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Out-patient, multi-modal behaviour therapy for obsessive–compulsive disorder

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Background With regard to long-term outcome, behaviour therapy is the first choice treatment for obsessive–compulsive disorder (OCD), with or without concomitant selective serotonin reuptake inhibitor (SSRI) medication. Yet, results from research trials, usually restricted to exposure with response-preventions and other symptom-directed techniques, may not be generalisable to people with OCD in community health care services.

Method For more than 20 years we have delivered out-patient behaviour therapy to unselected people with OCD from the community, including those with motivational and compliance problems. Prospective–retrospective follow-ups were carried out from 1–13 years after treatment.

Results This paper describes the applied multi-modal, strategic–systemic behaviour therapy, and our partially new model of OCD. Compliant patients achieved the usual success rate of 65–70%, but this drops to 50% if all patients treated are included in the analysis.

Conclusions For major subgroups of OCD, behaviour therapy is a very effective treatment modality. Exposure is essential, but additional ('causal') interventions are equally important in about half of the patients from unselected samples. Both behaviour therapy and drug-treatments need to be improved and predictive variables for outcome of either of them are urgently needed.

The term obsessive–compulsive disorder (OCD) encompasses a group of syndromes with variable symptom configurations serving a variety of different intra individual and interactional functions. The factors that cause and maintain its symptom expression over the life span range from genetically based vulnerabilities to a variety of psychosocial variables (educational styles in family and school, socialisation among peers, psychological traumata and culture- and society-bound norms). In at least half of people with OCD, one or more of the following problems or disorders arise before the first onset of OCD: increased proneness to anxiety-responses; low self-esteem; social deficits and social phobia; and latent aggression.

Behaviour therapy for many years has been regarded as the first choice treatment, not only for symptom-specific interventions but also for the modification of a variety of 'underlying causes' and maintaining variables from comorbidity, problems in daily life conduct, interpersonal deficits, to other risk-factors. In at least half of people with OCD, individualised, multi-modal behaviour therapy is required. Long-term outcome studies show success rates between 50–80%, the differences presumably depending on: pre-treatment selection criteria (regarding symptomatology, comorbidity and motivation for change); type and intensity of behaviour therapy; developmental deficits before onset of OCD; actual comorbidity; professional and private life situation; and operationalisation of 'success'. Whether those who do not respond in behaviour therapy will respond to drug treatment – and vice-versa – is not yet known. A relapse rate of around 70–80% after discontinuation of medication (Pato *et al*, 1988) is likely to be reduced by concomitant or immediately ensuing behaviour therapy (van Balkom *et al*, 1994).

MULTI-MODAL, STRATEGIC-SYSTEMIC BEHAVIOUR THERAPY

As in any 'treatment school', in behaviour therapy the development of concepts in research and in clinical practice did not always evolve together. The dominance of the early approach of behaviourism, which concentrated on the motor aspect of behaviour, has lately been challenged by cognitivism in cognitive therapy and cognitive–behaviour therapy – with both behaviourist and cognitivist approaches restricting 'behaviour' to only one aspect of behavioural complexity. The main question is: what is the most effective way of inducing changes in human behaviour? Do experiments with motor-behaviour lead to new emotional experiences which then induce changes in cognitive constructs and expectations? Or do cognitive interventions directly tackle maladaptive cognitive constructs and thus lead to changes in motor behaviour and emotions? This dispute sometimes seems to be more of a religious than of a scientific nature.

Clinical behaviour therapy over the past two decades has been mainly concerned with further refinement of early multi-modal behaviour therapy (Lazarus, 1976). Our own contribution to this has been the development of a strategic-systemic, multi-modal behaviour therapy (Hand, 1986).

The main contents of strategic-systemic, multi-modal behaviour therapy are:

(a) Behavioural, causal, functional, motivational and systemic analyses, including the analyses of intra-individual versus interactional functions of (illness-) behaviour. In this context, behaviour is regarded as a result of complex interactions of motor-behaviour, cognition, emotion and physiology.

(b) Concepts and techniques of systems-oriented treatment schools (e.g. Friesen, 1985), elements of which can be integrated into the strategy of behaviour therapy.

(c) Derivation of a causal–hierachical order based on symptom behaviour, comorbidity, 'other problems in life' etc. to build a first clinical hypothesis. The multi-modal treatment plan is derived from this hypothesis and not from DSM or ICD diagnoses.

