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An Operationalized Multisymptomatic Model of Neuroses (OMMON): Toward a Reintegration of Diagnosis and Treatment in Behaviour Therapy*

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Summary. Experimental results from a sample of 216 patients with four different "symptom-neuroses" show that 65% to 90% of these patients have different combinations of multiple symptomatology.

With a background of these data, we present an Operationalized Multisymptomatic Model of Neuroses (OMMON), based on self-rating assessment of these patients on 4 symptom scales. Individual ratings on each scale are dichotomized into (+) or (–) results with regard to defined cut-off points and the model is derived from their 16 mathematically possible combinations. Subsequent analysis of these data (from a single test application) with our Varying Cut-Off Point Assessment (VACOPA) leads to hypotheses regarding causal symptom interactions and prediction of symptom changes over time, easily evaluated by repeated test application.

In treatment research the model seems suitable to: (a) build more homogeneous diagnostic groups; (b) operationalize varying degrees of "neurotic" disturbance, from "normal" via "client" to "patient"–independently of existing illness theories; (c) support prognoses of individual developments within and without treatment; (d) specify treatment aims and optimal sequences of interventions; (e) monitor predicted outcome; (f) reconsider earlier apparently contradictory outcome studies; (g) evaluate theoretical concepts regarding "neurotic" symptom formations in neuroses, psychoses, and psychosomatic disturbances.

For treatment purposes, OMMON should only be used together with "clinical" hypotheses; its prognostic potential can be increased by additional application of our Operationalized Multivariate Model of Motivation (OMMOM).

All three diagnostic approaches may be used for mutual evaluation.

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1. Introduction

Separation of diganosis and treatment has become one of the major obstacles for further scientific advancement of behaviour therapy in the eighties. It has seriously affected communication between research workers and practitioners in our field.

1.1 History of Separation of Diagnosis and Treatment

Behaviour therapy in the 50s and early 60s was guided by the assumption "the symptom is the neurosis" (Eysenck 1959). Consequently, assessment of "neurosis" on a single-symptom basis seemed easy and adequate. For the vast majority of participants in the early analogue studies—rather well-adapted students with isolated or monosymptomatic complaints *(clients)*—this may have been a suitable assumption. Such an operationalization of "neurosis" of course is very different from the one used in psychiatry or psychoanalysis.

In the late 60s and 70s, behaviour therapy with severely disturbed neurotic, psychotic, and somatically ill patients has shown, that a number of behaviour modification principles, derived from studies with *clients* cannot directly be transferred to treatment of *patients* (frequently individuals with multi-symptomatic complaints and serious restrictions in private and professional life-style)—and vice versa.

Increasingly complex, "multi-modal" treatment strategies have been developed for these patients. Concomitantly, there have been numerous attempts to develop adequate diagnostic strategies to support, improve and evaluate these complex treatments, e.g. direct behaviour observation methods (Hutt and Hutt 1970; Jones et al. 1974; Liberman et al. 1974), goal attainment scaling (Kiresuk and Garwick 1975) or "multi-method clinical assessment" (Nay 1979). But, because of time and skill requirements regarding data collection and analysis, their application is usually restricted to well staffed research projects. On the other hand, attempts to reduce the complex reality of a patient to a few variables, have produced some assessment instruments which reliably measure isolated problem or symptom areas, but do not allow adequate assessment of neurotic disturbance. Therefore, until now development and conduction of complex treatment strategies have mainly been guided by clinical decision strategies (Kanfer and Saslow 1969; Kanfer and Grimm 1977, 1980; Hand 1981) and not by diagnostic assessment methods.

1.2 Consequences from Separation of Diagnosis and Treatment

While work with increasingly disturbed patients has induced the development of increasingly complex treatment strategies, at the same time it seems to have reduced our capacity to "objectify" indication and effects of the specific in-

gredients of such treatment packages (Paul 1961; recent discussion in Agras and Berkowitz 1980; Barlow 1980). In fact, treatment research has created quite some confusion in the communication between research workers and practitioners—due to inappropriate description of patients and inappropriate use of diagnostic and statistical procedures. The most common sources of confusion may briefly be summarized:

a) Monosymptom-centred diagnoses in spite of the fact that the vast majority of neurotic patients show multiple symptomatology such as functional somatic (visceral) complaints, phobias, social deficits, depression, and obsessions-compulsions. Monosymptom-centred diagnoses do not give the essential information, whether they describe patients with an isolated monosymptomatic complaint, or whether for specific purposes one among several symptoms (problems) is highlighted as the only treatment-relevant disturbance. For many behaviour therapy studies with phobic, obsessive-compulsive or depressive patients it is therefore difficult to evaluate whether their results can be generalized to the respective diagnostic groups in psychiatry and (or) psychoanalysis, where they are operationalized in a much broader way.

b) Monosymptom-centred treatment for patients with a variety of symptoms and problems. Such studies are confusing, when complex disturbance is described on a narrative level, but test diagnostic assessment and treatment are administered to only one symptom area, wihout a rationale being given for this restricted intervention.

c) Publication of monosymptom-centred treatment when in fact "broad spectrum" treatment was delivered. It used to be popular to publish only the symptom-directed techniques and to devaluate the sometimes much more time consuming other interventions as "nonspecific". Thus, reality was construed according to availability of clear-cut measurements and interventions, but not necessarily according to real-life relevance for the patient. Fortunately, several "nonspecific" interventions have now become the centre of systematic research.

d) Description of multimodal, complex treatment packages with little attempt to specify the indications for the variety of interventions nor to prove their specific effects; inevitably there are then also no rules for a successive or concomitant application of the different treatment interventions.

e) Prevalence of group statistical data anaylsis although this can only be adequate for studies with *clients,* who are generally well adapted to life and homogeneous with regard to one particular, isolated complaint. In research with neurotic patients, the monosymptom-centred diagnosis collects a cohort of people with different sets of symptoms and other complaints, wrongly assuming homogeneity (data in section 2 of this paper). If such a cohort receives an identical treatment programme, necessarily some individuals will benefit, some will get worse and some will not change. Group data analysis will mask any specific effects—which otherwise post hoc might have helped to identify predictor variables. This has led to an increasing interest in single case methodology.

f) Sophisticated single case designs have not yet been able to create a breakthrough in everday research practice. Single case methodology (Kratochwill

1978; Petermann and Hehl 1979) has largely remained theory-oriented, still far too complicated for clinical research. Further, it has not yet produced results, which allow generalization from findings with one particular subject to a specifically defined group of subjects. If single case studies do not help to identify individual variable–configurations or symptom-configurations (syndromes) for a new and more appropriate "grouping" of subjects, inevitably they will fail to cope with the innumerable individual variables and their variations of interdependence.

