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ORIGINAL PAPER

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Facial expressivity in the course of schizophrenia and depression

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Abstract This study investigates the nosological specificity and time stability of reduced facial expressivity in schizophrenia by means of objective measurement. Facial expression in an emotional interview was evaluated using the "Facial Action Coding System" in 33 acute schizophrenia patients and 23 acute depressive patients in comparison with 21 nonpatient controls, each assessed twice within 4 weeks, and in 36 partly remitted schizophrenia patients assessed twice within 3 months.

Acute schizophrenia patients showed reduced facial activity especially in the upper face and in facial activity often used as communicative signs or as signs of positive emotions. As depressive patients showed a comparable pattern of facial activity, nosological specificity is questionable. This pattern remained stable in the acute illness course and was almost identical in remitted schizophrenia patients, indicating a marked time stability of attenuated facial expressivity in schizophrenia and – for the acute phase assessed – in depression.

Key words schizophrenia · depression · facial activity · nosological specificity · stability

Introduction

According to the common definition of affect as "a pattern of observable behaviors that is the expression of a subjectively experienced feeling state (emotion)" (DSM-IV, American Psychiatric Association 1994), reduced expressive behavior is considered as the main indicator of affective flattening (Andreasen 1982), a core characteristic of negative symptoms in schizophrenia (Crow 1985). In particular, reduced facial expression plays a crucial role for the definition and the assessment of affective flattening. However, it is still under debate, whether the reduced facial expressivity in schizophrenia patients is due to an emotional deficit, an intentional/motor deficit, or both (Dworkin et al. 1996; Kring et al. 1993). Moreover, since expressive behavior in retarded depression clinically looks very much alike, the nosological specificity of this phenomenon has to be doubted.

This scarceness of detailed knowledge is at least partly a methodological problem. Usually affective flattening is measured within a clinical interview by means of observer-based rating scales for the assessment of negative symptoms, which at best provide a special subscale for affective flattening (e. g. Scale for the Assessment of Negative Symptoms, SANS; Andreasen 1982; Andreasen 1989) with facial activity assessed by a single item. For a more thorough investigation of, e. g., the nosological specificity or of neurobiological determinants of affective flattening, in particular facial activity, this approach is certainly not finegrained enough. Alternative approaches for the evaluation of facial activity are based on the objective measurement of facial movements. The "Facial Action Coding System" (FACS; Ekman and Friesen 1978) allows the registration of the overt activity of single facial muscles coded in terms of 44 single "action units" (AUs) by trained and certified observers. These 44 AUs are defined on an anatomical and muscular basis and represent the basic repertoire of facial expression. Thus intensity as well as variety of facial movements can be objectively evaluated, without the interference of interpretation. Other methods for the objective assessment of facial activity are computer-based automatic analysis of facial movements (Bartlett et al. 1999; Cohn et al. 1999; Schneider et al. 1990) as well as direct measurement of the facial muscle activity by means of electromyography (EMG). The present study

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uses the FACS in order to investigate facial expression in schizophrenia patients in a more detailed way than conventional observer based ratings allow for.

Previous studies on *spontaneous facial expressivity* in schizophrenia using FACS or its descendant EMFACS ("Emotional Facial Action Coding System"; Friesen and Ekman 1984) mostly confirmed the attenuated repertoire of facial reactions in schizophrenia patients and their reduced facial activity. In addition, these studies could often confine the latter disturbance to the upper face using a variety of situational stimuli (Ellgring and Gaebel 1994; Krause et al. 1989; Steimer-Krause et al. 1990; Steimer et al. 1988). However, by means of computer-based analysis of the overt movements of single facial muscles this effect could only be found during an experimental interview in acute inpatients, but not while watching emotion eliciting films (Schneider et al. 1990). Moreover, studies using EMG recordings of single facial muscles to investigate also more subtle unobservable activity could not find differences in the upper face activity (Mattes et al. 1995) or even reported stronger corrugator activity in schizophrenia patients (Kring et al. 1999).

As to be expected, reduced (observable) facial expressivity in schizophrenia patients was found to be most pronounced in patients or even restricted to patients who clinically were classified as blunted (Berenbaum and Oltmanns 1992) or as deficit patients (Earnst and Kring 1999). At the same time, Berenbaum and Oltmanns (1992), Iwase et al. (1999) as well as Kring et al. in a series of studies (Earnst and Kring 1999; Kring et al. 1993; Kring and Neale 1996) found a discrepancy between reduced facial activity and maintained emotional experience in schizophrenia patients, suggesting that symptoms of affective flattening represent a disturbance of emotional expression rather than one of emotional experience.

