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ORIGINAL PAPER

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Kinematic analysis of facial behaviour in patients with schizophrenia under emotional stimulation by films with "Mr. Bean"

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Abstract In schizophrenic patients, motor functioning is substantially disturbed. Kinematic analysis is useful in examining this motor dysfunction. Using kinematic analysis, we aimed to investigate facial movement in schizophrenic patients responding to humorous film stimuli ("Mr. Bean"). Ultrasound markers were attached to pre-defined facial points while subjects watched a funny film sketch. The study included 21 schizophrenic in-patients (13 men, 8 women; mean (S.D.) age: 32.1 (10.4) years) and 30 healthy individuals (12 men, 18 women; mean (S.D.) age: 35.7 (11.0) years). Unmedicated schizophrenic patients showed an abnormally high initial velocity of laughing (IV), while patients treated with typical neuroleptics demonstrated an abnormally low IV. There was a significant positive correlation between severity of negative symptoms and IV. Kinematical analysis of facial movement using IV could help to distinguish subclinical Parkinsonian syndromes induced by typical neuroleptics from negative symptoms of schizophrenia.

Key words facial behaviour · facial movement · schizophrenia · emotion

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Introduction

Affective and emotional disorder is one of the characteristics, if not the central characteristic, of schizophrenia. As described long ago by Bleuler [6] and Kraepelin [20], affects are expressed in a person's motor activity and manifest themselves in facial behaviour, gestures, gait and bearing, etc. How a person feels expresses itself in involuntary movements of the facial muscles, particularly in emotional situations [16]. In schizophrenics, this facial behaviour often take on a bizarre appearance, looking "affected", emotionless, uncontrolled and wooden. The pattern of this facial behaviour, which can reach the point of spontaneous dyskinesia and stereotypic behaviour, was described in the older psychiatric literature long before the advent of antipsychotic drugs. These pathological facial patterns must be distinguished from others more likely caused by drugs. Certain psychotropic drugs also induce conspicuous facial expressions. Antipsychotic drugs, for example, cause neuroleptic-induced movement disorders (NIMD), which may occur in the form of early or late dyskinesias or in the form of a rigid Parkinsonian expression.

The differentiation and classification of these facial expressions would be of great benefit, given that for the psychiatrist, daily clinical routine includes "reading" the patient's face—"the mirror of the soul"—and given their priority in psychiatric deliberations on diagnostics and therapy. It would be of particular benefit in cases where diagnosis is ambiguous. For example, the question of whether a schizophrenic patient is suffering from negative symptoms, from neuroleptic-induced Parkinsonism or from depressive inhibitions in the context of a post-remissive fatigue syndrome is not only of high diagnostic relevance but also entails enormous therapeutic ramifications. In the first case one would administer antipsychotic drugs, in the second case decrease the antipsychotics dosage, in the third case

supplement an antidepressant. Another important question in differential diagnosis concerns patients with creeping symptoms which could be attributed either to an incipient schizophrenia or to depression.

Routine clinical observation by the psychiatrist is inevitably flawed by its subjective character and cannot aspire to the accuracy required for this kind of diagnostic differentiation. More accurate diagnostic classification might be achieved if discrete subclinical facial movement features could be registered. Operationalising and objectifying measuring methods could then help guide important diagnostic and therapeutic decisions. Up to now, studies of facial motor behaviour in psychiatric patients using such objectifying measuring methods have been inconsistent. Ratings have been carried out by independent observers, with serial photography or with post-hoc film analysis. Coding has been performed by trained personnel (Facial Action Coding System (FACS), [9] with electromuscular gram (EMG), and computer supported methods [27]. In these studies unmedicated schizophrenic patients showed a low frequency of joy [2] and reduced upper facial movement [26]. The EMG studies document a low activity level in the joy-relevant zygomatic muscle in unmedicated schizophrenic patients [21, 26, 27, 29]. In a recent FACS study [10], attenuated facial expression was found prevalent in schizophrenic patients and acutely depressed patients.

The overall outcome of all these studies was unspecific, namely that the facial activity of schizophrenic and depressive patients with and without medication is reduced. This result reflects a general methodological problem. In general, there are two different and contrary types of measuring method, each with its own specific shortcoming. One type is aimed at registering the wholeness and complexity of facial behaviour, using methods, however, which are very subjective and rater-dependent (for example video analysis). The other type, with EMG as an example, is highly objective but entails methods which are overly reductionist, permitting deductions about the relationship between muscle activity and emotions which may be too speculative.

