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Non-verbal Behavioural Dysfunction in Schizophrenia
Vulnerability Indicator, Residual Marker or Coping Strategy?

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Psychiatric disorders are psychopathologically characterised by signs and symptoms. Although diagnosis and classification rely heavily on the patient's report of subjectively experienced symptoms, the assessment of the underlying psychopathological process can be impaired by the patient's distorted self-image, his cognitive abnormalities, and his limited capacity to express himself verbally. Non-verbal behaviour, however, lends itself to objective and generalisable assessments: it can be reliably and accurately observed and measured, and although subject to cultural influence, it is the infant's most elementary form of self-expression (Gaebel, 1990). In addition, it plays a major role in interpersonal communication, which is disturbed in all psychiatric disorders. Therefore, the analysis of non-verbal behavioural dysfunction offers an important approach to the complex biopsychosocial framework of mental disorders.

Non-verbal behaviour and schizophrenia

Definition

Non-verbal behaviour is comprised of facial action, hand and body movements, eye movements, and speech and voice characteristics. Different forms of behaviour, such as reflexive, expressive, instinctive, and purposeful instrumental behaviour can be distinguished by means of stimulus situation, target orientation, and adaptiveness (Leonhard, 1976). This paper focuses on expressive and instrumental behaviour. Behaviour is defined as an organism moving in space and time (Bente, 1978), and can be objectively assessed either by means of complex coding systems or by measurement techniques (Scherer & Ekman, 1982).

Fundamental symptoms of schizophrenia

According to Bleuler (1920, 1966), disturbances of association, affect, ambivalence, and autism (the four a's), and disturbances of will, action, and person are the basic symptoms of schizophrenia. A "defect of affective modulation, and affective rigidity" are the most important signs of the illness. Whether a number of behavioural abnormalities (e.g. stupor, hyperkinesis, stereotypes, automatisms) summarised under the heading of catatonic symptoms belong to the 'accessory' characteristics, are manifested all or only part of the time, they complicate the clinical picture (Bleuler, 1920).

Kraepelin (1896) and Bleuler (1920) viewed disturbances of affect as intrinsic diagnostic characteristics of dementia praecox or schizophrenia, respectively, whereas modern diagnostic systems influenced by Kurt Schneider's (1987) Symptom Theory (e.g. DSM-III-R; American Psychiatric Association, 1987) do not designate them such an important role. Affective flattening, on the other hand, is now considered to be a core feature of "negative symptoms" (Walker & Lewine, 1988), which is either characteristic of a distinct subtype of schizophrenia with distinct aetiopathogenesis (Crow's Type II, 1985), or complicates the course of the illness for various reasons. According to Frith (1987) behavioural disturbances would be a more appropriate term than negative symptoms, reflecting the particular kind of underlying biological deficit.

Primary and secondary negative symptoms

According to Strauss *et al* (1974), positive symptoms, negative symptoms, and social deficits are the three central psychopathological dimensions in schizophrenia. Carpenter *et al* (1985, 1988) distinguished between primary negative symptoms and social deficits, e.g. social withdrawal, on the one hand, and secondary negative symptoms due to depressive or extrapyramidal symptoms and social deprivation on the other. Primary negative symptoms could accordingly be understood as a form of non-verbal behavioural dysfunction related to the postulated disease process. Their exact empirical definition, nosological specificity, time specificity, treatment responsiveness, predictive validity, and pathogenesis are therefore important areas for future research.

Assessment and differentiation of non-verbal behavioural dysfunctions

A variety of authors have called for a more objective research approach to non-verbal psychopathological characteristics in schizophrenia, because of its superior data quality and biological validity (Hill, 1974; Helmchen & Renfordt, 1981; WHO/ADAMHA, 1983; National Institute for Mental Health, 1988).

If one agrees with most authorities that blunted affect is the central negative symptom, it must be defined in clear and operationalised terms, so as to be distinguished from 'emotion' and 'mood' (Berner, 1988). The DSM-III-R glossary gives the following definition:

> "Affect: A pattern of observable behaviours, i.e. the expression of a subjectively experienced feeling state (emotion) . . . the normal expression of affect involves variability in facial expression, pitch of voice, and hand and body movements." According to DSM-III, "affect is to mood as weather is to climate".