1.3 Toward a Multisymptomatic Model of Neuroses– Strategy of Experimental Evaluation and Basic Assumptions

Much of the criticism regarding research in behaviour therapy of neuroses is based on the widely shared clinical "experience" that neurotic symptomatology is usually many-fold. As there is little experimental evidence to support this opinion (Foulds 1976), we shall first present some data from our own experimental investigations in this field (section 2). We have investigated patients with four different clinical diagnoses, made by experienced psychiatrists with a behavioural orientation: phobia, social inhibition, depression, and obsessive-compulsive disorder. For each of these clinical diagnostic groups we investigated the occurrence of one or more of the following symptoms and their possible combinations in the individual patients: phobias, social inhibition, depression, and obsessions-compulsions. Results are presented in such a sequence, that we describe first the variations of obsessive-compulsive symptomatology and then of additional neurotic symptomatology in the clinical group of obsessive-compulsive patients (2.2.1. and 2.). Then we show variations of neurotic symptomatology in the four clinical groups (2.2.2.). In our conclusions (2.2.3.) from these results an attempt is made to derive criteria for an assessment of varying degrees of neurotic disturbance within a four-symptom diagnostic system.

As the data confirm our hypotheses we proceed with the attempt to develop a functional, multisymptomatic model of neuroses, built by the 16 possible combinations of the 4 investigated symptoms (3.1.). Using this model, we try to derive possible causal symptom interactions from the different symptom combinations and their variations over time. The latter is tried by simulation of individual, multisymptomatic developments with data from only one assessment point (3.2). In this context we show the practical application and possible treatment-relevant consequences from our approach by re-grouping all 216 patients according to their individual ratings: (a) within the 16 stage model and (b) with regard to the main identified individual developments through different stages of the model.

Our criticism regarding attempts to reduce a complex reality to very few variables can also be applied to our own four-symptom model. But, we assume that the practicability of such models largely depends on a selection of variables which all belong to the same "class". It might be unhelpful to build models from a heterogeneous variety of variables like: one symptom, duration of illness, age and sex. The four symptoms in our investigation all belong to the same "class" of variables, e.g. they are indicators of neurotic disturbance. Few such symptoms and their varying combinations (syndromes) have been major components in the

development of complex theories of neuroses, e.g. in psychopathology and psychoanalysis. Of the symptoms described in the theories of "symptom-neuroses" our model includes all but hysterical and functional somatic symptoms. For the latter, at the time when we conducted our investigations no valid and reliable self-rating scale was available (of course, the same holds true for social inhibition and deficits, where we made a compromise using the respective scale of a "personality" inventory). For hysterical symptoms there is also no self-rating scale avaiable—but this kind of symptomatology seems to occur only rarely, be it also because it is now frequently diagnosed f.i. as phobic or obsessive-compulsive symptomatology.

Independently of any theory of neuroses, we simply use the symptoms in our model as indicators of varying degrees of neurotic disturbance—not as a means to explain the complex processes assumed to "underly" many of the varying symptom-constellations (syndromal approach to diagnosis Scadding 1980; Berner and Küfferle 1982). We do not regard our model a "final solution" to the problem. Rather, we want to present it as a new methodological approach to reduce the problem. A more "final" version of this model may include ratings from reliable and valid scales for functional somatic complaints and social inhibition/deficits, whereas it may exclude depression (thus remaining a four-symptom model), which is already included in our "Operationalized Multivariate Model of Motivation", OMMOM (Hand and Zaworka 1981a; Zaworka and Hand 1981a and b). We already have the first evidence for the clinical usefullness of the separate and particularly of the combined use of both models in treatment projects with agoraphobic and with obsessive-cumpulsive patients (Hand and Zaworka 1981b and c; Zaworka and Hand 1981b; Hand and Zaworka 1982).

2. Experimental Investigation of Variations of Symptomatology in Different Groups of Neuroses

A "Static" Group Statistical Approach

2.1. Methodology

2.1.1. Patients

The four self-rating scales were given to a population of "normals" ($n=120$) and to four groups of psychiatric "patients" ($n=216$).

"Normal" participants were recruited from dentists' offices, a sports-club, and from North Sea ferry-boats. The mean age was 35.7 (18–50) years; in family and social status they should be fairly representative of the total population; sex distribution was 72 females compared to 48 males.

The "patients" came from the two major psychiatric clinics in Hamburg (Psychiatrische Universitätsklinik Eppendorf and Psychiatrische Klinik des Allgemeinen Krankenhauses Ochsenzoll) and were distributed over the four diagnostic groups in the following way: obsessive-compulsive neuroses (OC): $n=121$ (m=56 : f=65; mean age 38.6 years, s=11.3; mean duration of illness 11.9 years, s= 8.3); agoraphobia (PH): $n=34$ (m=5 : f=29; mean age 37.9 years, s=9.4; mean duration of illness 10.3 years, s=9.3); social inhibition (SI): $n=31$ (m= 19 : f= 12; mean age 32.3 years, s=9.4; mean duration of symptoms 9.6 years, s= 6.9); depression (DE): $n=30$ (m=8 : f=22; mean age 39.3 years, s=11.7; mean

duration of symptoms 8.6 years, s=6.9). ANOVA revealed no significant differences between groups with regard to age and duration of symptomatology.