In addition to disturbances in spontaneous facial expressivity, schizophrenia patients also seem to have difficulties in *voluntary facial expressivity* (Borod 1989; Gaebel and Wölwer 1992; Whittaker et al. 1994), but see (Berenbaum 1992), i. e. they show poorer performance in the task to imitate shown facial expressions or to simulate facial expressions of positive and negative emotions.

Studies concerned with the *nosological specificity* of schizophrenia patients reduced expressivity often found similar disturbances in depressive patients (Ellgring and Gaebel 1994; Schneider et al. 1990). Though Jones and Pansa (1979) reported nosologically specific differences of non-verbal behavior between schizophrenia patients and depressive patients, these effects were limited to specific situations and to only some of the multitude of variables assessed. On the other hand, there seem to exist at least qualitative differences in facial expressivity between schizophrenia patients and psychosomatic (Steimer-Krause et al. 1990) or parkinsonian patients (Alpert and Rush 1983).

There are only few studies examining deviations of facial activity in schizophrenia patients in the *course of time*. While Jones and Pansa (1979) reported differences between patients with depression and those with schizophrenia at the time of the initial examination, 2–4 weeks afterwards at the time of discharge they did not find differences anymore. Likewise, Mattes et al. (1995) suggested in their cross-sectional study that upper face hypomimia might be a reversible feature limited to the acute phase of schizophrenia, since they could not find attenuation of upper face activity in remitted schizophrenics as opposed to the findings of a predecessor study in acute schizophrenia patients (Schneider et al. 1990). Lower face hypomimia on the other hand seemed to be comparable for both acute and remitted schizophrenia patients.

In contrast to that, the longitudinal study by Ellgring and Gaebel (1994) revealed stable deficits of facial activity during the first month of an acute schizophrenic episode, and also a attenuation of facial activity in depressed patients still present at the time of discharge. Time stability of facial activity deficits in schizophrenia is also suggested by findings of Walker et al. (1993) who examined childhood and youth video clips of schizophrenia patients. Already prior to manifestation especially female schizophrenia patients showed reduced frequency of facial patterns expressing "joy" and more often facial expressions of emotions of negative valence in comparison to their healthy siblings.

Whether deficits in facial expressivity are responsive to *neuroleptic medication* is not clear yet. The study by Schneider et al. (1992) suggests a further reduction of facial activity in schizophrenia patients as a consequence of neuroleptic medication, whereas other studies could not find any relationship between neuroleptic medication and facial expression in schizophrenia patients (Berenbaum and Oltmanns 1992; Earnst et al. 1996).

In summary, there are still open questions concerning all the above addressed aspects of the disturbances of facial expression in schizophrenia. Therefore, the aim of the present study was to assess disturbances in facial expressivity in the course of schizophrenia by means of objective methods with regard to (1) the exact kind of the disturbances in spontaneous facial expressivity as well as (2) its nosological specificity and (3) its time stability. Secondary questions concerned medication effects on facial expressivity and the relationship between different methodological approaches to assess facial expressivity (observer based clinical rating vs. FACS-based assessment).

Methods

Subjects

Subjects included in the study were 33 acute schizophrenia patients (S_a; 21 male, 11 female; mean age ± standard deviation: 31.6 ± 10.5 years), 36 partly remitted schizophrenia patients from a psychiatric day-hospital (S_r; 19 male, 17 female; mean age: 35.9 ± 8.8 years) and 23 patients with acute major depressive disorder (D; 12 male, 11 female;

mean age: 40.5 ± 12.6 years). 21 nonpatient volunteers (N; 15 male, 6 female; mean age: 33.9 ± 10.4 years) were included as controls.

All subjects were assessed at least twice: in S_a and D assessments took place within three days after admission (T0) and after four weeks of neuroleptic or anti-depressive treatment (T1), respectively. Non-psychiatric controls were assessed twice within four weeks as well. According to the assumption that any changes of performance in remitted patients – if at all – would occur only after a longer time period than four weeks, S_r were assessed at least eight weeks after discharge from an acute ward (T2) and three months later (T3).

All patients were diagnosed according to Research Diagnostic Criteria (Spitzer et al. 1978) by a trained rater. Subjects with organic brain damage, drug abuse and subnormal intelligence were excluded from the study. In addition, S_r relapsing during the observation period were also excluded. Fourteen S_a, three S_r, and seven D were first episode patients. The remaining patients had mean illness durations of 6.1 years (S_a), 7.9 years (D), and 8.9 years (S_r), respectively.