Accordingly, affect and its disturbances are defined in terms of non-verbal characteristics which are indicators of subjective emotional states. An experimental approach seems appropriate to assess the relationship between behaviour, emotion, and both central and peripheral measures of activation ('arousal') under both normal and pathological conditions (Holzman *et al*, 1988).

Recent empirical research on negative symptoms relies on several rating scales which include different symptom domains (Walker & Lewine, 1988). While Crow (1985) considers affective flattening and poverty of speech to be the only core negative symptoms, Andreasen (1982) also includes a variety of cognitive, attentional, and social characteristics in her Scale for the Assessment of Negative Symptoms (SANS). However, in accordance with the affect definition of DSM-III-R, her scale dimension "affective flattening" is operationalised by means of observable behavioural characteristics (Table 1).

Table 1
Behavioural characteristics (SANS; Andreasen, 1982) and objective assessment of negative symptoms in schizophrenia

Item	Behaviour	Measurement
Unchanging facial expression Affective nonresponsivity Inappropriate affect	facial action	FACS
Decreased spontaneous movements Paucity of expressive gestures	hand-/body-movements	Coding
Poor eye contact	eye movements	IROG
Lack of vocal inflections Poverty of speech Increased latency of response	voice speech	Pitch frequency on/off pattern

The different behavioural sectors corresponding to the scale items can be assessed separately (to exclude possible audio-visual interference) by objective means. Moreover, a strict separation of 'molecular' (simple, individual), and 'molar' (complex, interactional) behavioural characteristics seems to be indicated, both for methodological and interpretative reasons.

Non-verbal behavioural dysfunctions as disease markers

Concept

Biological marker research aims at pinpointing pathogenetically relevant prephenomenal characteristics beyond the 'noise' of psychopathology. Accordingly, state or episode markers are characteristics present only during acute exacerbations, whereas trait markers persist during premorbid, acute, and post-acute phases of the illness. On the other hand, residual markers are characteristics which relate to non-remitting or chronic states of the disorder (Zubin *et al*, 1985).

> A precondition for this kind of differentiation would be, however, that the continuous illness course could be correctly divided into biologically relevant discontinuous stages by psychopathological means (e.g. "symptomatic" v. "asymptomatic"). Since positive, negative, and social symptoms have a different time course (Gaebel, 1989*a*), the possibility of such a differentiation seems doubtful. Last of all, a delimitation of illness-related time-stable characteristics from persisting personality characteristics is difficult.

'Vulnerability marker' is another term referring to familial or sporadic characteristics which are either stable or semi-stable ('mediating'), and reflects the disposition for schizophrenia (Zubin *et al*, 1985; Nuechterlein, 1987). A manifest schizophrenic episode is the result of a complex interaction between, on the one hand, intrinsic vulnerability and protective factors, and on the other hand, environmental stressors and protective factors.

According to Zubin (1985), negative symptoms are not markers of vulnerability:

> "Vulnerability theory takes the position that negative symptoms are neither an inevitable (Kraepelin) nor an intrinsic (Wing) feature of schizophrenic disorders. It is proposed, instead, that the clinical poverty syndrome . . . is essentially an artifact or a social consequence of having been identified, labelled and treated as schizophrenic."

Contrary to this apodictic statement, Pogue-Geile & Zubin (1988) have discussed various aetiological models of negative symptoms which cannot be decided between, due to lacking empirical data.

Research on the clinical status of non-verbal behavioural dysfunctions

Cross-sectional relationships of negative symptoms with other psychopathological characteristics, like positive symptoms and their longitudinal stability, are indicative of their particular association with the course of illness. From research using different psychopathological observer rating scales such as AMDP, BPRS, and SANS (Gaebel, 1989*a*), it is clear that an acute psychotic exacerbation, i.e. an increase in positive symptoms, is often paralleled by an increase of affective disturbances which partly decrease when remission takes place. This has led to the assumption that affective disturbances are partially responsive to neuroleptic treatment. However, due to their longer time course, their decrease usually lags behind psychotic remission (see Fig. 1).

