emotional specificity. However, this conclusion is based on only four weeks between the two assessments under acute and partly remitted conditions respectively. According to the cross-sectional study by Gessler et al. (1989), who reported a recognition deficit in a group of acute but not in a group of remitted schizophrenics, no difference might have been expected in our sample after full remission. A corresponding trend, though not significant, is indeed indicated in our data (see Fig. 2b). However, a final decision whether schizophrenics' recognition deficit is either state- or trait-specific has to be postponed until longitudinal studies are available, which assess schizophrenics' recognition performance over a longer time period than we did.

A further question to be addressed is the specificity for emotional stimuli of schizophrenics' recognition deficit, i.e. a differential deficit, since the reported differences between schizophrenics and normals could merely reflect a global cognitive deficit. As part of our larger study on emotional dysfunctions in schizophrenia, additional nonemotional control tasks were run. Preliminary results from RAVEN progressive matrices revealed a performance deficit only in acute schizophrenics, pointing to a global cognitive impairment. Under partly remitted conditions only their emotional recognition deficit seems to persist, pointing to a differential deficit. The findings of Feinberg et al. (1986) are in favor of a global cognitive impairment in schizophrenics. However, since they assessed their subjects only once by the second or third week of hospitalization, these patients may have still been in an acute state and their differential improvement in cognitive functions may have been overlooked.

Taking into account that all the experimental measures have been obtained under the standardized, artificial communicative situation of a videolink, it seems remarkable that almost all subjects actively engaged in the tasks. In fact, schizophrenics commented sometimes that they felt more relaxed under these laboratory conditions than in the live interview. On the other hand some patients did not respond to task requirements at all. Since this phenomenon was – as a rule – task-specific, a general motivational deficit seems to be implausible to account for this "non-reaction". Besides task related problems in input and/or output processes, already mentioned above, in some cases it may reflect a coping strategy in the sense of non-responding to arousing emotional stimuli. This interesting phenomenon certainly would have deserved closer inspection. For practical reasons, however, non-reaction was defined as an incorrect reaction. Although this might have underestimated patients' performance, none of the reported deficits was merely due to non-reaction.

Concerning medication effects, although spontaneous facial activity is unfavorably affected by haloperidol, neither a medication nor a drug-specific influence on experimental task performance was detectable. Similarly, no correlation between extrapyramidal side effects and experimental task performance could be found for schizophrenics at T1.

In conclusion, schizophrenics and depressives are characterized by different quantitative, qualitative, and temporal patterns of affect-related dysfunctions. Both have in common a discrepancy between subjective and objective evaluation of their expressive deficit, however, at different times of their illness course (Brown et al. 1979). In schizophrenics there seems to be a relatively stable deficit in affect recognition, as well as in spontaneous and voluntary facial activity. Hence, in schizophrenics both the processes of decoding and encoding of facial expression seem to be disturbed. In terms of a perceptual motor theory of emotion (e.g. Leventhal 1980), schizophrenics' disturbance might be interpreted as a stable *hypo*function of an (involuntary) expressive motor mechanism (reduced spontaneous facial activity), a stable *dys*function of a schematic (recognition deficit) and a (voluntary) conceptual mechanism respectively (imitation and simulation deficit). The latter deficit would be in accordance with the concept of a general dysfunction of higher-level motor control in schizophrenics (Frith 1987). In depressives, on the contrary, a similarly stable reduction of spontaneous facial activity (at least in an emotion-inducing interview) occurred, but no recognition deficit. Unexpectedly depressives' voluntary facial activity became dysfunctional during the 4-weeks observation period. Hence, in depressives particulary the processes of encoding of facial expression seem to be disturbed. Accordingly, depressives could be characterized by an acute *hypo*function of the expressive motor mechanism, which either normalizes or remains stable depending on the content and/or setting of the interview.

Patterning and timing of these deficits are probably related to either reversible or irreversible dysfunctions in underlying neural networks.

Circuits involved in emotional recognition and expression seem to be asymmetrically distributed in the brain (Campbell 1982, Silberman and Weingartner 1986). However, from studies in brain-lesioned patients, lesion side did not affect spontaneous or voluntary facial movements to the same extent as did lesion site: Reduced facial expression and impaired voluntary movements mainly occurred after frontal lesions irrespective of side, or after commissurotomy (Kolb and Milner 1981a, b, Milner and Kolb 1985). From the analysis of spontaneous facial expressions by means of FACS, however, even the influence of lesion site has been challenged (Mammucari et al. 1988). On the other hand, lesion side, irrespective of site, seems to be involved in matching of emotional faces (right hemisphere) or matching of verbal descriptions to appropriate verbal categories of emotional states (left hemisphere) (Kolb and Taylor 1981).

However, it cannot be expected that broadly defined psychopathological symptoms or even syndromes, e.g. affective flattening, are directly related to dysfunctions in circumscribed neural networks (Holzman 1988). Therefore, an experimental approach to psychopathology like the one used in the present study, which seems to allow a differentiation of emotional dysfunctions in schizophrenia and depression by referring to the in-

volved subprocesses, is an important step in mapping clinical symptomatology onto dysfunctional neurobiological systems.

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