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Table I Problem areas, kind of disturbance and interventions in obsessive–compulsive disorder

Problem area	Kind of disturbance	Intervention	Aim of intervention
Behavioural disorders	Symptom behaviours: Qualitatively new, 'non-normal' behaviour Excessive behaviours: Excessive 'normal' behaviours: eating, cleaning, gambling	Reduction of behaviour	Establishment of freedom for action
Behavioural deficits	Deficits in basic behaviours: Primary or secondary deficits in social competence; problem-solving strategies; distress-coping	Establishment of behaviour	Establishment of competence for action
Blockage of perception	Paratactic perception: Stereotype misperception of others Collusion: Complementary dyadic interdependence → 'Interaction–Personality'	Historical orientation: induction of transference-neurosis 'Here and now' orientation: communication training	Establishment of freedom for perception
Blockage of action	Conflict–ambivalence: Partly subconscious 'intention' for illness rather than alternative behaviours. Common cause: conflict between emotional needs and rational beliefs	Systemic functional analysis (behaviour therapy); Provocative prescription (family therapy)	Establishment of motivation for action

(d) The multi-modal treatment package may consist of specific techniques regarding the following areas of disturbance: (a) symptom-directed techniques to reduce pathological, maladaptive behaviour; (b) deficit-directed procedures to built up behaviours that are necessary for daily life conduct (social skills, problem-solving skills, self-induced alternative behaviours); (c) interventions for improvement of social perception; (d) interventions to reduce ambivalence and to increase motivation for change.

For OCD patients with differing core pathologies, treatment plans based on these analyses may be quite different: either symptom-directed interventions or interventions to reduce deficits and to improve skills; a combination of the first and the second approach; any other combination of interventions depending on the areas of functional disturbance present in the individual patient (see Table 1).

To give an example: a patient with checking and cleaning compulsions already had severe behavioural deficits (in social skills and problem-solving) before first onset of OCD; the behavioural freedom gained from symptom-reduction via symptom-oriented techniques will probably not be utilised for improved social interactions because of a lack of behavioural competence; symptomatic improvement in compulsions may result in painful re-exposure to social situations, leading either to a relapse in the original OCD or to the development of depression or other symptomatology. In such patients, symptom-specific interventions need to be preceded or at least accompanied or followed by competence training.

This is not to say, that in such a case social skills training would by itself lead to a significant reduction of obsessive–compulsive symptomatology. In severe cases, complex combinations of multi-modal interventions are often needed, including spouse or family interventions in addition to individual or group treatment (for a detailed description of the treatment model and its application see Hand, 1988, 1993*a*).

Prior to making the first, tentative treatment plan, it is helpful to check whether the following assessments have already been made from the information gained in the first few sessions with the patient:

(a) internal or external motivation for treatment and change;

(b) directly communicated or indirectly deducible problem areas other than the primary symptom complaints;

(c) delineation of causal and maintaining variables for symptom behaviours and problem areas, and their interactions in the past and at present time (biographical behavioural analyses);

(d) differentiation of intra-individual and interpersonal functions of the identified symptom behaviours and problem areas;

(e) consequences to be expected from reduction (removal) of symptoms – both the patient's and therapist's account;

(f) microanalysis of symptom behaviours (including diagnostic test assessments);

(g) symptom responses or other specific responses to stressors in earlier bio-social developmental stages (puberty, leaving parental household, leaving school, first intense dyadic relationship etc.);

(h) social competence (in groups as well as in dyadic relationships) and emotional expressiveness (e.g. actual patient–therapist interaction);

(i) intensity and quality of self-induced 'alternative' behaviours (to substitute illness-behaviour), in the past and at present.

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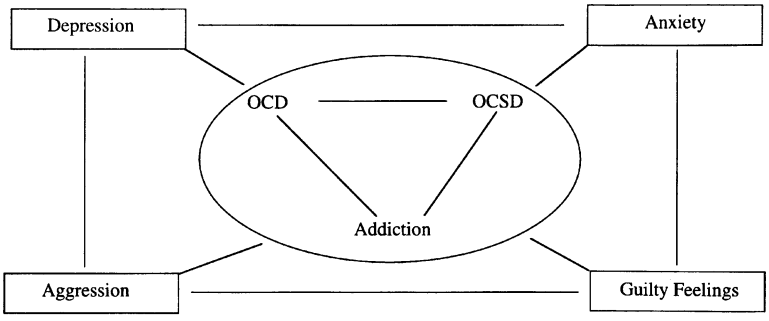


Fig. 1 Negative state and behavioural consequences. OCD, obsessive–compulsive disorder; OCSD, obsessive–compulsive spectrum disorder.

OBSESSIVE–COMPULSIVE DISORDER

Obsessive–compulsive disorder – pseudo-coping with negative emotions or archaic response to overstimulation?

Usually, OCD is classified as an illness entity. It is assumed that impulses for obsessions and compulsion are 'ego-dystonic', not at all wanted, and that the sufferer is simply unable to withstand them. A similar concept is applied to obsessive–compulsive spectrum disorders (OCSD), some of which are also labelled as 'impulse control disorders' or 'drug-independent addictions'. There is also a popular debate as to whether OCD and OCSD are 'behavioural addictions' or vice versa.

