The Rational-Choice Theory of Neurosis: Unawareness and an Integrative Therapeutic Approach

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In agreement with Freud (1915a, 1915b), patients' unawareness regarding the underlying causes of their behavior seems to be the key to understanding the development and treatment of neurotic disorders. However, Freud's concepts of the unconscious and repression have encountered serious problems that do not allow for their maintenance (see review by Rofé, 2008). An additional issue concerns the fact that none of the available theories can integrate the therapeutic effects of various interventions under a single theoretical framework. Both of these issues are addressed in this article by the rational-choice theory of neurosis (RCTN). RCTN maintains that patients consciously and deliberately adopt neurotic disorders when confronted with intolerable levels of stress. Unawareness is created through sophisticated cognitive processes, by which patients forget their own self-involvement. Subsequently, patients develop a self-deceptive belief that rationalizes the neurotic behavior, thereby preserving unawareness. According to this new theory, all therapies exert their effect either by disrupting patients' ability to preserve unawareness, increasing the cost of the symptom, decreasing the patient's emotional distress, or eliminating the stressor.

Keywords: neurosis, rationality, unconscious, unawareness, therapy

In recent years, Freud's (1915a, 1915b) theory of neurosis has encountered serious problems. A vast amount of studies have invalidated the concept of repression (e.g., see reviews by McNally, Clancy, & Barrett, 2004; Piper, Lillevik, & Kritzer, 2008; Piper, Pope, & Borowiecki, 2000; Rofé, 2008), which constitutes the "cornerstone on which the whole structure of psychoanalysis rests" (S. Freud, 1914, p. 16). Studies have also questioned the existence of the unconscious (see review by Rofé, 2008) and

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investigators such as G. O'Brien and Jureidini (2002) came to the conclusion that "far from supporting the dynamic unconscious, recent work in cognitive science suggests that the time has come to dispense with this [Freudian] concept altogether" (p. 141; see also Greenwald, 1992; Grünbaum, 2002; Kihlstrom, 1999, 2000, 2004).

Several cardinal problems relating to the development and treatment of neurosis also challenge the validity of other traditional theories of psychopathology, such as behavior, cognitive, and biological theories. Regarding the development of neurosis, these theories are incapable of accounting for factors affecting the prevalence of neurotic disorders, such as gender and sociocultural differences, and for fluctuations across different time periods. For example, it is unclear why women display a higher prevalence for neurotic disorders, such as conversion disorder (e.g., Alper, Devinsky, Perrine, Vazquez, & Luciano, 1995; Jones, 1980), agoraphobia and panic disorder (e.g., Ginsberg, 2004; Woodman, 1993), eating disorders (e.g., Sanford et al., 2005; Walcott, Pratt, & Patel, 2003), and dissociative identity disorder **(**DID; e.g., Coons, Bowman, & Milstein, 1988; Zywiak, 1996). Why should obsessive–compulsive disorder (OCD) be the only exception in this matter (e.g., *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed., text revision [*DSM–IV–TR*], American Psychiatric Association [APA], 2000; Lochner & Stein, 2001)? Similarly, it is also unclear why the prevalence of neurotic disorders should be affected by sociocultural factors, such as the significant relationship between OCD and religion (e.g., Abramowitz, Deacon, Woods, & Tolin, 2004; Raphael, Rani, Bale, & Drummond, 1996), the striking differences between Western and non-Western countries in the prevalence of neurotic disorders (e.g., Bhadrinath, 1990; Keel & Klump, 2003; Pate, Pumariega, Hester, & Garner, 1992), and the higher prevalence of conversion disorder among lower socioeconomic classes (e.g., Jones, 1980; Kuloglu, Atmaca, Tezcan, Gecici, & Bulut, 2003; Nandi, Banerjee, Nandi, & Nandi, 1992). Likewise, traditional theories have difficulty explaining the diagnostic fluctuations in neurotic disorders across different periods of time, such as the significant decrease in conversion disorder (e.g., Jones, 1980; Nandi et al., 1992), or the steep increase in cases of DID (e.g., Lilienfeld et al., 1999; Mai, 1995; Merskey, 1995; North, Ryall, Ricci & Wetzel, 1993) and eating disorders (e.g., Lucas, Beard, O'Fallon, & Kurland, 1991; Mitchell & Eckert, 1987; Willi, Giacometti, & Limacher, 1990).