2.1.2 Self-Rating Scales for Symptom Variables

We selected self-rating scales for the investigated symptomatology, which are well validated either for English or German speaking populations. For phobias (ph) a slightly modified Fear Survey Schedule, FSS (Hallam and Hafner 1978; excluding their two factors for "general symptoms" and "social" phobias) was used; this questionnaire consists of 45 items, representing four phobia-factors (agora, blood/injurence and small animal phobias and a variety of other common situational phobias that constitute the fourth factor). This is the best validated phobia scale in the Anglo-American behaviour therapy literature, but so far no validation-study for a German speaking population has been done; we found a test-retest reliability (3 weeks interval) of 0.96. For social inhibition (si) the respective scale from the Freiburger Persönlichkeitsinventar FPI, by far the best validated personality inventory in German-speaking countries (Fahrenberg et al. 1973) was used; we found a test-retest reliability (3 weeks interval) of 0.85. For depression (de) the Wakefield Scale (Snaith 1971), applied widely in England was used; again there is no validation study for a German-speaking population, but for the purpose of international comparability of behaviour therapy studies we used this scale in all our treatment studies (we shall soon publish correlations between the ratings on the Wakefield Scale, FPI-Depression Scale and the Depression Scale by v. Zerssen); we found a test-retest reliability (3 weeks interval) 0f 0.86; the scale in our slightly modified version consists of 12 items, each of which has a four grade scale from no to high symptomatology. For obsessions and compulsions (oc) we used the Hamburg Obsessive-Compulsive Inventory, HOCI (Zaworka et. al. 1982, in press) which consists of the following 6 subscales: (A) checking, repeating and "ruminating after an action"; (B) washing and cleaning; (C) putting things into order; (D) counting, touching and talking; (E) "ruminating before an action", ruminating of specific sentences, words or images; (F) ruminations of harming oneself or others (Zaworka and Hand, 1980); this questionnaire consists of 188 items, each of them allowing a "yes" or "no" answer; we found a test-retest reliability of 0.93 for the whole test (for details of the validity studies Zaworka et al. 1982, in press).

We did not use "third party" symptom ratings: in behaviour therapy studies patients' assessments on self-rating scales for (neurotic) symptoms have frequently been shown to correlate well with therapists' and (or) blind assessors' ratings for the respective symptomatology. It should also be remembered, that in "symptom-neuroses" only the patients' subjective reality – regardless of therapists' or assessors' ratings – indicates reliably whether an individual will regard and present himself as ill and in need of professional help.

Psychophysiological variables are also excluded from this model, as in the vast majority of treatments for neurotic patients their assessment has not yet gained sufficient importance.

2.1.3. Data Analysis

In our data analyses we do not use the raw scores of the symptom scales. Instead, the ratings on each scale are indicated by a (+) or (–) symbol: A patients gets a (+)

for his ratings on a particular symptom scale when his score is above the mean score of the clinical reference group with that particular symptom; he gets a (–) when his score is below this mean (example: when a patient with the clinical diagnosis "obsessive-compulsive neurosis" has a (+) on the FSS, this means, he has higher phobia ratings than the average of all patients with the clinical diagnosis "phobia").

As two of the four scales (FSS and HOCI) are composed from items that represent independent factors within each scale, we had to decide whether to use the total score of a scale or the factor scores separately. On the HOCI, an individual already gets a (+) with above average rating in one factor. This appears justified, as the cut-off points for each factor are derived from investigations with a high *n* of obsessive-compulsive patients; also, from a clinical point of view, an obsessive-compulsive patient may well be severely handicapped in daily life by continuing involvement in obsessive-compulsive behaviour representing only one of the factors of the HOCI.

Ideally the same approach ought to be used for all symptom scales. For the Wakefield and the FPI-social inhibition this is not possible because there are no independent subscales. On the FSS we also had to use the total score because (a) we only had a relatively small *n* of phobics, which would have made it difficult to obtain reliable cut-off points for the subscales; and (b) for one of the four factors that we used – i.e. animal phobias – it appears complicated to derive a cut-off point even with a much larger *n,* as patients tend to have more isolated animal phobias within this factor rather than multiple ones.

With our definition of the cut-off point for a (+) rating on each scale we ensured that every such rating on any of the symptom scales really reflects severe symptomatology. The "clinical reference groups" for definition of the cut-off points were the patients of this study; we keep collecting data from our ongoing research to obtain more "definite" cut-off points for each scale. Current cut-off points: phobias = 60; social inhibition = 8; depression = 22; obsession-compulsions = 5.

The intercorrelations of the ratings on the four scales (of our total sample, $n = 337$) are between 0.20 and 0.40, only once reaching 0.47 (obsessions-compulsions and depression).

Comparability of Ratings on the Four Scales. A direct comparison of "high" and "low" ratings on each of the scales in the sense of "high" and "low" pathology appears problematic for simple methodological reasons: we did not investigate whether a (+) on one scale and a (–) on another scale are both rather close or far away from the cut-off point of their respective scale.

In spite of this source of error we tried to investigate experimentally whether this simple methodological approach leads to a more meaningful test-diagnostic supplement of clinical assessment and indication for specific therapeutic interventions.

A safeguard against major errors from a direct comparison of the scales is described in section 3, where we use three different cut-off points for the same rating of one patient in order to derive causal interactions of symptoms; operationalization of the criteria for the three different cut-off points is the same for each of the scales.