In this discussion, one of the key assumptions is that obsessive–compulsive activities – including OCD, OCSD and subtypes of alcohol and drug abuse – subjectively serve to avoid or escape negative or painful (emotional–cognitive–physiological) states (Gazzaniga, 1992). This is a theory of negative reinforcement, where obsessive–compulsive and obsessive–compulsive spectrum activities are supposed to reduce the subjective experience of negative states like depression, anxiety or guilty feelings, regardless of their causes. Positive reinforcement hardly occurs, in contrast to 'normal' cleaning or gambling behaviour, where the activity itself or its outcome may be satisfactory or joyful. In our research into OCD, pathological gambling, pathological shopping, trichotillomania and bulimia we follow such a model. Therefore, patients with similar core-problem constellations across these diagnostic groups may receive almost identical interventions, whereas patients with the same phenomenological symptomatology may receive quite different multi-modal treatment packages. The hypothetical interactions between obsessive–compulsive spectrum behaviours and negative emotions are shown in Fig. 1.

OCD and OCSD could also be characterised as 'behavioural excess disorders'. Apparently normal, everyday behaviours are exaggerated far beyond social norms, while other necessary complex daily life activities are extremely reduced, in order to avoid their painful elements (detailed description in Hand, 1997, 1998).

It seems relevant to broaden this concept by describing some recent research in the brain mechanisms involved in stereotyped behaviours in humans and animals. Development of ritualised and stereotyped modes of thinking and acting is regarded as a prerequisite for the development of psychological health and social adaptation in humans (Erikson, 1978). It seems to enable a person to cope with the continuous influx of environmental stimuli without responding as if they were entirely new every time. Ritualisation does help to economise cognitive functioning and decision-making. Psychosocial health may be the result of an adaptive balance between flexibility and rigidity in the process of establishing the behaviour patterns of individuals, as well as in groups and in organisations.

From a more biological point of view, excessive, maladaptive behavioural ritualisation occurs, when the 'orbital cortico-basal ganglionic-thalamic circuit' in the brain is pathologically triggered. This archaic, primitive response then gains control over more adaptive, inhibitory behavioural response patterns (for a comprehensive review of this research in humans and animals see Baxter *et al*, 1996). One easy way to trigger such responses in lizards is to put two individuals into a territorial conflict situation. Does this not resemble the striking tendency of many people with early and chronic social deficits and social anxiety to develop OCD or OCSD? About 50% of the subjects with OCD in our studies show this comorbidity, often associated with secondary depression. This type of person with OCD will exhibit an acute increase in obsessive–compulsive behaviour when approached too closely by others, or he or she will try to stop them by pseudo-assertive aggression. The application of such a model will be shown below.

Treatment-relevant functions of obsessions and compulsions

In many people with OCD and their relatives long-term success from short-term behaviour therapy may only be achieved by elaborating and changing the intra-individual as well as the interactional functions of obsessive–compulsive behaviours. Nevertheless, quite a few of the patients and relatives, after having contributed to identifying these functions, refuse to co-operate in changing them and only accept symptom-directed techniques. In those cases, profound changes in the interactional function can sometimes be achieved when symptom-treatment is used as hidden couple therapy (Hand *et al*, 1977).

Some frequent intra-individual and interpersonal functions of OCD are summarised in Table 2.

Intra-individual functions

Washing and cleaning compulsions of moderate to medium intensity strongly resemble phobic anxiety and avoidance. People with OCD believe that they are effectively avoiding or escaping contamination. In severe cases, they have lost the cut-off point for 'enough' (washing). Life mainly consists of washing/cleaning or even staying in bed to avoid contamination.

Controlling and putting things into order often occurs in people with OCD with primary low self-esteem. These people generally feel disliked and hope to gain social reward by 'more than perfect' functioning. If, for whatever reason, reward is

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Table 2 Intra-individual and interpersonal functions of obsessions and compulsions

	Degree of disturbance	
Obsessive–compulsive symptom	Moderate to medium	Severe
Washing, cleaning	As in phobias: avoidance of trigger. Compulsion equals undoing. Belief in self-efficacy	Life has become continuous attempt to avoid or escape trigger Loss of belief in self-efficacy leads to magical thinking to cope with uncertainty
Controlling, putting things into order	Coping with low self-esteem; reduction of fear of social rejection by belief that perfect performance will enforce social acceptance Coping with psychological or organic impairment of short-term memory Hope for self-efficacy	The less social reward for obsessive–compulsive behaviours, the more obsessive–compulsive behaviours enforced Loss of hope for self-efficacy leads to magical thinking to cope
Counting, repeating, touching	Prevention of harm/disaster from others or themselves Belief in efficacy of magic	The less social reward for obsessive–compulsive behaviours, the more obsessive–compulsive behaviours enforced Loss of hope for self-efficacy leads to magical thinking to cope
Ruminating before or after an action	(The same as in controlling, above)	The less social reward for obsessive–compulsive behaviours, the more obsessive–compulsive behaviours enforced Loss of hope for self-efficacy leads to magical thinking to cope
Ruminating of harming others or themselves	Fear of self-helplessness	Conviction of self-helplessness

no longer gained, they do not learn from failure but continuously apply more of what has already proven to be ineffective. Subjectively, this is due to their belief that their behaviour is right, only something else is going wrong! Objectively, is it just the consequence of a short-circuit in certain aspects of the relevant neuronal brain circuitry, for example repetitive triggering of the orbital cortico-basal ganglionic-thalamic pathway?