At T0, 18 S_a were drug free and had never or not for a long time (> 3months) been treated with anti-psychotics; the remaining 15 S_a where pre-medicated with anti-psychotics during the three months prior to hospitalization. After T0 S_a were allocated to oral treatment with either perazine (n = 21) or haloperidol (n = 12). The mean daily dosage in chlorpromazine equivalents (CPZE) in the T0-T1-interval did not differ significantly (perazine: 438 mg CPZE/day, haloperidol: 524 mg CPZE/day). Among S_r ten patients were treated with clozapine (412 mg CPZE/day), 21 were receiving typical neuroleptic drugs either orally or as depot medication (466 mg CPZE/day), and five patients were drug-free in the T2-T3-interval. Five S_a but none of the S_r received anticholinergic medication. Depressive patients were treated with various antidepressives, depressive patients on neuroleptics were excluded.

Procedure

After informed consent a clinical interview was accomplished in psychiatric patients by a single trained rater. The following rating scales were used:

- Brief Psychiatric Rating Scale (BPRS, (Overall and Gorham 1962)
- Scale for the Assessment of Negative Symptoms (SANS, Andreasen 1989)
- Hamilton Depression Ratingscale (HAM-D, Hamilton 1967)
- Extrapyramidal Side Effects Ratingscale (EPS, Simpson et al. 1970)

Following the clinical assessment a semistructured experimental interview took place in a special laboratory for basic behavioral research. The same interview was used at both times of assessment. This interview lasted about 5 min and included 3 standardized questions (supplemented by individual inquiries), focusing on the present state and present or past good and bad experiences to recall corresponding positive and negative emotions. The 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. Subjects were comfortably seated in front of a videomonitor, on which the interviewer was visible. During the interview the subjects' facial as well as hand/body movements were recorded separately by two video cameras. Both visual information together with the task displayed were stored simultaneously on videotape for subsequent analysis using a split videoscreen. Data on gestures and speech activity have been published elsewhere (Gaebel et al. 1994).

Data analysis

Data reduction for experimental data

Facial expressivity during the emotion inducing interview was analyzed using the Facial Action Coding System (FACS, Ekman and Friesen 1978). The analysis was done by two trained and certified FACS-raters blind for diagnosis, treatment and time of assessment. Their mean index of agreement according to the algorithm designated by the FACS investigators manual was 0.81, which is far beyond the necessary criterion for reliable data. In order to reduce the complex raw data, two summarizing variables were defined: "Intensity of facial expression" was defined as the number of identified action units (AU) per minute, whereas the "repertoire of facial expression" refers to the number of different AUs shown at least twice during the interview. For more detailed analyses, the ten AUs and combination of AUs (which in fact occured) most often shown by the nonpatient control group were analyzed with respect to their rate per minute. Each AU was only counted once in this analysis, i. e. AUs already counted in a combination of AUs were added to the frequencies of the single AUs.

Data reduction for clinical data

The item "unchanging facial expression" of the SANS subscale "affective flattening" (SANS-AF), was used as indicator of altered spontaneous facial expression during the clinical interview in schizophrenics and depressives. As a more comprehensive parameter of affective flattening the SANS-AF items "poor eye contact", "decreased spontaneous movements", "paucity of expressive gestures", "unchanging facial expression", "affective nonresponsivity", and "lack of vocal inflections" were summed (SANS-AFS). The SANS-AF items "inappropriate affect" and "subjective rating of affective flattening" were not included because of their low item-subscale intercorrelation reported by Andreasen (Andreasen 1982). The subjective rating of affective flattening was used as a measure of the patient's awareness of his/her expressive deficit. BPRS subscales "activation", "hostility", and "thought disturbance" were summed to monitor schizophrenia patients' positive symptoms (BPRS-PS). HAM-D and EPS sumscores were used to assess depressive and extrapramidal symptoms, respectively.

Statistical analyses

The data analysis was performed in two steps, respectively: at first S_a was compared with D and N regarding facial activity and facial repertoire in the interval T0-T1 by means of univariate 3x2 repeated measures ANOVA (i. e. 3x2- MANOVA group x time). Because of the different length of the observation interval, data for S_r were analyzed separately. However, in order to relate data for S_r in the interval T2-T3 at least approximately to a standard level, data for N in the interval T0-T1 were included into univariate 2x2 MANOVA (group x time) accepting limited comparability of the observation interval, which has to be taken into account in the interpretation of results. The more detailed analysis of the most frequent AUs was performed in a second step using two multivariate repeated measures MANOVAs (group x time x AUs, i. e. 3x2x10-MANOVA for S_a, D, N and 2x2x10-MANOVA for S_r, N).