What is needed is a measuring method that accomplishes both aims: that on the one hand accurately records an entire movement reaction to a well-defined emotional stimulus, and that on the other hand records objectively and reliably. At the same time this method should exhibit a high spatial and temporal resolution to ensure the accurate registration of even discrete, subclinical facial behaviour. This last point is particularly important given evidence that in schizophrenics the facial movement features are *specifically* impaired, that is they are impaired not as a whole but rather in isolated phases, for example in the onset-phase of the movement [7]. The method used in this study is a so-called *active measuring method* used originally in neurology and orthopaedics, and works with high-frequency light or ultrasonic markers fixed, in this case, to the face. This facilitates the exact and objective registration of the movements comprising a facial behaviour and allows a detailed and precise analysis of individual phases of movement with high spatial and temporal resolution.

The aims of the present study were to assess impaired facial behaviour in medicated and unmedicated schizophrenic patients using kinematic analysis, to investigate the effects of typical and atypical neuroleptics on facial expressivity in schizophrenia and to examine the relationship between movement parameters (initial velocity of laughing) and clinical variables such as intensity of negative symptoms in schizophrenic patients.

Methods

Subjects

Eight unmedicated and 13 medicated (six with typical and seven with atypical neuroleptics) schizophrenic in-patients were studied (ICD-10: F20.0, F20.1, F20.5; 13 men and 8 women; age: 32.1 ± 10.4 years (unmedicated: five men, three female, 31.9 ± 8.3-years-old; typical: four men, two female, 33.4 ± 11.1-years-old; atypical: four men, three female, 32.7 ± 9.8-years-old). The Brief Psychiatric Rating Scale Score (BPRS) [24] was on average 55.9 ± 12.7 points. The score of the Scale for Assessment of Negative Symptoms (SANS) [2, 8] was on average 53.7 ± 24.0 points. Three patients received biperiden for clinically observed extrapyramidal symptoms. None of these patients had extrapyramidal symptoms during the observation period. The control group consisted of 30 healthy individuals (12 men, 18 women; age: 35.7 ± 11.0 years).

The healthy controls were comparable to the schizophrenic patients in age and gender distribution. The normal subjects were not medicated and had no diagnosis of mental disorder. Exclusion criteria for the healthy subjects included medical disease at the time of investigation. The schizophrenic patients had neither neurological disease nor any other major medical disease at time of the study.

Written informed consent was obtained from all subjects according to the guideline as listed in the Declaration of Helsinki [30].

Kinematic analysis of facial behaviour

Facial behaviour analysis was performed using the active movement gauge CMS 70 (ZEBRIS Ltd., Tübingen, Germany). This instrument controls ultrasonic markers with high-frequency signals (35 kHz) which are registered in real-time with a maximum measuring rate of 200 Hz/number of markers. Evaluation of the markers is completely digital, which allows the exact three-dimensional determination of the markers' spatial coordinates in a temporal sequence of a few milliseconds and with a spatial resolution of 0.1 mm. The noise of the measuring system is below this spatial resolution and is therefore not a source of error. Head movements were eliminated by means of a reference marker attached to the headband. The ultrasonic markers were affixed with double-faced adhesive tape to the left and the right corners of the mouth. Activity in the greater and lesser zygomatic muscle, the risorius muscle and the depressor muscle of the angle of the mouth caused the ultrasonic markers to move. Two additional ultrasonic markers attached to the left and to the right medial corners of the eyes recorded the activity of the orbicular muscle. All of these muscles are innervated by the facial nerve.

The subject sat facing the ultrasonic-measuring-recorder, a video camera and a television set which presented stimulus material (a short "Mr. Bean" film) to induce the positive emo-

Fig. 1 Laughing movement after presentation of an emotional stimulus

tion "joy/amusement". After the recording, the subject was asked to evaluate how "funny" he or she found the sketch by means of a visual analogue scale (VAS funny, 16 cm long). During the test, which lasted at the most 5 min, facial movement was registered by the measuring system and a video recording of the subject's face was made simultaneously in order to match the emotional reactions and the ultrasonic measurement data. In addition to watching the Mr. Bean sketch, the subject was asked to perform arbitrary oral and ocular movements ("stretch the corners of your mouth wide apart", "squint your eyes") and this motor activity was also registered. This would provide a means of distinguishing arbitrary movements from expressive facial activity induced by the film. By means of a frame code generator, the film, the video recording of the subject's face and the movement measured by the ultrasonic markers were synchronized to the millisecond, making possible an investigation of the dynamics of the "laughing" movement in close relation to Mr. Bean's stimuli. The facial activity thus measured was analysed further with the integrated 3DA software. The data was corrected for measurement errors, and possible errors were reduced to a negligible degree by filtering the data with algorithms [15]. In this filtering process, a kind of sliding average was calculated with simultaneous data recording. "Laughing" was analysed in five film sequences which had been rated as particularly "funny" by three independent average persons prior to measurement.