At the level of severe disturbance, both groups of compulsions can be reduced by the people with OCD using 'magical' rituals, like repeating one particular washing procedure three times × three times × three times. The magical power of the 'good' number three 'cleans' their whole body, even though all they did was only wash their hands in accordance with a 'magical' ritual.

Counting, repeating, touching and speaking compulsions based on 'magical formulas' can even be observed among people with moderate to medium forms of OCD. Ruminations before and after acting seem to serve mainly the same functions as controlling compulsions.

A frequent, different intra-individual function of these compulsions is distraction from primary depression.

Interpersonal functions

Important interactional functions of OCD seem to occur particularly in people with chronic social deficits, social withdrawal and low self-esteem. Symptom behaviour is used in such cases either to keep others at a distance or to fight interactional battles in close relationships (particularly with parents or spouses). It is our clinical impression that half of the more severely disturbed people with OCD have such a case history (summarised in Fig. 2).

Early social anxiety resulting from early social deficits is spontaneously replaced by latent aggressivity (aggression as a counter-conditioning emotion for anxiety), subsequently leading to a continuous preparedness for aggressive responses whenever there is the slightest indication of dislike from others (detailed discussion in Hand, 1991; McColloch & Gilbert, 1991). This type of pseudo-coping with deficits and anxiety often becomes unpleasant for the immediate

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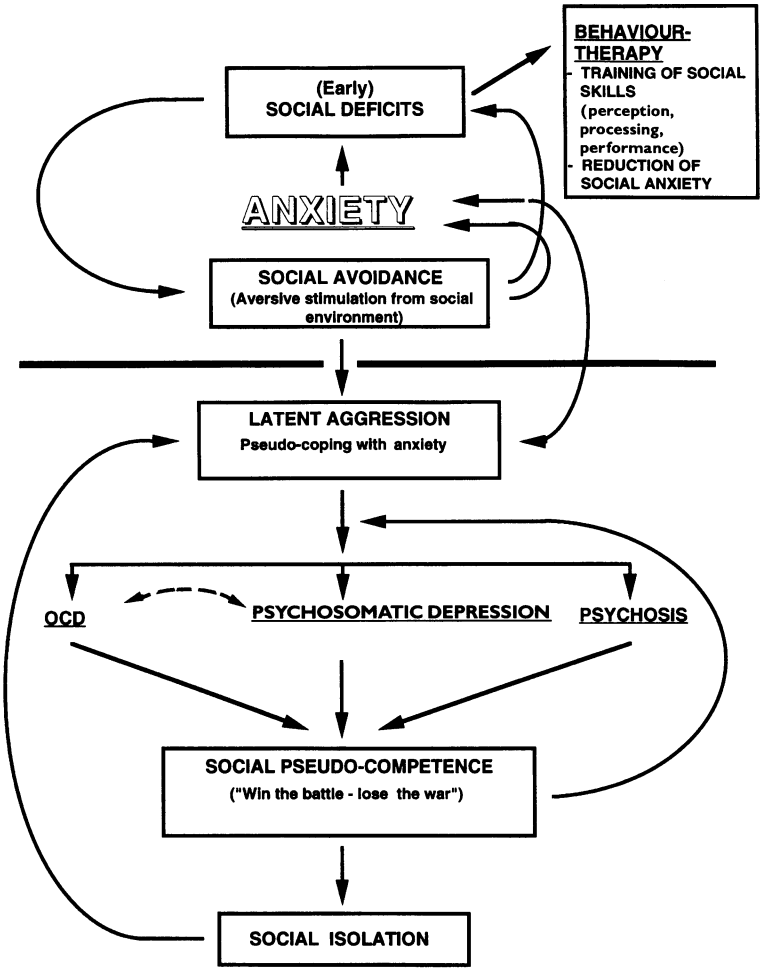


Fig. 2 Social deficits, anxiety and aggression complicating obsessive–compulsive disorder.

social environment (from family members to the therapist) and precedes dropping-out of treatment either by the patient or the therapist.