2.2 Results

2.2.1. Variations of Symptomatology in Obsessive-Compulsive Neurosis

2.2.1.1. Variations of Obsessive-Compulsive Symptomatology in Obsessive-Compulsive Neurosis. In two previous studies (Zaworka and Hand 1980; Hand and Zaworka 1981) we found that variations in quality and quantity of obsessive-compulsive symptomatology do to some extent allow differentiation of less and more disturbed individuals. Patients who had only isolated ruminations or isolated compulsions showed significantly lower subjective impairment by the symptoms and had received less inpatient treatment than those with multiple or combined obsessions or compulsions. Against the background of these results we investigated the frequency of one, two or more (+) factors of obsessive-compulsive symptomatology (Table 1), tested with the HOCI, and evaluated with the methodology described above.

Table 1 shows, which of the HOCI (+) factors tend to appear more often in isolation and which are more likely to be found in conjunction with others. It appears that factor B (washing and cleaning) is frequently an isolated (+) complaint and the least frequent factor in individuals suffering from five (+) factors at the same time. On the other hand, factor A (checking etc.) never appears as a mono-factor, but occurs most frequently in patients with three or more (+) factors. Table 2 confirms these results in a more detailed layout.

Apart from 14 (of 121) patients without any (+) ratings, only 19 of the remaining 107 patients show a mono (+) factorial symptomatology. Of these 19 10 are washers and cleaners (factor B), the remaining 9 are distributed over factors C

Table 1. Frequency of obsessive-compulsive factors in obsessive-compulsive patients

No. of factors with (+) ratings	Factors of the HOCI						*n* patients
	A	B	C	D	E	F	
1	0	10	3	2	1	3	19
2	5	11	13	8	15	12	32
3	11	3	6	8	11	6	15
4	11	7	8	7	9	6	12
5	15	9	15	10	15	11	15
6	14	14	14	14	14	14	14
Patients	56	54	59	49	65	52	107

(No (+) ratings in $n=14$)

Factors of the HOCI:
A = checking, repeating, ruminating after an action
B = washing, cleaning
C = putting things into order
D = counbting, touching, talking
E = ruminating before an action ruminating of specific sequences of thoughts, words or images
F = ruminations of harming oneself or others

Table 2. Mono-factor and multi-factor obsessions-compulsions (oc) in obsessive-compulsive patients

Factors of the HOCI						*n* patients
A	B	C	D	E	F	
No. oc						
−	−	−	−	−	−	14
Mono-factor oc						
+	−	−	−	−	−	0
−	+	−	−	−	−	10
−	−	+	−	−	−	3
−	−	−	+	−	−	2
−	−	−	−	+	−	1
−	−	−	−	−	+	3
Two-factor oc						
+	−	+	−	−	−	3
+	−	−	+	−	−	1
+	−	−	-	−	+	1
−	+	+	−	−	−	6
−	+	−	+	−	−	1
−	+	−	−	+	−	2
−	+	−	−	−	+	2
−	−	+	+	−	−	1
−	−	+	−	+	−	1
−	−	+	−	−	+	2
−	−	−	+	+	−	5
−	−	−	−	+	+	7

Factors of the HOCI						*n* patients
A	B	C	D	E	F	
Three-factor oc						
+	−	−	+	+	−	4
+	+	+	−	−	−	2
+	−	+	−	+	−	2
−	−	−	+	+	+	2
+	−	+	+	−	−	1
+	−	−	+	−	+	1
+	−	−	−	+	+	1
−	+	−	−	+	+	1
−	−	+	−	+	+	1
Four-factor oc						
+	−	+	+	+	−	3
+	+	+	−	−	+	2
+	−	−	+	+	+	2
+	+	+	+	−	−	1
+	+	+	−	+	−	1
+	+	−	+	+	−	1
+	+	−	−	+	+	1
−	+	+	−	+	+	1
Five-factor oc						
+	−	+	+	+	+	6
+	+	+	−	+	+	5
+	+	+	+	+	−	4
Six-factor oc						
+	+	+	+	+	+	14

to F. Two-factor (+) symptomatology consists particularly of the combinations B and C, D and E, and E and F. Three-factor to six-factor (+) symptomatology is much more equally distributed over all factors.

As already mentioned above, we do not interprete these results as static, but as symptom constellations representing only on a group statistical level the state of the patients at the time of assessment, and we are aware of the possibility of quite different ratings for the same patients at different times. For this reason also, we did not try to test the symptom configurations with a configuration-frequency analysis for their above or below chance occurrence.

2.2.1.2. Variations of Additional Neurotic Symptomatology in Obsessive-Compulsive Neurosis. In the next step we investigate whether obsessive-compulsive patients show differential combinations of other neurotic symptoms, which may also allow the assessment of varying degrees of pathology. The data layout of Tables 3 and 4 is similar to that of Tables 1 and 2.

Table 3 shows how many obsessive-compulsive patients have (+) ratings on one or more of the four neurotic symptoms simultaneously: phobias, social inhibition, depression, and obsessions-compulsions.

Table 3. Frequency of symptoms of neuroses in obsessive-compulsive (oc) patients

No. of symptoms	Kind of symptoms				*n* patients
	ph	si	de	oc	
1	0	3	0	30	33
2	6	9	19	34	34
3	9	22	25	28	28
4	15	15	15	15	15
Patients	30	46	59	107	110

Symptoms: ph = phobias
si = social inhibition
de = depression
oc = obsessions-compulsions

Table 4. Combinations of symptoms of neuroses in obsessive-compulsive (oc) patients

Types of combinations of symptoms	Kind of symptoms				*n* patients	
	ph	si	de	oc		
1	−	−	−	−	11	0-fold
2	+	−	−	−	0	
3	−	+	−	−	3	1-fold
4	−	−	+	−	0	
5	−	−	−	+	30	
6	+	+	−	−	0	
7	+	−	+	−	0	
8	+	−	−	+	6	2-fold
9	−	+	+	−	0	
10	−	+	−	+	9	
11	−	−	+	+	19	
12	+	+	+	−	0	
13	+	+	−	+	3	3-fold
14	+	−	+	+	6	
15	−	+	+	+	19	
16	+	+	+	+	15	4-fold
				Patients	121	

Symptoms: ph = phobias
si = social inhibition
de = depression
oc = obsessions-compulsions

Of the 121 patients 11 show no (+) rating on any of the symptom scales. Of the remaining 110, 30 (27%) have a (+) only in obessions-compulsions, another 3 only in social inhibition (i.e., the latter got a "wrong" clinical diagnosis). Together with

Table 3, Table 4 shows that the two-symptom (+) combination preferably consists of obsessions-compulsions and depression (twice as frequent as the combination of obsessions-compulsions and phobias). In the three-symptom (+) combination the additional symptom in most patients is social inhibition.