In Fig. 1, the categorical subtypes I and II could be mistaken for trait-like characteristics at a certain cross-section, if the phase lag of those syndromes is not taken into account by longitudinal observation. Longitudinally, disturbances of affect and drive are neither an exclusive nor a stable characteristic of a schizophrenic subgroup. They are more or less fluctuating characteristics which are present in most schizophrenics at different times of their illness. Accordingly, from cross-sectional research, positive, negative, and nil correlations have been described between negative and positive symptoms (Walker & Lewine, 1988). Contrary to Zubin's (1985) view, the alternative hypothesis should be tested that behavioural indicators of affective disturbances do have the status of a disease marker, since they reflect a fluctuating (hypothetical) vulnerability or some related 'adaptive' coping mechanism.

> Clinical experience does not demonstrate the validity of defining vulnerability in terms of a time-stable functional biological characteristic. This concept has been criticised, for example, with regard to a possible age-dependency of vulnerability (Katschnig *et al*, 1990). For reasons of simplicity, however, a time dimension has not yet been included in the vulnerability concept (Zubin, personal communication).

Accordingly, disturbances of affect could indeed be intrinsic to schizophrenia, caused by vulnerability-related dysregulatory and/or counter-regulatory psychobiological mechanisms. The vulnerability theory could assign them the status of either an intermediate vulnerability indicator (longitudinally), or a residual marker in the post-acute phase (cross-sectionally). On the assumption that the presence of acute psychotic symptoms is related to a relative increase in vulnerability, the presence of affective disturbances could be related to a secondary mechanism.

Nosological specificity

Biological psychiatry has increasingly moved away from explaining biological correlates of psychiatric disorders in terms of nosology and towards an underlying-systems' dysfunction. Psychopathological symptoms/syndromes and their pathobiological correlates are hence indicators of dysfunctions of

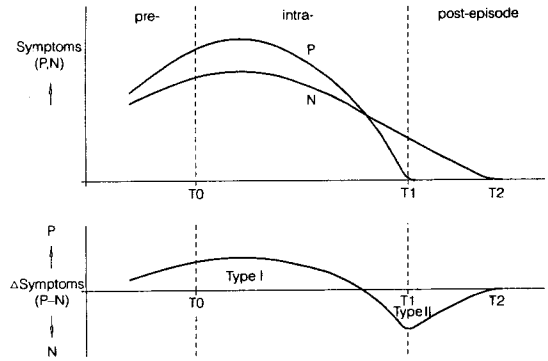

Fig. 1 Schematic representation of positive (P) and negative (N) symptoms in the pre-, intra-, and post-episodic phase of schizophrenia. Type I or type II subtypes of the illness could be erroneously diagnosed at T_0 or T_1 respectively by cross-sectional subtraction of positive and negative symptoms (P – N). Longitudinally, however, both subtypes are dependent on the phase of the illness.