For an analysis of laughter movement which can be imaged by 3DA as a gradient in space and time (Fig. 1), the following parameters were determined for all the ultrasonic markers: the relative distances and angles between the markers, the angles to a reference level, time measurements and from that, the initial velocity of laughter (start to maximum amplitude), the spatial amplitude, duration and speed of decrease of the laughter movement, and the latency between emotional stimulus and start and maximum of the facial reaction ("emotional reaction times").

The data reported in this study are for the initial velocity of laughter of the corners of the mouth and the facial reaction times obtained in the voluntary motor task. For the variables studied here, there was no significant influence by covariates such as age, gender, education etc. Test–retest-studies showed the parameters to be sufficiently stable, with a Pearson coefficient of approximately $r = 0.85$. Since no relevant differences between right and left side were detected, we present only data from the left side (see for further methodological details [23, 24]).

Statistical analysis

The kinematic parameters for facial behaviour were analysed separately using the statistics package SPSS for Windows (version 10.0). All values were normally distributed due the Kolmogoroff–Smirnov test ($P > 0.05$). Differences between groups were examined using *t*-tests for independent sample comparison. We also computed Spearman–Brown correlation coefficients between clinical rating scale scores (BPRS, SANS) and the initial velocity (IV) of laughing. All statistical tests were two-sided and the significance level was set at $P < 0.05$. In view of the exploratory character of our statistical analyses, the significance level was not alpha-adjusted.

Table 1 Emotional reaction times (time interval between presentation of an emotional stimulus ("Mr. Bean") and the onset/maximum amplitude of laughing movements in 21 schizophrenic patients and 30 healthy controls (arithmetical means derived from five film situations) for the left side in seconds

	Healthy subjects	Schizophrenic patients	*t*-Test
Onset of laughing	1.5 ± 0.7	2.1 ± 1.1	0.05
Maximum amplitude of laughing	2.6 ± 0.9	2.8 ± 1.4	n.s.
Difference	1.1 ± 0.4	0.7 ± 0.5	0.02

Results

Emotional reaction times

In the healthy controls, laughing started on average 1.5 s after presentation of the emotional stimuli (Table 1). The maximum motor response was reached 1.1 s later. Schizophrenics reacted much later than did the healthy individuals, starting to laugh only 2.1 s after emotional stimuli began. Nevertheless, the schizophrenic patients had already reached the maximum laughing behaviour another 0.7 s later, and were thus significantly faster than the healthy individuals. Regarding how the film was experienced emotionally, both the schizophrenic patients and the healthy subjects experienced approximately the same level of joy/fun (VAS-funny: 9.6 ± 3.4 vs. 11.0 ± 3.8, *t*-test: n.s.), but schizophrenic patients laughed significantly less often (10.6 ± 6.8) than did the healthy controls (26.1 ± 9.1) ($P < 0.001$; *t*-test).

Initial velocity (IV)

Non-medicated schizophrenics showed a significantly higher IV following the emotional stimuli (Mr. Bean) than did the control group (Fig. 2). Those patients who had been medicated for weeks with typical antipsychotics and who for part of this time had received biperiden for extrapyramidal symptoms showed a distinctly slower initial speed than did healthy subjects, whereas those patients who had received atypical antipsychotics (clozapine, olanzapine) exhibited a velocity similar to that of the healthy controls.

The differences observed seemed to pertain to involuntary motor laughing, because no difference was found between the groups in the initial speed of the voluntary "stretch the corners of your mouth wide apart" movement performed on our arbitrary request (Fig. 3).