In case of significant group effects Tukey's Honestly Significant Difference test was used for post hoc comparisons. According to 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

Experimental data

Compared to N, both S_a and D showed a stable attenuation of their facial activity and facial repertoire during the four week interval between T0 and T1 (main effects group: facial activity $F = 14.2$ $p < 0.001$, Tukey-Tests: $S_a < N$ $p < 0.001$, $D < N$ $p < 0.001$; facial repertoire $F = 9.6$ $p < 0.001$, Tukey-Tests: $S_a < N$ $p = 0.001$, $D < N$ $p = 0.001$; see Fig. 1a, b). The facial expressivity of all three groups decreased in the interval T0-T1 (main effect time: facial activity $F = 6.7$ $p = 0.012$; facial repertoire $F = 9.6$ $p = 0.003$), but differences between groups remained sta-

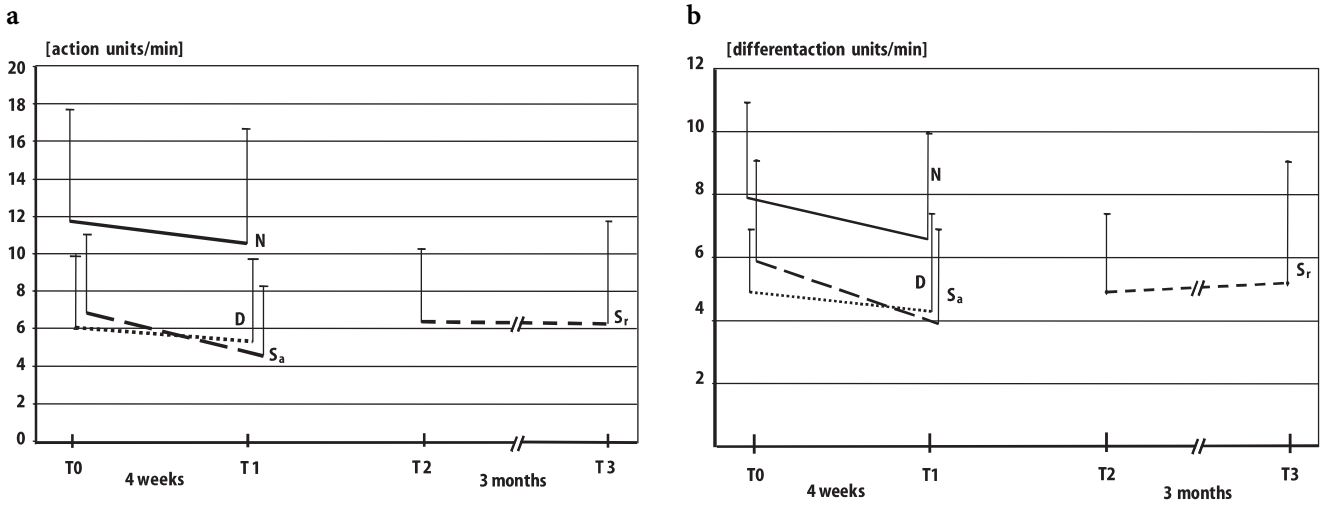

Fig. 1 **a** Facial activity during an experimental interview in acute patients with schizophrenia (S_a) or depression (D) at admission to the hospital (T0) and four weeks later (T1) as well as in nonpsychiatric controls (N) assessed twice four weeks apart and in remitted schizophrenia patients (S_r) assessed twice three months apart. **b** Facial repertoire during an experimental interview in acute patients with schizophrenia (S_a) or depression (D) at admission to the hospital (T0) and four weeks later (T1) as well as in nonpsychiatric controls (N) assessed twice four weeks apart and in remitted schizophrenia patients (S_r) assessed twice three months apart (T2/T3)

ble, as indicated by insignificant group by time interactions.

Comparably, S_r also showed a stable attenuation of their facial expressivity at T2 and T3 in comparison to N at T0 and T1 (main effects group: facial activity $F = 14.6$ $p < 0.001$; group: facial repertoire $F = 9.6$ $p = 0.003$) without time or interaction effects.