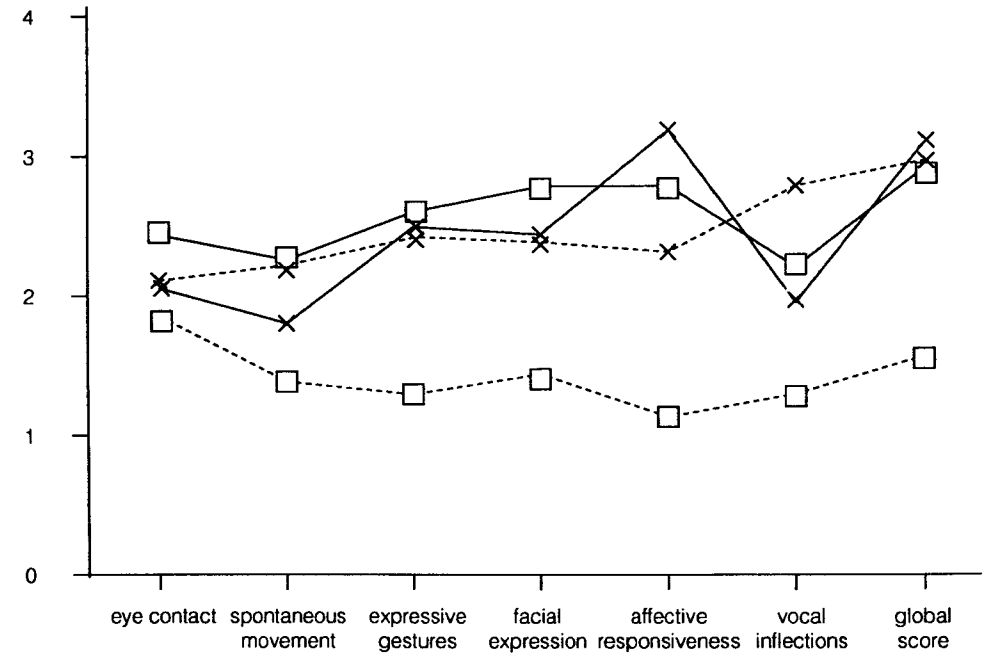

Fig. 2 Profiles of affect in 15 schizophrenic (—) and 15 major depressive (- - -) patients (RDC) in their acute (T_0(×)) and postacute phase after 4 weeks of treatment (T_1(□)). Depicted are non-verbal behavioural characteristics from the dimension "affective flattening" of the SANS (0 = none, 4 = marked).

certain neurobiological systems which basically might be involved in various mental illnesses.

Obvious difficulties in differentiating affective disturbances in schizophrenics from depressive or neuroleptically-induced extrapyramidal symptoms, as well as from affective disturbances in depressive or Parkinsonian patients, seem to confirm the nosological non-specificity of this family of syndromes.

From Fig. 2, for example (Gaebel & Renfordt, unpublished), it is obvious that in the acute stage of their illness, schizophrenic and depressive patients show no difference in the intensity and profile of affective disturbances, as measured by the SANS. However, both groups can be distinguished from one another by means of the time course of affective disturbances.

Needless to say, the dimension of subjective experience which informs about conscious or unconscious psychological coping processes may not be excluded (Strauss, 1989*b*). Accordingly, affective behaviour and subjective experience are often incongruent, particularly in schizophrenia (Bouricius, 1989).

Since biological functions, e.g. the electrodermal orientating response, are similarly disturbed in schizophrenics with negative symptoms and in retarded depressives, a common biological origin of both syndromes seems to be evident (Heimann, 1985).

Non-verbal behavioural dysfunctions as adaptive strategies

Concept

Nuechterlein (1987) devised a tentative working vulnerability/stress model of the development of negative symptoms in schizophrenia. Reduced available processing capacity and subclinical anhedonia are presumed to be personal vulnerability factors, whereas individual protective factors are not mentioned. In a more general conceptual framework of possible factors in the development of schizophrenic episodes, personal vulnerability factors on either pathobiochemical, pathophysiological, or phenomenological levels are dopaminergic dysfunction, reduced available processing capacity, autonomic hyperreactivity to aversive stimuli, and schizotypical personality traits. On the other hand, coping abilities, self-efficiency and antipsychotic medication are supposedly personal protective factors.

The assumption that trait characteristics are indeed indicators of vulnerability and do play a pathogenetic role in the causal chain of illness manifestation, has to date not been verified. Moreover, the term 'vulnerability factor' is often not adequately defined so as to distinguish it clearly from such terms as 'risk factor', 'indicator' or 'predictor' (Katschnig *et al*, unpublished), and furthermore, there is no evidence that vulnerability factors can be clearly distinguished from protective ones, nor protective factors from 'compensatory' or 'repair' ones. 'Coping', on the other hand, can be interpreted as an active and conscious psychological act that does not seem to coincide with a passive and unconscious defence, although it is sometimes very difficult to distinguish between them. Because of this confusion in terminology, 'adaptation' might be a preferable term for an integrative concept which takes into account the interaction of biological, psychological, and social processes which follow from and shape a primary illness-related dysregulation.