MULTI-MODAL INTERVENTIONS IN OCD

The complex analyses mentioned above usually lead to one of three types of possible interventions:

(a) symptom treatment as the only or main intervention;

(b) treatment 'around' the symptom, the main target being the causes and maintaining functions of OCD;

(c) multi-modal treatment, with symptom- and causal interventions, symptom interventions either being applied at the start of or later on during treatment.

In severe OCD, by far the most frequently used intervention is the third. The other two are more suited for people with medium to moderate disturbance.

In people with chronic OCD and chronic relationships, it is necessary to do a more elaborate diagnostic screening of spouses or family members regarding their deficits and the associated problems that result in their tolerating impairment of their own life by OCD. The integration of relatives as co-therapists therefore needs careful consideration, in order to avoid the fostering of pathological role differentiation (e.g. sane versus sick). A co-patient role is often more appropriate.

Symptom-directed interventions

The most effective and most frequently applied symptom-intervention in OCD is exposure both *in vivo* and in imagination to those stimuli that elicit symptom behaviour. Once these stimuli have provoked the typical emotional and cognitive reactions, coping skills can be learned through training. The traditional term 'exposure response prevention' (ERP) is misleading, as in the more recent modes of exposure only the motor-avoidance part of the compulsive response is omitted at the beginning. The cognitive–emotional–physiological response patterns are thus enhanced either in part or entirely. This creates the preconditions for training in anxiety–panic management, which then can be extended to training in depression or aggression (negative state) management.

During such an exposure *in vivo* or *in sensu* two basic responses are possible: (a) development of high emotional arousal occurs (as expected or feared); (b) high emotional arousal does not occur, contrary to the person's expectation. 'Reality testing' finally demonstrates to the person that what originally may have occurred no longer does, and a simple extinction process is thus induced.

In the case of (a), high emotional arousal is mostly experienced as anxiety. Not infrequently though, this may be transformed into anger, aggression or depression during extended exposure. Regardless of the emotion triggered, the patient is guided to describe it precisely, to find out the accompanying cognitions and to compare the real events with his original expectations (cognitive construct). In this approach, cognitive restructuring is the consequence of the patient's (motor) behavioural 'experiment' in the treatment process, leading to new emotional experience and cognitive set (the same applies for obsessions in cognitive exposure *in sensu*). In some cases, deeper self-analyses of the patients under increased emotional arousal will help to detect (sometimes in a catharsis-like process, e.g. Marks, 1987) causes for OCD, that previously were not consciously accessible. Once improvement in OCD has been achieved, the learning process in this exposure response management (ERM; Hand, 1993*b*) can be generalised to coping with subjective negative (emotional) states caused by other distress situations.

This concept could be labelled an 'emotional theory' of exposure, with behavioural and cognitive constituents. In ERM the patient learns that it is not the external trigger but the internal emotional–cognitive–physiological reaction that needs to be coped with (this is a similar concept to that

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in cognitive treatment of OCD; Salkovskis & Kirk, 1989). Regarding this mechanism, there is no major difference between exposure to 'real' external triggers and exposure *in sensu*. Unfortunately, in many earlier studies with behaviour therapy this procedure has not been applied. This may be the main reason why obsessions have frequently been regarded far less treatable than compulsions. We have used this procedure for about two decades, with similar effects in obsessions compared to *in vivo* exposure for compulsions (Lacher *et al*, 1988).

Contrary to a widely held belief in behaviour therapy publications over the last few years, that modern cognitive therapy can further improve the results of behaviour therapy in OCD, the few recent studies to investigate this assumption do not support it. James & Blackburn (1995) presented a meta-analysis of 15 published studies with cognitive therapy in OCD (only five of those controlled). However, they did not manage to find significant, additional effects ascribable to cognitive therapy. Van Oppen *et al* (1995) carried out what they called the 'first' controlled comparative study between self-help exposure *in vivo* versus cognitive therapy with additional behavioural experiments (actually also *in vivo* self-exposure). With univariate analyses, there were no statistically significant outcome differences in the main variables. Multivariate analyses revealed some superiority of cognitive therapy. Van Oppen *et al* concluded 'that cognitive therapy may be even more effective than exposure *in vivo*'. Yet, the opposite hypothesis could be proposed: in this study the least effective version of exposure (pure self-help application) was compared with cognitive therapy, which in itself was confounded by additional advice for exposure *in vivo* (self-help exposure). Therefore, a comparison of therapist-guided exposure *in vivo* with pure cognitive therapy may have produced results much in favour of exposure.

Even with regard to symptom techniques it becomes evident that separating (motor) behavioural and cognitive elements is counterproductive, as both occur together in human beings. In the treatment of individuals it is necessary to decide which of the four aspects of behaviour is most easily accessible at the beginning of therapy.