The differential analyses of facial activity on the basis of the 10 most often occurring AUs and combination of AUs confirmed the significant group effects (S_a, D vs. N: multivariate $F = 3.11$ $p < 0.001$; S_r vs N multivariate $F = 3.30$ $p < 0.001$) and showed that the flattened facial expressivity in S_a, S_r and D originated exclusively from an attenuation of AUs and combinations of AUs representing facial activity of the upper face (AU1 + AU2: inner and outer brow raise, AU4: brow lowering) and/or AUs, which are frequently shown in relation with positive emotions (AU6 + AU12: cheek raise and lip corner pull) or with communicative functions (AU12; see Fig. 2a, b; every univariate post hoc comparison for these 4 AUs or AU combinations $p < 0.05$ with S_a, S_r, D < N). On the contrary, group differences regarding AUs of the lower face, usually occuring in the context of the expression of negative emotions (AU 14: dimpler, AU17: chin raise, AU20: lip stretch, AU24: lip press, AU25: lips part), could not be found (none of the univariate post hoc comparison for these 6 AUs or AU-combinations approached significance). Visual inspection of Fig. 2a, b shows that these 6 AUs occured less frequently in the repertoire of N, but were shown with the same or even higher frequency by S_a, S_r and D within their individual AU profile resulting in non-significant group comparisons to N.

A time effect resulted only from the 2x2x10 MANOVA, with scores at the first assessment being slightly significant higher than at the second assessment (multivariate $F = 1.92$ $p = 0.066$). No interaction group x time emerged.

Clinical data

After 4 weeks of inpatient drug treatment, S_a and D were significantly improved in their clinical status (Table 1), i. e. S_a – but not D – were significantly improved regarding positive symptoms (BPRS-PS, 2x2 MANOVA group x time; interaction $F = 10.1$ $p = 0.002$) and D – and S_a to a lesser extent – were significantly improved regarding depressive symptoms (HAM-D, interaction $F = 9.9$ $p = 0.003$). Concerning the SANS-AFS S_a and D did not differ significantly at T0, but D were significantly more improved than S_a at T1 (interaction $F = 6.67$ $p = 0.013$). Looking specifically at the rating of facial activity, data of the SANS-AF item "unchanging facial expression" indicated an improvement between T0 and T1 both for D and S_a (main effect time: $F = 13.7$ $p < 0.001$; no significant interaction). In spite of their clinical improvement, S_a still showed more positive and negative symptoms at T1 than S_r at T2 (BPRS-PS: $F = 27.6$, $p < 0.001$; SANS-summary score: $F = 3.3$, $p = 0.075$). S_r did not show any significant changes in their clinical data during the interval T2-T3.

Despite their similarity in the observer based clinical rating of affective flattening at T0, S_a subjectively complained less about affective flattening than D (SANS-AF item "subjective rating of affective flattening, 2x2 MANOVA group x time; main effect group $F = 7.3$ $p = 0.009$). In accordance with their clinical improvement in the SANS-AFS; however, D's complaints significantly decreased from T0 to T1, whereas those of S_a remained almost stable (interaction $F = 7.15$ $p = 0.01$) on their low level.