Molar behaviour

It has long been supposed that certain forms of schizophrenic behaviour, such as emotional and social withdrawal, are coping strategies acquired to minimise cognitive disturbances (e.g. Hemsley, 1987). Overt withdrawal could occur if adaptive perceptive styles ('reducing', 'minimal scanning' – Schooler & Silverman, 1969) are no longer appropriate to control the intensity and complexity of stimuli. Similarly, emotional turmoil arising from dysfunctional social interaction (deficits in decoding, encoding, or problem solving concerning social signals) could increase withdrawal tendencies. If these forms of behaviour are targets of therapeutic interventions, a functional behavioural analysis has to precede them. In the case of a purely behavioural deficit, a simple 'motor' skills model might be adequate; in the case of adaptive, though dysfunctional social behaviour, interventions based on a more complex (e.g. problem solving) model would be more appropriate (Bellack *et al*, 1989).

Molecular behaviour

Facial action

Facial muscles play an important role in emotional expression. The facial expression of the six basic emotions (fear, surprise, anger, disgust, sadness, happiness) is universally encoded and decoded, pointing to a common neurobiological basis of expression and impression (Ekman & Friesen, 1980). Normally, affective expressions result from involuntary ('emotional') and modulating voluntary ('display rules') innervations, originating from different neuronal structures (Rinn, 1984).

Since facial muscles are often involved in mixed emotional and non-emotional states, a separate assessment of facial activity by means of the Facial Action Coding System (FACS; Ekman & Friesen, 1978) by trained raters is required. Videotechnology is an important tool for this kind of approach.

Objective assessments of facial action in schizophrenics date back to the 1950s. Spoerri & Heimann (1957) reported both typical facial expressions and a syndrome of disintegration in schizophrenia (Heimann & Spoerri, 1957). Such distintegrative phenomena have to be distinguished from subtypes of tardive dyskinesia. Concerning differences between schizophrenic negative symptoms and extrapyramidal akinesia, there is evidence that Parkinsonian patients exhibit less facial emotional expression than schizophrenics (Alpert & Rush, 1983).

Pitman *et al* (1987) used an ethological scoring system, and found that non-paranoid schizophrenics more often demonstrate speech-independent autistic eyebrow movements than paranoid schizophrenics and non-schizophrenic psychiatric controls. According to Steimer *et al* (1988), schizophrenics show a decrease of affective facial expression, particularly in the upper face, but an increase of voluntarily produced social signals. Hence, schizophrenics were rated as showing less authentic affect and fewer facial illustrators and regulators.

The particular pattern of facial muscle innervation has to be taken into account when considering the biological meaning of these findings (Rinn, 1984). Whereas the lower face muscles – because of their relationship with the motor speech centre – are better voluntarily controlled, the upper face muscles are more involuntarily controlled due to their connection with the affective centres.

The central integrator of emotional information is the amygdala, which receives information from the thalamus and the cortex and projects on to affect-specific effector systems (Le Doux, 1986). Although it is still unclear which subsystem of this complex network is disturbed, the subordinate modality-independent cortex (hippocampus) could play a crucial role. For an adequate differentiation of schizophrenic expressive disturbances on the phenomenological level, a task-specific assessment (decoding: perception of affect; encoding: spontaneous expression, simulation, imitation) should be adopted during the course of the illness under different therapeutic interventions (Gaebel & Renfordt, unpublished). Based on this kind of research, the question could

be answered whether disturbances of facial action in schizophrenia represent a biologically-determined vulnerability indicator or a coping strategy for emotional arousal. In terms of the adaptive effects of self-induced behaviour, it must be clarified whether control mechanisms such as deintensification, neutralisation, and masking could assist in dampening emotional experience by means of 'facial feedback', or if a primary deficit in experienced emotions is responsible for certain expressive deficits (e.g. anhedonia). The complexity of this problem can only be solved within a multidimensional approach.

Gaze

Gaze is another non-verbal characteristic which can be coded reliably by trained raters in terms of directionality, frequency, and duration. It is also accessible to objective and exact assessment and analysis. In addition to electro-oculography (EOG), infra-red reflection techniques (infra-red oculography, IROG) have been developed which do not require any application at the patients' head (corneal reflection–pupil centre measurement).