The symptom-directed interventions should preferably be conducted on an out-patient basis. Self-directed exposure with the use of self-help manuals (Marks, 1978; Baer, 1991; Foa & Wilson, 1991; Schwartz & Beyette, 1996) is probably effective in a subgroup of people with OCD, and promising results were achieved in a study with computer program-guided self-help (Greist & Jefferson, 1998, this volume). Much more research is required to determine, with a precision similar to what is now possible for the anxiety disorders, the exact indication for pure self-help or manual-guided exposure in OCD.

Provided the individuals and their immediate social surroundings are adequately motivated, therapist-guided exposure *in vivo* usually does not take more than 2–5 sessions, each lasting for 2–4 hours. Some therapists extend this to between 50 and 80 hours, with single sessions lasting up to 10 hours. Convincing indications for this mode of exposure are lacking. In very severe cases or where regional out-patient treatment is not available, in-patient strategic-systemic behaviour therapy can be of significant benefit (Hohagen *et al*, 1998, this volume); but prospective follow-up studies are needed to decide for whom additional subsequent out-patient behaviour therapy is necessary.

Causal interventions for OCD

Other typical problem areas besides OCD symptomatology, which are frequently encountered, include:

(a) early social deficits and social phobia – with lowered self-esteem, often latent aggressiveness and increased interactional vulnerability;

(b) reduced tolerance for intense emotions;

(c) reduced emotional awareness (anhedonia; alexithymia);

(d) deficits in problem-solving;

(e) deficits in self-initiated alternative behaviours, not only during the time of illness, but in the biographical development before first onset of illness;

(f) interactional and other traumata in the biography, often with hyper-memory for those events (with otherwise normal long- and short-term memory).

The causal and symptomatic interventions also try to reduce the following features of people with OCD: perfectionism, striving for more than 100% security; need for 100% predictability of future events; fear of losing control (uncontrollable aggressions, developing schizophrenia, etc.); exaggerated magical thinking and acting, to cope with negative emotions. The multi-modal behavioural interventions for these problem areas and their application in individual couple, family and group settings are summarised elsewhere (Hand, 1988, 1993*a*).

Combination of out-patient behaviour therapy and drug treatment

Regarding a combination of behaviour therapy and drug treatment (mainly selective serotonin reuptake inhibitors (SSRIs) or clomipramine), we currently have to rely more on clinical impressions than on data from comparative research. The following recommendations are therefore only preliminary.

Medication prior to behaviour therapy may be indicated if:

(a) severe, secondary (to OCD) depression is present. Cognitive and behavioural flexibility of those patients may be too reduced for optimal use of behavioural interventions;

(b) extreme obsessive–compulsive symptomatology exists, associated with high anxiety. Motivation of these patients to try exposure may be difficult to attain without medication;

(c) support of the self-help activities (using behaviour methods), when expert behaviour therapists are not available.

Medication during behaviour therapy may be indicated if:

(a) there is slow treatment progress, possibly due to initial underestimation of the severity of the disturbance;

(b) there is a severe depressive reaction to treatment interventions.

Medication after behaviour therapy may be indicated if:

(a) behaviour therapy has been only moderately effective despite good co-operation;

(b) a relapse occurs, to foster self-help potential when further out-patient behaviour therapy is not available.

LONG-TERM OUTCOME OF OUT-PATIENT BEHAVIOURAL THERAPIES

Between 1973 and 1986 some 12 follow-up studies on behaviour therapy in OCD

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(mainly exposure treatments) were published. Success ratings varied from 60–80% (Marks, 1987).

In our own retrospective and prospective long-term follow-up studies (up to 13 years after treatment) on patients with anxiety disorders (simple phobia, agoraphobia, social phobia) and OCD we found the following response rates: agoraphobia 75% (follow-up compliance in 83% of the treated patients); social phobia 69% (follow-up compliance in 64% of the treated patients); OCD 49% (follow-up compliance only 53% of the treated patients).

While the first two results are almost identical to those in international publications, the outcome in our OCD patients is clearly worse. There may be major differences in our treated populations compared with those in research projects, as all our patients were unselected and treated as part of our service to the community. Quite a high proportion of our subjects were ambivalent about treatment in the initial interviews, but we felt obliged to try to change their motivation by offering treatment in spite of their doubts. Furthermore, people with OCD who qualify for research projects may, by this criterion, be a less disturbed sub-population. We found that the vast majority of those 47%, who refused to participate in follow-up, did so because they did not want to undergo all the OCD-related ratings again. They feared this might trigger forgotten memories of their earlier symptoms or worsen the acute symptomatology. Even among those who participated in our follow-up, quite a few had severe problems in going through all the rating scales again. Therefore, we selected a subgroup of 34 who did not have this worry and calculated their outcomes separately. In these subjects, we found a rate of response to treatment of around 70% (Lacher, 1989).

Of course, this is no proof for our assumption concerning the reasons for treatment non-response. Direct comparison of study outcomes is difficult in any event, because of unresolved problems regarding the operationalisation of success, and because of the quite different assessment scales that have been applied by different research groups over the years.