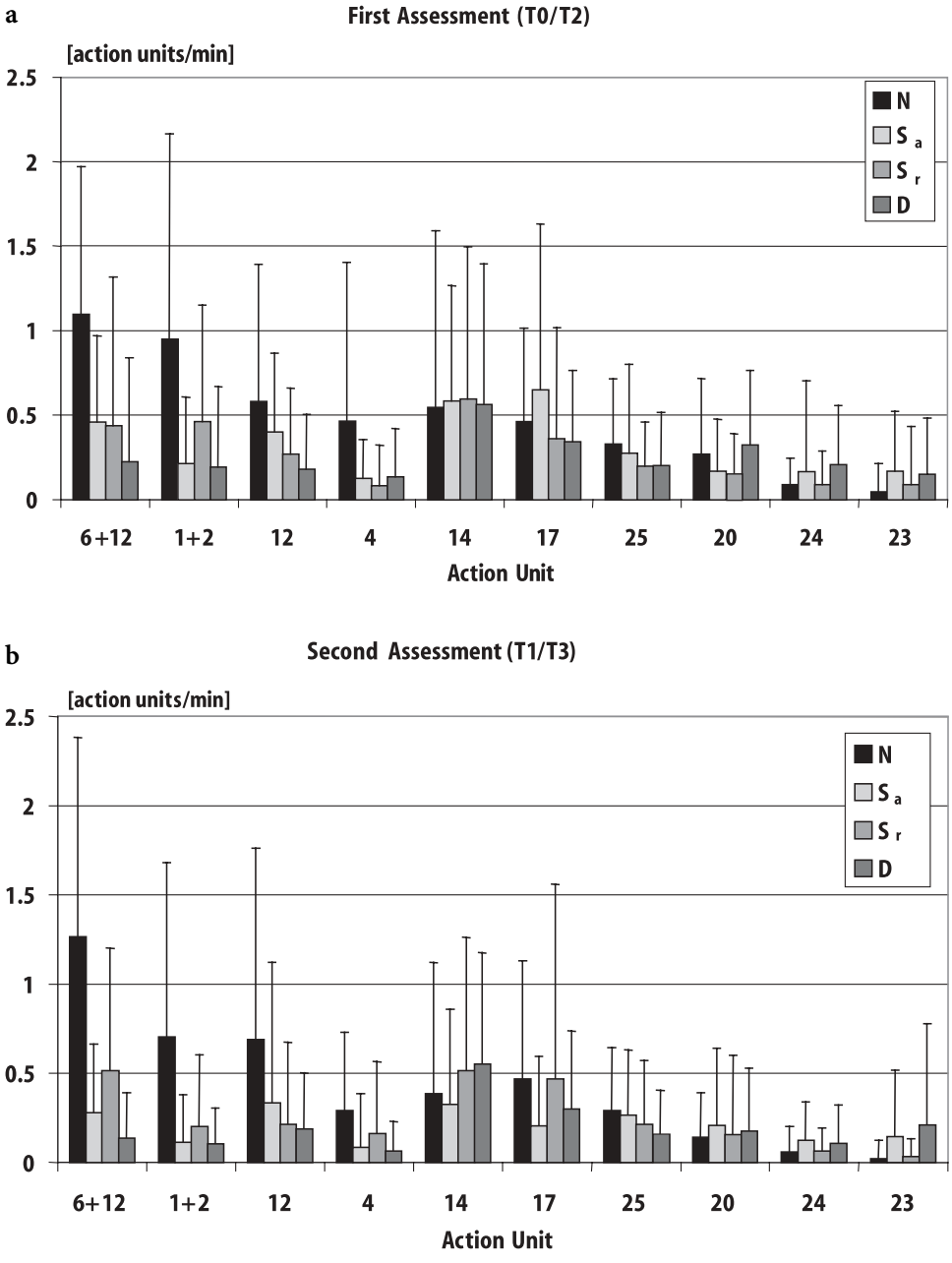

Fig. 2 Frequencies of different facial action units during an experimental interview in acute patients with schizophrenia (S_a) or depression (D) as well as in nonpsychiatric controls (N) and in remitted schizophrenia patients (S_r) at their first time of assessment (**a**) and at their second time of assessment (**b**), respectively (see results text for explanation of action unit numbers)

A correlation analysis at the first time of assessment (T0 in S_a + D and T2 in S_r) revealed no significant relationship between objectively assessed facial activity and the SANS observer ratings of "unchanging facial expression", "global assessment of affective flattening", or SANS-AFS.

Medication effects

Exploratory analyses of differential treatment effects of haloperidol or perazine treatment revealed a significant medication effect regarding the facial repertoire and a trend regarding facial activity in the phase of acute schizophrenia (2x2 MANOVA medication x time: interaction facial activity $F = 3.4$, $p = 0.075$; facial repertoire $F = 6.5$, $p = 0.016$). In the context of the general decrease of facial expressivity in the interval T0-T1 in every group (see above), this attenuation was more pronounced in patients treated with haloperidol than with perazine. However, even under perazine treatment facial expressivity of S_a remained on a level significantly below the level of N.

There were no correlations of the medication with the mean daily dosage in T0-T1, at T1, nor with the incidence of EPS. Moreover, a comparison at T0 of 19 S_a, who had never or not for a long time (> 3 months) been treated with neuroleptic drugs, with 15 S_a, who where medicated with neuroleptics during the three months prior to hospitalization, did not show significant differ-