Regarding treatment, traditional theories are also challenged by their inability to address the mechanism by which therapeutic change occurs and are incapable of integrating each others' findings in this area (e.g., see reviews by Feixas & Botella, 2004; Lampropoulos, 2000, 2001). For example, there is no theory to date that can account for the therapeutic efficacy

of various interventions, such as cognitive, behavioral, psychoanalytic, religious (e.g., shaman), drug, and placebo therapies, for different neurotic disorders. Why should both drug therapy (e.g., D. D. Miller, 2000; Putnam & Loewenstein, 1993) and psychoanalysis (e.g., Kihlstrom, 2005; Kluft, 1995) be effective for DID, while CBT (cognitive–behavioral therapy) is the treatment of choice for panic disorder and agoraphobia (e.g., Taylor, 2000)?

The fundamental cause for the theoretical deadlock in psychopathology, which did not allow us to resolve the aforementioned difficulties, might be the common assumption of all traditional theories that neurotic disorders are determined by irrational-mechanistic processes. Although this assumption never received empirical validation, it appeared plausible, given the senseless nature of the neurotic symptom, patients' inability to account for these behaviors which severely disrupt their daily life, and their inability to resume normal functioning. In retrospect, however, this interpretation that neurotic behaviors necessarily reflect irrational processes may be seen as the major cause of the theoretical standstill in understanding the development and treatment of neurotic disorders.

The idea that rationality may be crucial for understanding human behavior has been acknowledged by various fields of social sciences, such as economics (e.g., Aumann & Hart, 2002; Becker, 1976; Climaco & Ramos, 2004; Miljkovic, 2005); criminology (e.g., Guerette, Stenius, & McGloin, 2005); political science (e.g., Liu, 2005; Margolis, 2001; Quattrone & Tversky, 2004); sociology (e.g., Krausz, 2004; Pedriana, 2005); and even psychology, in the realm of suicidal behavior (e.g., Kelleher, 1998; Lester, 1988a, 1988b, 1997; Werth, 1996; Yufit & Lester, 2005).

After a century of theoretical deadlock in psychopathology, the time has arrived to examine whether this trend in social science can also be applied to account for the development and treatment of neurosis. Accordingly, a new theory has been suggested, the rational-choice theory of neurosis (RCTN; Rofé, 2010), which accounts for the development and treatment of neurosis in conscious-rational terms. RCTN is a revised version of psychobizarreness theory, presented in the book, *The Rationality of Psychological Disorders* (Rofé, 2000; see also Lester, 2002; Sarma & Garfield, 2001). The basic assumption of RCTN is that when individuals are confronted with intolerable levels of stress, response options become limited. The main choices include suicide, substance abuse, aggressive measures to eliminate the stressor, or simply remaining highly distressed. Some individuals, however, will choose a neurotic behavior, primarily because of its high distractive value that enables them to actively repress stress-related thoughts, thereby relieving their emotional distress. Thus, contrary to the psychoanalytic doctrine, but in accordance with S. Freud's (1915a, 1915b) original thought (see also Erdelyi, 1990, 2001, 2006) and

with the bulk of experimental studies in this area (see Holmes, 1974, 1990), *repression* is defined by RCTN as a deliberate and conscious distraction. From this standpoint, repression is the consequence, rather than the cause, of neurotic symptoms.