Besides more basic oculomotor disturbances (Gaebel, 1989*b*), some of which are possible candidates for traitmarkers, reduced eye contact of schizophrenics during interviews has been reported (Rutter & Stephenson, 1972). A significant reduction of the duration of eye contact has been found in non-paranoid compared with paranoid schizophrenics and non-schizophrenic psychiatric controls (Pitman *et al*, 1987). Pansa-Henderson *et al* (1982), however, reported generally reduced eye contact and more frequent gaze deviation in schizophrenics, compared with depressives. According to Meya & Renfordt (1986), these gaze characteristics improve in treatment responders, but not in non-responders. The meaning of this finding is ambiguous: on the one hand, the maladaptive strategy could be abandoned when the underlying disturbance improves with neuroleptic treatment; on the other hand, this maladaptive behaviour could be part of the clinical symptoms, decreasing with clinical improvement.

The perceptual object in this kind of research is the human face. Since schizophrenics, in addition to their expressive disturbances, have a stable deficit in perceiving and recognising facial affect (Morrison *et al*, 1988; Gaebel *et al*, 1989), the role of explorative eye movements, which assist instrumentally in fixating, and hence centrally analysing, emotionally relevant facial elements, should be focused on. This relationship can be assessed under controlled conditions using a standardised set of affective stimulus photos of faces. It has been found that schizophrenics not only have deficits in the ability to decode negative affects, but also show abnormal gaze behaviour, with longer fixation durations compared with healthy controls. Longer fixation durations have already been found in schizophrenics with negative symptoms and poor perceptual performance, and have been related to the perceptual style of 'minimal scanning' (Gaebel *et al*, 1987). Luborsky & Blinder (1965) have described similar behaviour as a correlate of a defence mechanism ('repression') in healthy volunteers, but have left open its possible biological foundaton. Again, to classify these findings either as indicators of a personality-related adaptive perceptive style or of a dispositional information-processing deficit requires a multidimensional approach.

Moreover, a shift of the gaze axis into the right visual hemisphere particularly for negative emotions displayed in facial cues, has been found in post-acute schizophrenics (Gaebel *et al*, 1989). The possibly underlying dysfunction of this residual marker (asymmetrical effect of neuroleptics on lateralised attentional structures, hemisphere-specific functional deficit, active exploration of the more informative left half of the face, etc.) is not yet understood. However, these findings demonstrate that behavioural methods can be used to analyse interactional processes ('face-to-face' contact, 'expressed emotions') and their importance as determinants of the course of the illness.

Speech behaviour and voice characteristics

Speech behaviour and voice characteristics are also accessible to objective assessment and analysis. Measured in terms of speech phases and speech pauses, speech activity can be analysed as an on/off pattern, irrespective of speech content (Krüger, 1989). Moreover, certain voice characteristics, e.g. frequency, modulation, are objective indicators of emotional and psychopathological states (Wallbott, 1989).

Spoerri (1961) described certain vocal characteristics (*Würgstimme*) in chronic schizophrenics. Those with pronounced negative symptoms have been described as having aprosodia, similar to that in neurological patients with right-hemisphere dysfunction (Fricchione *et al*, 1986). Andreasen *et al* (1981) have reported a reduced variability of amplitude and frequency of voice in schizophrenics with affective flattening. Decrease of the frequency of pitch after treatment in acute schizophrenics and depressives has been interpreted as a nosologically non-specific reduction in arousal (Tolkmitt *et al*, 1982). Concerning 'poverty of speech', Ragin *et al* (1989),

Table 2
Mean pitch frequencies (F0) and coefficients of variation (CV) in male schizophrenics ($n = 7$) and depressives ($n = 4$) at T_0 and T_1 (speech probes 1–7)

	Schizophrenics				Depressives			
	F0		CV		F0		CV	
	T_0	T_1	T_0	T_1	T_0	T_1	T_0	T_1
1 Happy	127.6	126.1	12.9	13.5	142.0	129.8	10.2	10.7
2 Sad	122.4	117.8	12.9	11.6	137.1	132.1	9.2	11.4
3 Angry	122.9	121.2	12.5	11.6	127.3	119.6	10.1	10.7
4 Neutral	121.5	121.8	11.4	11.5	131.0	128.5	7.3	10.1
5 Counting	120.8	120.6	7.1	7.5	131.5	126.2	5.6	6.8
6 Yes	110.0	108.0	1.9	3.8	124.8	118.5	3.4	5.0
7 No	111.0	112.6	2.7	3.3	125.4	120.3	3.8	4.8

observed by objective means that depressed patients revealed a higher level of this than schizophrenics at admission. On the other hand, in the course of treatment, the reverse applied. These findings correspond to the findings mentioned above on vocal inflexions (see Fig. 2), and underscore the necessity of internosological longitudinal research.