Contrary to expectation, phobias in obsessive-compulsive patients are the least frequent (+) symptomatology. As approximately 70% of the patients have a two-fold or more (+) (i.e. severe) symptomatology, the question arises, which additional criteria led the clinicians to make the diagnosis of obsessive-compulsive neurosis?

2.2.2. Variations of Neurotic Symptomatology in Different Groups of Neuroses and Normals

We now extend our investigations from the group of obsessive-compulsive patients to three additional groups of neurotic patients: phobics (PH), socially inhibited (SI) persons, and depressives (DE). These four groups are compared with the sample of the normal population. Table 5 shows how frequently individuals of any of the five groups show any of the four symptoms or any combination of them on the (+) level.

Normals are largely confirmed as "normal," 91 (75.8%) of the 120 show no (+) rating in any of the symptom scale, though 21 show (+) ratings on one scale – 50% of these in obsessions-compulsions. This may confirm that obsessions-compulsions not only occur as "symptoms," but for a number of people may be effective coping or adaptation strategies (Hand 1982).

No (+) ratings in any of the symptom scales are shown by 32.2% of the phobics, 22.6% of the socially inhibited, 10% of the depressive, and 9.1% of the obsessive-compulsive patients. These data may also be interpreted as supportive for an approach building a "hierarchy of pathology" including as one of the variables the kind of neurotic symptomatology.

Only between 10% and 25% of the neurotic patients show a monosymptomatic complaint in accordance with the clinical diagnosis. This stands in sharp contrast to the everyday use of monosymptomatic diagnosis for these patients in behaviour therapy.

On the other hand, approximately 45% of the phobics and between 65% to 80% of the patients of the other diagnostic groups have multiple (+) symptomatology. Again, this cannot at all be inferred from clinical monosymptom oriented diagnosis.

2.3. Conclusions

This series of studies (Tables 1 to 5) confirms some of the assumptions that have led us to look for a new and more treatment-relevant diagnostic model for neuroses:

1. Most neurotic patients with one of the usual monosymptom diagnoses actually show multiple (+) symptomatology. With only 10% to 25% of these patients showing one (+) rating and 45% to 80% suffering from multiple (+) symptomatology, it remains an open question why and how (behaviour) therapists select one of the symptoms for the diagnostic labelling.

Table 5. Combinations of symptoms of neuroses in four different groups of patients and in normals

Types of combinations of symptoms					Clinical diagnosis				*n* patients	*N*
					ph	si	de	oc		
1	–	–	–	–	11	7	3	11	32	91
2	+	–	–	–	[8]	0	0	0	8	1
3	–	+	–	–	0	[4]	1	3	8	1
4	–	–	+	–	0	1	[3]	0	4	4
5	–	–	–	+	2	3	4	[30]	39	15
6	+	+	–	–	[3]	[0]	0	0	3	0
7	+	–	+	–	[0]	0	[0]	0	0	0
8	+	–	–	+	[1]	0	0	[6]	7	0
9	–	+	+	–	0	[3]	[1]	0	4	0
10	–	+	–	+	0	[5]	1	[9]	15	1
11	–	–	+	+	2	0	[9]	[19]	30	3
12	+	+	+	–	[0]	[0]	[0]	0	0	0
13	+	+	–	+	[3]	[0]	1	[3]	7	0
14	+	–	+	+	[1]	1	[1]	[6]	9	0
15	–	+	+	+	0	[5]	[2]	[19]	26	0
16	+	+	+	+	[3]	[2]	[4]	[15]	24	0
					34	31	30	121	216	121

Clinical diagnoses: PH = phobia
SI = social inhibition
DE = depression
OC = obsessions-compulsions
n = normals
□ = clinical and test-diagnosis are identical

2. It appears possible to obtain important information regarding an individual's position on a continuum from no to low to high "pathology" – or, in our terminology, from normal via client to patient – merely by assessing the quality (kind), quantity (number) and the combinations of symptoms. Within obsessive-compulsive patients, some factors (like "washing") indicate lower probability of additional obsessive-compulsive or other symptomatology, whereas others (like "checking") indicate a rather high probability of additional symptomatology (2.2.1.1.). There is also some evidence, that the four clinical diagnostic groups can be placed on the low to high pathology continuum with the following sequence: phobias, social inhibition, obsessions-compulsions and (or) depression – the latter two being difficult to differentiate (2.2.2.). More data to support these results from a group hierarchical approach will be published in the future.

With regard to behaviour therapy research, one example may illustrate the consequences of such a broader assessment approach. Our data show that studies with mono (+) obsessive-compulsive patients are representative for only 25% of

the total sample with the clinical diagnosis of obsessive-compulsive neurosis. Studies which include those obsessive-compulsive patients with (+) ratings on phobias and social inhibition, but exclude those with (+) depression still are only representative of 50% of this group of patients. Unfortunately, those studies seem to dominate the literature. Yet, both make a clear selection toward lower pathology than the one characteristic for this patient population. While they have helped to improve specific symptom-directed treatment techniques, they have also created confusion between researchers and practitioners with regard to the generalization of their results. "Washers," for instance, may much more resemble phobic than obsessive-compulsive patients. The particularly good results of exposure treatments for "washers" – as opposed to the much less encouraging results with "checkers" or "ruminaters" – may thus be due to the similarity to phobics, and phobics may respond so well to treatment because of their relatively low pathology as compared to the other clinical diagnostic groups of our study.