Table 1 Mean values (M) and standard deviations (SD) of clinical data

Variables	Acute Schizophrenia Patients (n = 34)				Acute Depressive Patients (n = 23)				Remitted Schizophrenia Patients (n = 38)			
	T 0		T 1		T 0		T 1		T 2		T 3	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
BPRS Sumscore "Positive Symptoms"	26.1	8.2	17.5	4.9	13.8	3.7	11.9	2.4	12.2	3.1	13.7	4.4
SANS												
Item "Unchanging Facial Expression"	2.6	1.4	2.2	1.6	2.9	1.5	1.7	1.4	2.8	1.1	2.8	1.1
Item "Subjective Rating of Affective Flattening"	1.8	1.5	1.6	1.6	3.2	1.0	1.8	1.3	2.8	1.0	2.8	1.0
Sumscore "Affective Flattening"	14.3	7.1	13.3	7.9	15.5	6.5	10.0	7.1	14.6	4.8	13.8	5.9
Global rating "Affective Flattening"	3.0	1.1	2.5	1.3	3.3	1.2	1.9	1.2	2.2	1.2	1.9	1.4
Global rating "Alogia"	2.8	1.3	2.0	1.3	1.9	1.4	2.8	1.2	1.3	1.3	1.1	1.3
Global rating "Abulia"	2.2	1.4	2.1	1.3	2.7	1.1	1.4	1.1	2.5	1.2	2.1	1.3
Global rating"Anhedonia"	2.9	1.2	2.6	1.1	3.3	0.9	2.1	1.4	2.6	1.1	2.7	1.3
Global rating "Attention"	2.6	1.2	2.0	1.2	2.1	1.3	1.1	1.2	0.9	1.1	1.2	1.4
HAM-D Sumscore	15.5	6.9	9.4	6.0	23.4	9.0	10.6	7.2	5.7	4.6	5.1	4.5
EPS Sumscore	0.7	1.6	3.2	4.3	0.5	1.7	0.2	0.6	1.0	1.9	1.2	2.3

ences in either of the experimental variables on facial expressivity.

An analysis of medication effects in the T2-T3 interval suggested only trends for a differential development of facial activity, indicating an improvement under clozapine, on the one hand, and a worsening under typical neuroleptic drugs, on the other hand, but this remained statistically as well as clinically insignificant.

Discussion

In accordance with several former findings (e. g. Gaebel et al. 1994; Krause et al. 1989; Schneider et al. 1990) it could be shown by means of FACS-based assessment that schizophrenia patients show reduced facial expressivity, regarding both quantitative as well as qualitative aspects of facial activity. Since these positive studies also used dyadic interactions, as opposed to negative findings obtained in non-communicative situations like film or picture watching (Kring et al. 1999; Mattes et al. 1995; Schneider et al. 1990) these results affirm the importance of a social interactive situation as context for the occurrence of flattened facial expression in schizophrenia. Ellgring (1989) came to a similar conclusion on a more theoretical basis, stressing the functional meaning of facial expressivity in a situation of social interaction. On the other hand, most negative findings were obtained with assessment methods (EMG, computer-based movement analysis) able to measure very subtle muscle activity not necessarily visible for an observer (e. g. Mattes et al. 1995). Thus, the phenomena measured with these methods may only partly overlap with what is defined as facial expression from the viewpoint of an observer or social interaction partner.

Also the qualitative analyses point to the importance of the communicative aspect of the assessment situation and to the communicative function of facial expressivity: as already partly described in former studies showing attenuated facial activity in schizophrenia, this hypomimia is particularly noticable concerning the upper face (Ellgring and Gaebel 1994; Krause et al. 1989; Steimer-Krause et al. 1990; Steimer et al. 1988), concerning facial movements important as communication signs (raising eyebrows: AU 1 + AU 2; courtesy smiling: AU 12), and concerning the expression of emotions with positive valence, i. e. the expression of happiness (AU 6 + AU 12). Raising the eyebrows is known to be the primary facial regulator accompanying speech, in order to emphasize the one's statements, to comment on the statement of the conversation partner or to signal that one wishes to take over the speaker's role (Ekman 1979). Schizophrenia patients' attenuated use of such regulators emphasize their difficulties in social interaction described as "impression of less involvement" in the conversation or "greater distance" by Krause (1989). On the other hand, we found that activity in the lower face usually occuring in the context of the expression of negative emotions is not impaired. This pattern of results is consistent with findings of Walker et al. (1993) who reported reduced frequencies of facial patterns expressing "joy" and increased frequencies of facial expressions of negative emotions in preschizophrenic girls, i. e. already prior to the manifestation of schizophrenia. Later Grimes and Walker (1994) additionally proved that a higher rate of negative emotions during late childhood/adolescence is associated with later age at onset of schizophrenia. They conclude that negative affect in childhood may be both a sign of vulnerability and an indicator of more favorable prognosis.

Alltogether, our results show that facial activity is not generally reduced in schizophrenia, but has to be considered in a differentiated manner dependent on the kind of expression. The unchanged lower face activity may support the interpretation that paucity of facial expression in schizophrenia might be a disturbance of expression – or a disturbance of communication regarding our results mentioned above – rather than an emotional

disturbance put forward by authors finding a dissociation of reduced expressivity together with maintained emotional experience (e. g. Berenbaum and Oltmanns 1992; Iwase et al. 1999; Kring et al. 1993). In the present study schizophrenia patients also rated their affect as only slightly flattened as opposed to their marked attenuation in facial expressivity.