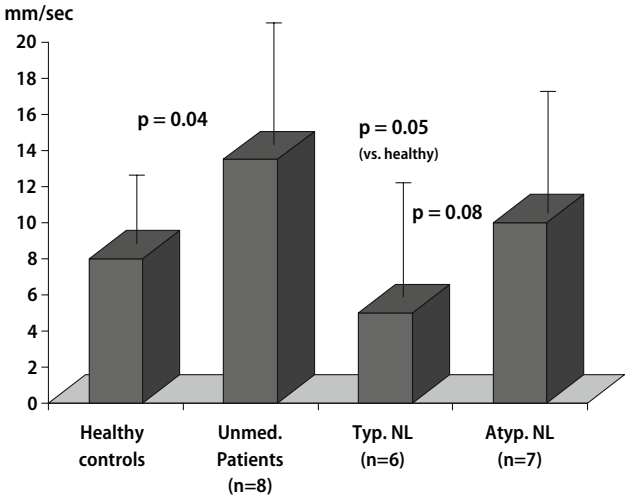

Fig. 2 Initial velocity of laughing computed for the left corner of the mouth after presentation of an emotional stimulus ("Mr. Bean") in unmedicated schizophrenic patients, schizophrenic patients treated with typical neuroleptics, schizophrenic patients treated with atypical neuroleptics and healthy controls

On the one hand, there were significant correlations between IV and the psychopathological condition of the schizophrenic patients: the higher the severity of the illness, the higher the initial velocity. The IV of schizophrenic patients tended to correlate with the BPRS total score, and its correlation with the BPRS sub-score "anxiety/depression" was significant (Table 2). On the other hand, there were no significant correlations with other sub-scores (e.g. "thought disorders"). There were also positive correlations with the SANS, particularly with affect scores, and to a lesser extent with scores such as "alogia" or "attention". The initial velocity of laughing tended towards a positive correlation with "blunted affect" and "abulia/apathy", and correlated significantly with "anhedonia" and the SANS total score.

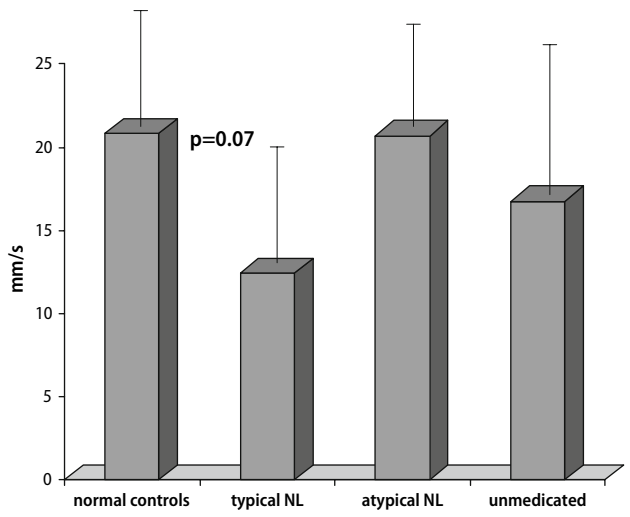

Fig. 3 Initial velocity of voluntary movements of the left corner of the mouth ("stretching the corners of your mouth apart") in unmedicated schiziophrenic patients, schizophrenic patients treated with typical neuroleptics, schizophrenic patients treated with atypical neuroleptics as well as healthy controls

Discussion

Emotional reaction time and IV in both groups showed divergent trends and seemed to be independent. Compared to the healthy controls, the schizophrenic patients showed an abnormally low laughing frequency and a delay in their facial reaction to Mr. Bean's pranks. Their expressive facial reaction to an emotional stimulus which they found "funny" was delayed in comparison to the response by the control population. Their facial reaction in and of itself, however, was significantly faster. This is not the pattern shown by depressed patients [17, 23]. At the same time, the schizophrenic patients experienced the film with much the same intensity and emotion as did the healthy controls. This contradiction between divergent timing and common emotional experience has been described by other authors [4]. The differences in reaction time must have another explanation.

A variety of reasons can account for the impaired facial behaviour in schizophrenic patients. There is a large body of evidence on cognitive impairments in schizophrenia. The delay in schizophrenic patients could be a result of impaired emotion recognition [19], of social cognitive impairments (such as an impaired theory of mind) [5, 22, 28] and of attention dysfunction [1, 12]. But the delay of facial reaction could also be a result of ineffective integration of emotion and cognition [14]. In the voluntary "cortical" task, however, there were no speed parameter differences between the schizophrenic patients and the healthy controls. Difference in speed parameters related only to emotional involuntary "subcortical" laughing. There is also evidence that in the different response–time paradigms—the schizophrenic group responded with delay—it was not attention performance which was impaired but rather the reaction itself, i.e. the involuntary "decision" to execute a motor action [25].