It follows that for treatment purposes diagnostic groups of neurotic patients should be built on the horizontal (one to more symptom combinations) rather than the vertical (clinical diagnoses I–IV) level of Table 5. Grouping under one of the clinical diagnoses gives the misleading impression that individuals with no (+) and mulitple (+) symptomatology do build homogeneous diagnostic groups. It appears much more useful to group individuals according to their combinations of (+) and (–) ratings, e.g. according to the different "positions" in Table 5. Table 5, derived from a group statistical approach, already allows single case assessments. It does not allow conclusions about the varying interdependence and possible causal interactions between symptoms. It also does not allow conclusions about individual developments over time. An attempt in this direction will be made in the following section.

3. Simulation of Individual Variations of Symptom-Configurations Over Time

A "Dynamic," Single Case Approach

So far we have only looked at our data in the "classical" static way: e.g. pretending the patient remains stable in the symptom state which has been diagnosed (indirect trait orientation of the usual symptom – i.e. state-diagnosis). In a next step, we try to simulate individual development of symptom-configurations over the course of time (with experimental evaluation by observation of "real" developments being under way). For this purpose, we first introduce a model of all possible 16 combinations of the (+) and (–) ratings on the four symptom scales and their interdependencies. As this model is also supposed to reflect developments of individuals over time, these positions will be called "stages" from now on.

3.1. An Operationalized, Multisymptomatic Model of Neuroses (OMMON)

Figure 1 shows the 16 theoretically possible stages built by the (+) and (–) ratings on the four symptom scales.

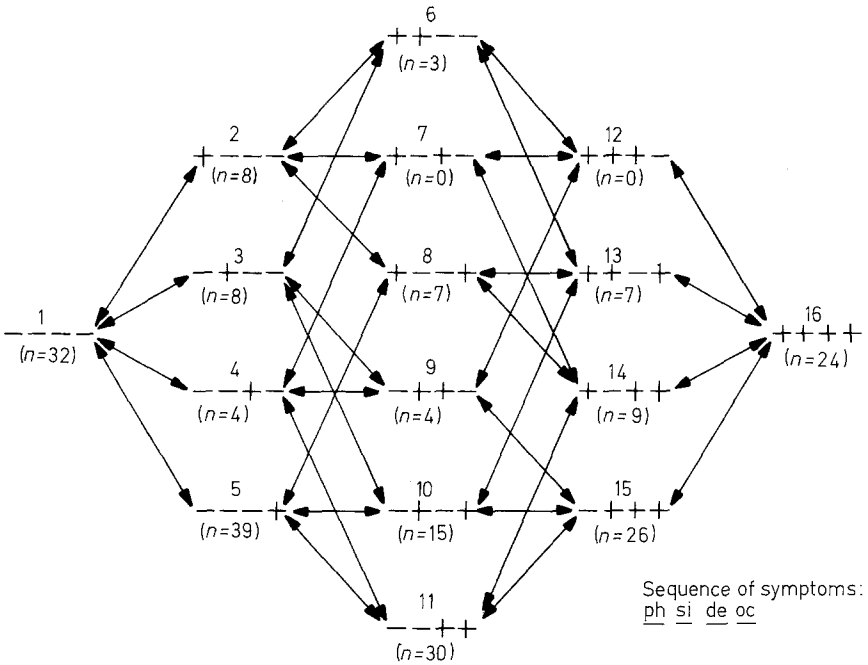

Fig. 1. An Operationalized, Multisymptomatic Model of Neuroses (OMMON) (Frequency distribution of 216 patients with neuroses over the 16 stages of the OMMON)

From left to right the ratings represent monosymptom to four-symptom stages. This can be interpreted as an additive model from no or low pathology in stage 1 to high pathology (c.f. 2.1.1.3.) in stage 16.

The arrows indicate the theoretically possible pathways between the stages – in both directions – under the assumption that only one change can occur at a time. The duration of such a change cannot be specified and may range from extremely short (seconds) to long periods (years) of time (Zaworka and Hand 1981a). A separation of almost concomitant changes of two or more variables may seem clinically irrelevant – but it is a necessary prerequisite for the development of the logic of the model. The clinical usefulness of the model itself should not be judged from this underlying assumption, but from results of experimental application of the model in clinical research.

3.2. Simulation of Individual Developments Over Time Within OMMON

In the following we shall try to simulate in three steps individual developments of "neurotic" symptomatology over time (through OMMON), with data from a single assessment occasion only.

3.2.1. Simulation of Individual Routes Through OMMON: A Group Statistical Approach

In the first step we transpose our experimental results (except for the group of normals) from Table 5 into OMMON (Fig. 1). Several "routes" through the

model can easily be identified from the number of patients in each stage. No routes seem to pass through stages 7 and 12. The *main route* with our patient sample is characterized by the following stages: start at stage 1 with no (+) symptomatology; stage 5 with mono (+) obsessive-compulsive complaints; stage 11 with additional (+) depression; stage 15 with additional (+) social inhibition; and finally, stage 16 with additional (+) phobias. This could be regarded as a characteristic route for many obsessive-compulsive developments, which start with obsessive-compulsive symptomatology. But, as the model shows, once stage 1 is left, there are always several routes for people having started with different (+) symptomatology, to move into the "obsessive-compulsive development" and vice versa.