Concerning nosological specificity, patients suffering from schizophrenia or depression could not be differentiated by means of objectively assessed facial expressivity during the observation period. As similarly reported by Ellgring and Gaebel (1994) and Schneider et al. (1990) the two groups of patients did not differ in intensity or qualitity of their facial activity nor in the cross-sectional comparison in the acute state of the illness or in the longitudinal view across time. As already mentioned, the chosen experimental situation of social interaction may contribute to this findings of nosological unspecifity of facial expression. Although affective responsivity may be reduced for different reasons and with different time course (state vs trait) the overt result may be similar in depression and schizophrenia. However, as the results on the subjective experience of affective flattening demonstrate, there is a difference between the two disorders. Hence, as Peralta and Cuesta (1998) have stated, patients' subjective experience may be important in discriminating disorders with similar behavioral manifestation. Given the difference in the neurocircuitry underlying emotional experience and affective expression (Rinn 1984) the neurobiological mechanisms behind behavioral similarities may be very different. However, it cannot be ruled out that even the observed simularities vanish at a closer look at facial muscular activities using higher resolution techniques such as EMG.

In spite of a clinical improvement concerning positive symptoms in schizophrenia patients and depressive symptoms in patients with depression, the impairment in spontaneous facial expressivity remained stable for both groups over the acute period of four weeks (T0-T1). Thus, objectively assessed negative symptomatology showed a different development over time than clinical course as rated by the physician. Moreover, even clinical observer ratings of facial expression (SANS) differed from objectively assessed parameters as indicated by the poor correlation coefficients of both data levels. Only regarding group means in the acute phase at T0 did the objective measures of facial expression and the corresponding observer based rating coincide, but in the course of time the objective measurement proved a stable attenuation of facial expressivity for both schizophrenia and depressive patients, while affective flattening and in particular facial activity were rated significantly improved by the physician after four weeks of treatment at T1. There could be two reasons for this discrepancy. On the one hand the different situational conditions in the clinical versus the experimental interview could have had an effect on the facial activity, on the other hand there could be an observer bias in the clinical interview toward the expectation of improvement, as the rater was not blind regarding time of measurement and diagnosis, which was the case for the FACS coding of the experimental interview.

The marked time stability of schizophrenia patients' attenuated facial expressivity in the upper face shown in the acute phase of the illness is further supported by the results in remitted schizophrenia patients, who showed comparable deficits of facial activity both at the beginning and at the end of the three month observation period. Taken together with the results of Walker et al. (1993), who found deviations in facial expressivity already in preschizophrenic children and adolescents, such deviations may represent a vulnerability marker.

The stability of attenuated facial expressivity in schizophrenia is further stressed by the limited effects of medication. Though these limited effects have to be interpreted cautiously regarding the uncontrolled allocation of patients, the homogeneous pattern of results provides no hint for the interpretation that the attenuated facial expressivity was predominantly due to drug treatment. Medication effects occurred only in the acute phase between T0 and T1 with schizophrenia patients treated with haloperidol showing a greater reduction of facial expressivity than those medicated with perazine. The latter also decreased in facial expressivity but only to a comparable amount as shown on average of the three groups in the interval T0-T1. This general reduction across time most probably indicates a habituation process to the interview. Considering the extrapyramidal side effects of classical neuroleptics, a medication-related reduction of facial activity (additional to this habituation) could be expected and was already shown in former studies (Schneider et al. 1992). In fact haloperidol produced stronger extrapyramidal side effects than perazine, but since there was no meaningful correlation with facial activity, the differential effect of neuroleptic medication on facial reactivity seems not to be caused solely by different side effect characteristics. Moreover, as premedicated and nonmedicated patients did not differ in their facial expressivity at T0, medication does not seem to be a critical factor for the pathogenesis of the general attenuation of facial expressivity in schizophrenia patients. Considering the size of medication effects on facial activity found in the acute phase in relation to the size of the corresponding group difference between healthy controls and schizophrenia patients, it has to be concluded that the effect size of the typical neuroleptic medication used in the present study is too small to be of clinical significance. However, objective measurement could be a very helpful instrument in proving and developing new generation antipsychotics with more convincing effects on facial acitivity – or more general on affective flattening and negative symptomatology – in the future.

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