Again, emotion-specific changes of voice modulation can be observed in schizophrenics during the acute illness course (Table 2).

While depressed patients in remission show an increase of voice modulation (coefficient of variation of pitch frequency) in different speech probes (emotional and neutral sentences, counting, yes/no), schizophrenics only show an increase in emotionally positive or in neutral sentences, and a decrease in emotionally negative samples (Gaebel & Renfordt, unpublished). As in the case of facial action, these findings contribute to the question of adaptive feedback control of negative emotions being the basis of non-verbal behavioural dysfunction in schizophrenics.

Underlying biological mechanisms

Dopaminergic system

The dopamine hypothesis of schizophrenia has served as a biological model for the occurrence of positive as well as negative symptoms (Meltzer, 1985). Briefly, positive symptoms are presumably related to a hyperactive, and negative symptoms to a hypoactive, mesotelencephalic dopaminergic system. Analogous to extrapyramidal motor symptoms, dysfunctions of inhibitory and excitatory dopaminergic mechanisms are a possible explanation of both clinical syndromes (Alpert & Friedhoff, 1980). On the other hand, variability of dopaminergic activity during the course of illness has been linked to adaptive functions of the dopaminergic system (Csernansky *et al*, 1983). Decrease in receptor density with ageing in animals could explain an increase in negative symptoms during the course of the illness (Csernansky *et al*, 1985). In cybernetic terms, the function of the dopaminergic system could be a physiological 'buffer' which is responsible for restitutive and/or homeostatic brain mechanisms (Davila *et al*, 1988). Accordingly, affective disturbances in the framework of negative symptoms could reflect an autonomous downregulation of the dopaminergic system as an adaptive mechanism, following certain 'stressors'. There is pharmacological accordance with this kind of dynamic pathogenetic model (Carlsson, 1987). Corresponding to a functional model of psychotic and affective symptoms, vulnerability to illness and its clinical indicators should be seen as a dynamic process.

Cholinergic system

Besides the dopaminergic system, other transmitters and neuromodulators play a role in the development of schizophrenic symptoms. Accordingly, the noradrenergic, serotonergic, GABAergic, and cholinergic system are possibly involved in affective disturbances. The interaction between dopaminergic and cholinergic systems is particularly well-known from extrapyramidal motor disturbances and their pharmacological control. In cybernetic terms, corresponding to earlier concepts of an imbalance of ergotropic and trophotropic vegetative functions in the course of functional psychoses (Selbach, 1961), compensatory cholinergic hyperactivity due to dopaminergic hyperfunction has been related to non-verbal behavioural dysfunction (Tandon & Greden, 1989). Accordingly, the development of residual symptoms in the post-acute phase of psychosis has been related to a delayed normalisation of the cholinergic system's functions.

An integrative model of non-verbal behavioural dysfunctions

The diagnosis of schizophrenia is still made descriptively by means of defined clinical characteristics. Psychopathological characteristics are a key to understanding the pathogenesis of this illness due to their biological foundation. Nevertheless, because of their different pathognomic importance, a distinction between fundamental and accessory symptoms has been established. This differentiation relies in part on the temporal stability of characteristics, as in marker and vulnerability research, but even if established by longitudinal assessment, temporal stability would not sufficiently define a pathognomonic characteristic. On the other hand, a persistent characteristic is probably so far from the mechanisms of manifestation of the illness that it will not contribute to the understanding of the pathogenesis of symptoms. Attributes covarying with symptoms, however, are difficult to distinguish from epiphenomena.