Our follow-up studies, and the outcome of drug studies in OCD when compared with those on anxiety disorders, seem to support the European view in psychiatry that OCD is not a subgroup of the anxiety disorders. The main differences in OCD are:

(a) symptom-behaviour has a positive subjective function;

(b) motivation for change is often initially restricted;

(c) treatment outcome with psychotherapies or pharmacotherapies is worse than in anxiety disorders;

(d) relationship distress is a very important variable for treatment/resistance or relapse;

(e) interactional vulnerability is more similar to schizophrenia;

(f) latent aggression frequently leads to dropping out of patient (or therapist) for treatment.

RECOMMENDATIONS TO IMPROVE LONG-TERM EFFECTIVENESS OF OUT-PATIENT BEHAVIOURAL THERAPY IN OCD

The efficacy, acceptance and cost-effectiveness of short-term interventions for people with OCD should certainly be further improved. The identification of factors which predict treatment response is a major goal. Systematic research to increase the response rates in unselected treatment samples is as important as long-term strategies for the treatment of people with severe depression or schizophrenia. For the time being, pragmatic measures already undertaken in several therapeutic institutions to help people with treatment-resistant OCD may be summarised as follows.

First, extension of initially intense short-term treatment (with or without spouses, relatives) to a long-term low intensity supportive programme, keeping people in contact with their therapist(s) for a period of up to three or five years. The costs of such a maintenance treatment may prove to be rather low in comparison to the costs that arise when these patients relapse – not to mention the costs of long-term maintenance treatment with expensive medication like selective serotonin re-uptake inhibitors.

Second, transfer of people with chronic OCD into behaviourally-oriented self-help groups (with regard to the symptom, as well as causal interventions). Our own experience and that in other countries suggest that such self-help groups should be trained by behaviour therapists for about a year. The therapists first conduct each weekly meeting, and then gradually discharge the group into complete self-help, while remaining available on request. We already have encouraging results from a pilot study with seven such groups (only the first 10 sessions being therapist-assisted) of severely disturbed people with chronic OCD (Münchau *et al*, 1996).

Third, in long-term therapy for chronic OCD involvement of relatives is usually necessary. Interventions may, as for depression and schizophrenia, range from psychoeducational sessions for the relatives, to joint treatment of sufferers and relatives (even in groups; Hand & Tichatzki, 1979), to separate additional treatment for spouses or parents. Interventions for relatives are not only necessary to relieve their burden of suffering from OCD (Calvocoressi *et al*, 1995), but also for their own primary deficits.

Fourth, systematic research regarding the indication for the biobehavioural self-help approach of Schwartz (1998, this volume). With the keynote 'it's not me, it's my OCD', this approach tries to alienate the individual from his or her symptoms and encourage them to work around or 'live in spite of' their symptoms. This PET study-based approach thus turns out to be a version of cognitive behaviour therapy utilising a somatic illness concept of OCD. In contrast, the multi-modal behavioural approach also emphasises the possible signal function of symptom (re-) occurrence and trains people to improve their awareness of unresolved problems, behavioural deficits, or failures in daily life conduct. The biobehavioural mode may be attractive both for people whose biological variables play a decisive role and for those who recognise their severe conflicts but do not wish or dare to tackle them in treatment. It would be very interesting to compare the acceptance and the effectiveness of both these approaches, and to consider the possibility that they may display synergy when used together. Research into this direction may enhance our ability to improve both the efficacy and cost-effectiveness of the treatment of OCD.

REFERENCES

Baer, L. (1991) *Getting Control*. Boston, MA: Little, Brown & Co.

Baxter, L., Saxena, S., Brody, A., *et al* (1991) Brain mediation of obsessive–compulsive disorder symptoms: evidence from functional brain imaging studies in the human and nonhuman primate. *Seminars in Clinical Neuropsychiatry*, **1**, 32–47.

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Calvocoressi, L., Lewis, B., Harris, M., *et al* (1991) Family accommodation in obsessive–compulsive disorder. *American Journal of Psychiatry*, **152**, 441–443.

Erikson, E. (1978) *Toys and Reasons: Stages in the Ritualization of Experience*. London: Marion Boyars.

Foa, E. & Wilson, R. (1991) *Stop Obsessing. Self-Help for Obsessions and Compulsions*. New York: Bantam Books.

Friesen, J. (1985) *Structural-Strategic Marriage and Family Therapy*. New York: Gardner Press.

Gazzaniga, M. S. (1992) *Nature's Mind: The Biological Roots of Thinking. Emotions, Sexuality, Language and Intelligence*. New York: Basic Books.

Greist, J. H. & Jefferson, J. W. (1998) Pharmacotherapy for obsessive–compulsive disorder. *British Journal of Psychiatry*, **173** (suppl. 35), 64–70.