3.2.2. Simulation of Individual Developments Within OMMON: A Single Case Approach with Varying Cut-Off Point Assessment (VACOPA)

In the second step, we try to derive hypotheses regarding individual developments of multisymptomatic neuroses by single-case application of a newly introduced variation of our data analysis with dichotomized scales. Whereas up to now (+) or (–) ratings for each of the symptom scales were defined by higher or lower ratings than $\bar{x}$ of the specific reference groups, from now on each patient's ratings are assessed with three different cut-off points, with varying difficulty to reach (+) ratings. This "Varying Cut-Off Point Assessment" (VACOPA) uses the cut-off points "$\bar{x} + \frac{1}{2}$ SD," "$\bar{x}$," and "$\bar{x} - \frac{1}{2}$ SD" of each clinical reference group (all the following varying cut-off point assessments will be made with this sequence, i.e. from the high to the low cut-off point). So every individual gets three "diagnoses" in the 16 stage model. Two examples may illustrate this procedure (Table 6):

Example 1 shows a person with a high phobia rating ((+) in "$\bar{x} + \frac{1}{2}$ SD), medium social inhibition ((+) in "$\bar{x}$") but no or very low depression and obsessions-compulsions (not even a (+) in "$\bar{x} - \frac{1}{2}$ SD"). This pattern, translated into the stages of the model, would give the rating 2.2.6. Example 2 shows a person with high obsessions-compulsions, medium depression, low social inhibition, and very low or no phobias (5.11.15.).

It must be remembered that "medium," "low," "very low" and "no" symptomatology in this context is operationalized by dividing each scale into four segments built by the three cut-off points. Comparison of ratings on different

Table 6. Simulation of individual courses through stages of OMMON (with varying cut-off point procedure)

Example 1						Example 2					
Cut-off point	ph	si	de	oc	Stage	Cut-off point	ph	si	de	oc	Stage
$\bar{x} + \frac{1}{2}$ SD	+	–	–	–	2	$\bar{x} + \frac{1}{2}$ SD	–	–	–	+	5
$\bar{x}$	+	+	–	–	6	$\bar{x}$	–	–	+	+	11
$\bar{x} - \frac{1}{2}$ SD	+	+	–	–	6	$\bar{x} - \frac{1}{2}$ SD	–	+	+	+	15
Individual course 2.6.6						Individual course 5.11.15					

scales as well as changes of ratings within one scale therefore is a comparison of identically defined segments of each scale. Although a direct comparison of scales is problematic for test theoretical reasons, for the purpose of an operationalization of clinical judgment of "pathology," comparison with the described procedure appears useful.

Generally, the three positions of an individual in the model can be identical or different: (a) he may remain in one stage in all three ratings, indicating stable monosymptomatic (5.5.5.) or stable multisymptomatic (15.15.15.) pathology. If we exclude all 1.1.1. persons ($n = 6$), then 18% of our whole sample falls into this group; (b) he may fall somewhere in between two stages, again indicating either lower (1.1.5.) or higher (15.15.16.) pathology, 43.8% of the total sample follows this "route"; (c) he may fall into entirely different stages, which happens for 14.3% of the whole sample. If a patient gets three different ratings, these may resemble a continuous sequence of steps through the model, again either on a lower (1.5.11.) or higher (11.15.16.) level of pathology; (d) finally, he may omit two or more stages: d.1) moving from no/very low to higher pathology (1.11.11.) or d.2) from preexisting to even higher pathology (11.11.16.). The first route is taken by 12.9%, the second by 11% of our total sample.

All these stages in the model derived from one single assessment occasions do not allow safe predictions regarding the direction of possible developments (routes) through OMMON (i.e. whether they will go to the left or to the right, out of a given stage in the model).

Such information can be derived from further assessments either during a longer base-line or through treatment and follow-up. Example: A first assessment of 5.5.11. followed by a second assessment of 5.11.11. clearly indicates a move towards stage 11, i.e. increase of pathology; on the other hand, a second rating of 5.5.5. would indicate reduction of pathology, i.e. improvement. With a "one cut-off point assessment" ("$\bar{x}$"), the first direction of change would also be detected 5.→11.), whereas the second direction of change would not be identified (5.→5.).

3.2.2.1. Prediction of Individual Direction of Change Within OMMON, with Assessment of Causal Symptom Interactions. In spite of the described restrictions, analysis of data (from a single assessment) with VACOPA may help to derive first hypotheses: (A) regarding the likelihood of change into either left or right direction in the model (resulting in prognosis of development), (B) regarding unidirectional or reciprocal causal interactions between symptoms (resulting in specific indications for interventions). These hypotheses can only be made in an individual for the time of assessment and not with regard to long-term symptom development, as we assume changing symptom configurations and interactions over time, which can only be detected with retests.

For hypotheses in both areas we need some basic assumptions, in addition to the logic inherent in OMMON: (Ad A) We generally assume that there is a development from left to right in the model – unless there is evidence for the opposite, from variables outside the model. To account for the latter, the patient is asked the simple question: Over the past few weeks, has your condition: improved – not changed – deteriorated? (subjective improvement scale). The therapists'

negative assumption for a development towards more pathology is only changed when a patient rates "improved" (the additional possibilities resulting from concomitant application of our Behavioral Resistance Scale, BRS, and of OMMOM cannot be described here). The hypothesis derived from such a first, single assessment is checked by the results of a retest (usually after 4 to 24 weeks), immediately before treatment; (Ad B) Additional assumptions for derivation of unidirectional or reciprocal causal symptom interactions: (1) A (+) rating on one scale above a higher cut-off point than a (+) rating on another scale excludes the possibility of the "higher" symptom being causally influenced by the lower symptom. The same holds true for two or three equally high (+) ratings in relationship to one lower (+) rating. The lower ratings are either independent of or causally influenced by the higher ratings; (2) Two or more equally high (+) ratings either reflect a reciprocal, causal interdependence or independence.

Clinical illustration: on his first assessment a patient shows (+) ratings on three symptom scales, but only above the lowest cut-off point. At this time, all three "low" symptoms could be caused by the same "underlying conflict," yet be independent from each other. If, at a much later retest, the same symptom scales show (+) ratings above the medium or even high cut-off point, then one could assume reciprocal causal interdependence of the symptoms: after "disconnection" from or disappearance of an original "underlying" cause, the symptom behaviours themselves may to a considerable extent have caused mutual increase in severity ("vicious circle" model of symptom chronicity). Both differently interpreted constellations at the different points in time have the same therapeutic consequence: no single-symptom treatment at this point in time.