Although biological research is especially concerned with the isolation of trait markers, episode markers could promote insight into pathogenetically relevant processes. Moreoever, certain assessment levels usually reflect adaptive, compensatory, or reparatory processes rather than the illness process itself. Analogously, it is difficult to decide if a phenomenon corresponds to loss of function or active inhibition, functional increase or disinhibition of involved functional systems. To analyse a functional disturbance, i.e. a hypofunctional, hyperfunctional, or dysfunctional state, requires a cybernetic model which is derived from the regularity of normal functions. Starting from here, findings of different assessment levels (e.g. psychopathology, pathophysiology, and pathobiochemistry) could be pathogenetically linked to each other. Until the pathogenetic links between biological basis, stressors, and clinical symptoms are clarified, vulnerability theory should refrain from claiming that time-stable characteristics are vulnerability indicators (Brenner & Böker, 1986).

Accordingly, behavioural dysfunctions not only for methodological reasons, but also because of their intimate linkage with the course of illness are a relevant psychopathological surface structure for the pathogenetic understanding of schizophrenic disorders. Adequate research strategies will enable investigators to decide if non-verbal maladaptive behaviour reflects a vulnerability indicator, a residual marker, or a coping strategy. In a behavioural–biological framework with adaptation as a core characteristic of living systems and under consideration of bio-cybernetic regulatory principles, all three aspects are not independent but complementary (Fig. 3).

A certain behavioural baseline characterises a premorbid homeostatic starting point (Fig. 3a, b, c), where (hypothetical) excitatory and inhibitory processes are in a relative balance, indicating the respective vulnerability or decompensatory readiness of an individual. Schizophrenics tend to manifest a reduced level of behavioural activity, the equilibrium thus

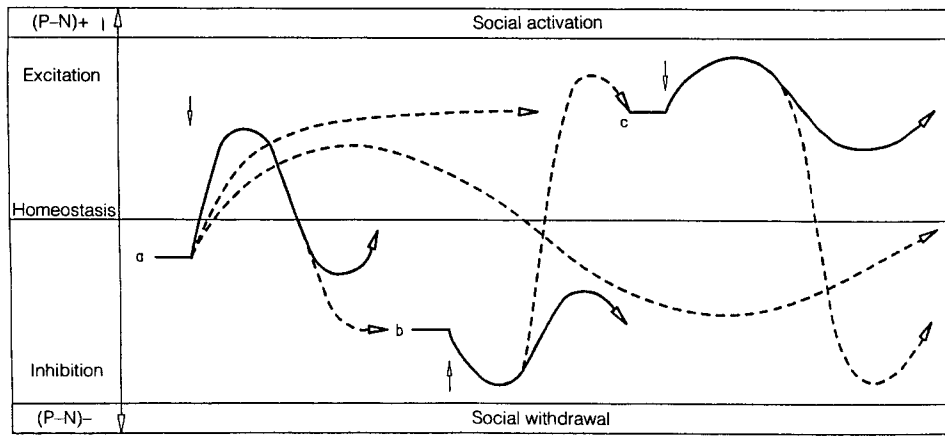

Fig. 3 Functional model concerning onset and course of psychopathological and social symptoms in schizophrenia (see text). (P = positive symptoms, N = negative symptoms; ↓ = stressor destabilising the system's functional equilibrium; a, b, c = types of premorbid function equilibrium (asymmetrically excited or inhibited) and their possible outcomes after destabilisation; degree and direction of functional asymmetry could be related to vulnerability characteristics.) (Modified from Selbach, 1962.)

being shifted towards the inhibitory pole (a, b: preceding defect, schizophrenia simplex). Nevertheless, many transitional forms are theoretically possible and swings towards the excited pole can also be observed (c: hebephrenic behavioural activation). Should decompensation occur for whatever reason, i.e. a relative overactivation of the excitatory system, the inhibitory system will be induced ('coping'): positive symptoms and behavioural disturbances then increase. Depending on the time course of the respective functional system, a variety of more or less fast counter-regulations and oscillations have to be taken into account. Persistent dysregulation (*Sollwertverstellung*), elicited by therapeutic interventions or other influences, can produce prognostically important residual changes.

From this preliminary functional model, it is clear that a micropsychopathological approach to the developmental course of illness can promote insight into auto-regulatory processes which reflect the interaction between vulnerability, coping, and residual changes (see Strauss, 1989*a*). A multi-dimensional research approach will ultimately open up new vistas in reaching a better understanding of transactional processes involved in the onset and course of schizophrenia.

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