Hand, I. (1986) Verhaltenstherapie und Kognitive Therapie in der Psychiatrie. In *Psychiatrie der Gegenwart 1* (eds K. P. Kister, H. Lauter, J. E. Meyer, *et al*), pp. 277–306. Berlin: Springer Verlag.

—— (1988) Obsessive–compulsive patients and their families. In *Handbook of Behavioral Family Therapy* (ed. I. R. H. Falloon), pp. 231–256. New York: Guilford Press.

—— (1991) Aggression und soziale Defizite bei psychischen Erkrankungen. In *Aggression und Autoaggression* (ed. W. Pöldinger), pp. 27–46. Duphar Med. Script, Bd. 8, Hannover: Duphar.

—— (1993*a*) Verhaltenstherapie für Zwangskranke und deren Angehörige. In *Therapie Psychiatrischer Erkrankungen* (ed. H. J. Möller), pp. 508–528. Stuttgart: Ferdinand Enke Verlag.

—— (1993*b*) Exposition-Reaktions-Management (ERM) in der strategisch-systemischen Verhaltenstherapie. *Verhaltenstherapie*, **3**, 61–65.

—— (1997) "Zwangs-Spektrum-Störungen" oder "Nicht-Stoffgebundene Abhängigkeiten"? In *Psychotherapie in der Psychiatrie* (eds Ch. Mundt, M. Linden & W. Barnett), pp. 209–219. Berlin: Springer Verlag.

—— (1998) Pathological gambling: a negative state model and its implications for behavioural treatments. *CNS Spectrums*, **3**, 58–71.

——, Spoehring, B. & Stanik, E. (1977) Treatment of obsessions, compulsions and phobias as hidden couple-counselling. In *The Treatment of Phobic and Obsessive Compulsive Disorders* (eds J. C. Boulougouris & A. D. Rabavilas), pp. 105–114. Oxford: Pergamon Press.

—— & Tichatzki, M. (1979) Behavioral group therapy for obsessions and compulsions: first results of a pilot study. In *Trends in Behavior Therapy* (eds P. O. Sjöden, S. Bates & W. S. Dockens, III), pp. 269–298. New York: Academic Press.

Hohagen, F., Winkelman, G., Rasche-Räuchle, H., *et al* (1998) Combination of behaviour therapy with fluvoxamine in comparison with behaviour therapy. *British Journal of Psychiatry*, **173** (suppl. 35), 71–78.

James, I. A. & Blackburn, I.-M. (1995) Cognitive therapy with obsessive–compulsive disorder. *British Journal of Psychiatry*, **166**, 444–450.

Lacher, M. (1989) Langzeitaffekte von Kurzzeit-Verhaltenstherapie bei zwangsneurotikern. PhD thesis. University of Hamburg.

Lazarus, A. (1976) *Multi-Modal Behavior Therapy*. New York: Springer.

Marks, I. M. (1978) *Living with Fear*. New York: McGraw-Hill.

—— (1987) *Fears, Phobias and Rituals*. Oxford: Oxford University Press.

McColloch, M. & Gilbert, B. (1991) Development and maintenance of aggressive behavioral patterns. In *Personality, Social Skills, and Psychopathology* (eds D. Gilbert & J. Conolly), pp. 185–210. New York: Plenum Press.

Münchau, N., Hand, I., Schaible, R., *et al* (1996) Aufbau von Selbsthilfegruppen für Zwangskranke unter verhaltenstherapeutischer Expertenanleitung: empirische Ergebnisse. *Verhaltenstherapie*, **6**, 143–161.

Pato, M. T., Zohar-Kadouch, J. & Murphy, D. L. (1988) Return of symptoms after discontinuation of clomipramine with obsessive–compulsive disorder. *American Journal of Psychiatry*, **145**, 1521–1525.

Salkovskis, P. & Kirk, J. (1989) Obsessional disorder. In *Cognitive Behaviour Therapy for Psychiatric Problems* (eds K. Hawton, P. M. Salkovskis, J. Kirk & D. M. Clark), pp. 129–168. Oxford: Oxford University Press.

Schwartz, J. M. & Beyette, B. (1996) *Brain Lock: free Yourself from Obsessive–Compulsive Behavior*. New York: Regan Books.

Van Balkom, A. J., van Oppen, P., Vermeulen, A. W., *et al* (1994) A meta-analysis on the treatment of obsessive compulsive disorder: a comparison of antidepressants, behavior, and cognitive therapy. *Clinical Psychology Review*, **14**, 359–381.

Van Oppen, P., De Haan, E., Van Balkon, A., *et al* (1995) Cognitive therapy and exposure in-vivo in the treatment of obsessive–compulsive disorder. *Behavior Research and Therapy*, **33**, 379–390.

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