Any theoretically possible unidirectional causal connection between two equally high (+) ratings could only be detected in subsequent tests, provided the rating in one scale has changed by then. This then would have immediate therapeutic consequences.

Because of the shortage of space we have to leave it to the reader to construe clinical hypotheses, applying the described assumptions to the "routes" a to d outlined earlier in the paper.

3.2.3. Evaluation of a Main Individual Route Through OMMON: A Group Statistical Approach, Based on Single-Case VACOPA Results

With regard to individual developments over time, so far we have tried two different ways of deriving hypotheses from a single assessment occasion only. In a first step (section 3.1.) we hypothesized individual development through OMMOM from a group statistical approach. Using the medium ($\underline{x}$) cut-off point, patients were re-grouped according to their stages in OMMON, and e.g. the "main individual route" through OMMON was derived from the most frequently "occupied", neighbouring stages.

In a second step (section 3.2) with a single case approach, we introduced our extended methodological "technology" VACOPA, assumed to simulate hypothetically the course of time with regard to individual symptom changes. Also, VACOPA results led to an operationalization of two typical "intermittent" stages between each pair of neighbouring "main" stages of the "main route" of the model.

In a third step, we returned to a group statistical level, and investigated how many of these "intermittent" stages (step 2) of the "main route" (step 1) were actually occupied by one or more patients. As we found all of them occupied by at least one patient, this–having moved from a "macroscope" towards a "microscope" group analysis–lends strong support for the "real" existance of the simulated, hypothesized individual main route (details from this analysis will be published later, together with the experimental evaluation of "real" individual developments over time).

4. Discussion

With OMMON we hope to contribute to the development of treatment-relevant assessment procedures in psychotherapy. Our model is meant to comprise "neurotic" symptomatology from "normal" via moderately to severely "disturbed". Our results seem to confirm the clinical knowledge that "... certain symptom constellations are much more effective for diagnostic assignment, prognosis and therapy than single symptoms" (recent discussion in Berner and Küfferle 1982). With the model it also seems possible to improve assessment and (or) prediction of the likelihood of spontaneous improvement versus impending danger of increasing or chronic disturbance.

Accordingly, we suggest operationalization of the ill-defined terms "normal", "client", and "patient" without referecne to theories or ideologies–merely for the assessment of the degree of need for (psycho) professional help or indication for psychotherapy respectively.

More specifically, OMMON seems to offer a number of possibilities in narrowing the current gap between diagnosis and treatment, as well as between behaviour therapy/psychotherapy research and practice–particularly with regard to the following questions:

(1) Effects of monosymptom directed interventions on the respective symptom, and possible generalizations to other symptom and problem areas.

(2) Effects of "unspecific" interventions on specific symptoms.

(3) Indication and application of specific interventions within broad spectrum treatment packages, and specification of optimal sequences of application of the single ingredients of such packages.

(4) Operationalization of the concept of "symptom-substitution", which so far has been ill-defined in psychoanalytic literature, and has mainly been a semantic problem among psychotherapists of different orientation.

For these problem areas (1.-4.) we have already presented first data from treatment trials with agoraphobic and obsessive-compulsive patients (Hand and Zaworka 1981b, c and 1982).

(5) Effects of life-events and life-distress on symptoms and symptom configurations.

(6) Comparative evaluation of physiological (Ost et al. 1981) and psychological symptom response patterns to acute and chronic distress.

(7) Further evaluation of a hierarchical, developmental model of neurotic

symptomatology and its variations in neurotic, psychosomatic and psychotic patients (Foulds 1976).

(8) Besides all these intraindividual assessments, indirect assessment of interpersonal processes may be tried by also administering the questionnaires to the spouses of patients. If the concept of "neurotic" relationship is practically meaningful, changes in the patient's symptom-configurations should be accompanied by respective changes in the ratings of the spouses (such studies are also under way, with relatives of neurotic and schizophrenic patients).

The clinical usefulnes of OMMON can be improved, when it is used together with OMMOM (1.3.).

So far, we have mainly emphasized the use of the model in behaviour therapy research. We do not see any limitations against using the model in the evaluation of outcome of psychoanalytic or other modes of psychotherapy. Three open methodological issues have to be remembered for further work with the model:

(1) It may be necessary to include functional somatic complaints as one of the symptom scales into the model, and exclude depression (1.3.).

(2) On methodological grounds it seems impossible to draw conclusions from one single assessment about the development and the interdependence of variables over time. Using VACOPA and with the logic of the model, we nevertheless seem to be able to formulate rather specific and practically meaningful prognoses regarding a "better or worse" development from a patient's single rating (which of course ought to be tested by repeated assessment).

(3) It may be advantageous to define the cut-off points by quartiles of the scales, rather than by means and standard deviations (Baumann, personal communication 1982).

For the reader it may be interesting, to compare the methodological logic of our model with that applied by Wottawa (1981) in a purely mathematical-statistical approach to the problem. Although his model does not specify the variables and emphasizes mathematical-statistical procedures to extract those few variables which are representative of a larger sample of variables—this is in contrast to our use of "clinical logic" for identification of the "representative" variables—the basic reasoning for the model is in several ways similar to ours. He calls for "heuristic methods on the basis of logically structured combinations of the variables". Interestingly, both models were developed concomitantly, without the respective authors knowledge of the other.

If future results from the application of OMMON in psychotherapy research can confirm our assumptions, then—last but not least—this should also help to improve communication between the providers of psychotherapy and the "agents" (government agencies and insurance companies) and between them and the consumers of psychotherapy. The newly developed and relatively restrictive prerequisites for future psychotherapy funding by government grants and reimbursement by insurance companies in the USA (London and Klerman 1982; Parloff 1982) indicate the increased pressure to provide evidence that psychotherapy does provide *real* help for people with *real* health problems.

We would like to encourage the reader to ruminate about realizations of the theoretical consequences of this model and its therapeutic implications, which in this paper could only be tentatively indicated.

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