The comparison of the initial speed of schizophrenic patients to that of healthy controls showed that the un-medicated schizophrenics exhibited a higher initial velocity in laughing, i.e. that, once emotionally stimulated, the schizophrenics reached the maximum amplitude earlier than did the healthy controls. This rapid and shooting movement in the otherwise motionless face of the schizophrenic patient might be a correlative of the bizarre and parathymic physical and facial behaviour described very early by Heimann as a disintegration of facial behaviour [13].

A difference between typical and atypical antipsychotic drugs was also found. Patients on typical antipsychotic drugs showed a significantly lower initial velocity compared to controls. This could be seen as an indication of a Parkinsonian condition in these patients manifesting itself only fragmentarily in the form of hypomimic facial motion. This finding is consistent with the results of an earlier study [27]

Table 2 Spearman–Brown correlation coefficients reflecting the association between initial velocity of laughing (mm/s) computed for the left corner of the mouth and the psychopathological condition in 21 schizophrenic patients

	BPRS: total score	BPRS: anxiety/depression	BPRS: anergia	BPRS: thought disturbances	BPRS: activation	BPRS: hostility
Velocity	0.44^{+}	0.59**	0.36	−0.01	0.29	0.19
	SANS: total score	SANS: affective flattening	SANS: alogia	SANS: abulia/apathia	SANS: anhedonia	SANS: attention deficits
Velocity	0.48*	0.43^{+}	0.13	0.44^{+}	0.62**	0.04

$^{+}$ $P < 0.1$; * $P < 0.05$; ** $P < 0.01$

suggesting that typical neuroleptics lead to further reduction of facial activity in schizophrenic patients. In contrast to this, the initial velocity of laughing of patients on atypical antipsychotics, which as a rule do not induce EPMS, was faster than that of untreated patients but slower than that of healthy controls. This difference was not significant because of the small number of patients in these two subgroups treated with typical respectively atypical antipsychotic drugs. If a distinction could be verified, the initial velocity of laughing examined with facial movement analysis might possibly be used as a characterization of typical and atypical neuroleptics.

With respect to psychopathological differentiation, the main result was the eminently high initial velocity of laughing displayed by patients with distinctive negative symptoms. The same result was found in a small pilot study with chronic schizophrenic patients who suffered from distinctive residual schizophrenia, primarily with negative symptoms [18]. Notably, the initial velocity rates in the analysis of facial movement were related above all to those psychopathological SANS and BPRS scores which pertain to emotion and mood rather than to those representing cognitive functions.

The positive correlation of initial velocity with the BPRS sub-score "anxiety and depression" suggests that the assessment on the scale is mainly influenced by negative symptoms, which in most cases cannot be differentiated clinically. It remains to be clarified whether these associations are restricted to emotional reactions to humorous stimuli, since another study [11] which evaluated facial behaviour of schizophrenic patients in an emotional interview using FACS did not reveal any significant correlation between facial activity and the SANS observer rating of "global assessment of affective flattening".

The differentiation and classification of negative symptoms, depression and Parkinsonian symptoms present conceptual problems which have always flawed rating-scales and technical methods and which account for unspecific results [3, 11]. The method of facial movement analysis presented in this article opens a new and promising approach. Regarding the initial velocity of laughing, patients with negative symptoms showed eminently high rates, patients on typical antipsychotic drugs and presumably with subclinical parkinsonianism low rates. First results for depressive patients [17, 23] show that they have the lowest initial rates of all groups. If these results could be replicated in a larger sample, facial movement analysis could be applied with an active measuring method to distinguish patients with negative symptoms from those with Parkinsonian symptoms and those with depression. It might then be possible to objectify specific, particularly subclinical, conspicuous facial behaviour for the various groups of diseases. Thus, facial movement analysis may contribute part of the answer to challenging questions of differential diagnosis and better decision-making for the therapy of schizophrenic patients.

Several limitations of this study have to be considered. First, both groups were not matched for gender. The control group contained a higher proportion of women compared to the patient group. Since women tend to have shorter emotional reaction time and/or lower initial velocity in general, this could explain in part the significant differences between the two groups. Secondly, subgroups of schizophrenic patients were very small and the results were not corrected for multiple testing. Therefore, results were of preliminary nature and further studies have to verify our hypothesis.

Conclusions

Unmedicated schizophrenic patients showed a significantly higher IV as healthy controls, while patients treated with typical neuroleptics demonstrated low IV. There was a positive correlation between severity of negative symptoms and IV. Kinematical analysis of facial movement using IV could help to distinguish subclinical Parkinsonian syndromes induced by typical neuroleptics from negative symptoms of schizophrenia.

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