The rational approach to neurosis enables us to account for the variability in the prevalence of neurotic disorders (i.e., choice of symptom). Just as in an economic decision-making process, in which people purchase a specific product (e.g., Wänke & Friese, 2005), a vast amount of research and clinical evidence indicate that the choice of neurotic symptom is determined by three main principles: (a) controllability, that is, the individual's need to exercise control over the stressor (e.g., soldiers during World War II developed conversion symptoms because they increased their ability to escape from combat stress; see Ironside & Batchelor, 1945; Mucha & Reinhardt, 1970); (b) availability of certain symptoms through various channels of information (e.g., media, peer group, family; see Spanos, 1996; Spanos, Weekes, & Bertrand, 1985); and (c) cost-benefit analysis (e.g., men are less likely to choose neurotic symptoms due to damage to their work abilities and social embarrassment). These three principles resolve the aforementioned difficulties regarding gender differences, sociocultural differences, and diagnostic fluctuations in the prevalence of neurotic disorders (Rofé, 2000, 2010).

The fact that patients with neurotic disorder are unaware of the underlying causes of their behavior appears inconsistent with RCTN's idea that neurotic disorders are conscious and deliberate. If neurosis is truly consciously and deliberately adopted, how does the patient remain unaware of their conscious involvement in this process? It is also unclear why therapeutic interventions that emerge from traditional theories, antithetical to RCTN, should exert positive therapeutic change. Why should CBT, psychoanalysis, religious therapy, punishment-reinforcement therapy, and drugs, which seemingly have nothing to do with rationality, be effective? Moreover, why should patients invest their time and money in therapy, if they consciously and deliberately adopt and maintain these behaviors? The answer to both of these questions, unawareness and therapy, constitute the goal of the present article. As will be shown later, the understanding of the issue of unawareness is critical for the clarifying of the mechanism by which therapeutic change occurs.

UNAWARENESS: A SELF-DECEPTIVE PROCESS

RCTN agrees with psychoanalysis that the mechanism by which patients with neurotic disorder become unaware of the underlying cause of

their deviant behavior is the key to understanding the neurosis (e.g., Shevrin & Dickman, 1980; Woody, 2003). However, contrary to psychoanalysis, RCTN accounts for the lack of awareness in conscious and rational terms, and attributes this effect to two psychological mechanisms. One mechanism, elaborated in previous works (Rofé, 2000, 2010), relates to repressive processes that are produced by the powerful distractive value of the symptom. This form of unawareness enables patients to remove stress-related thoughts from attention by focusing on the symptom. As a result, patients are no longer aware of the original stressor that motivated symptom adoption.

The second mechanism, discussed in the Creation of Unawareness and Preservation of Unawareness sections, relates to the psychological processes by which patients become unaware of their conscious and deliberate involvement in the adoption and maintenance of the symptom. This psychological process by which patients create unawareness of the knowledge of self-involvement (KSI) is essential for maintaining the repressive value of the symptom. The symptom would lose its distractive value if patients were to remain aware of their conscious involvement in its adoption. Accordingly, any therapeutic intervention that disrupts unawareness of KSI should damage the repressive value of the symptom and thus increase the patient's tendency to abandon this behavior. Indeed, as shown in the Integrative Therapy Model of Neurosis section, various therapeutic interventions, such as CBT, psychoanalysis, and religious therapy, obtain their therapeutic benefit by damaging the psychological maneuver by which patients achieve and maintain unawareness of KSI.

The manner through which patients become unaware of KSI is composed of two stages. In the first stage, a number of factors disrupt the encoding of KSI and impair its retrieval. In the second stage, patients preserve this state of unawareness by developing self-deceptive beliefs, which enable them to attribute the cause of their symptom to factors beyond their control. These two stages are addressed below.

Creation of Unawareness

As the neurotic symptom is adopted, several disruptive factors weaken the encoding of KSI. Subsequently, a number of memory-inhibiting mechanisms further diminish KSI, to such an extent that its initial retrieval becomes impossible. The phenomenological result of these processes is a temporary lack of awareness regarding the patient's active and conscious involvement in producing the deviant behavior.

Encoding-Disruptive Factors

When a patient decides to adopt a neurotic symptom, the encoding of KSI may be weakened by any of the following four factors:

1. *Impaired cognitive functioning:* A vast amount of studies have indicated that the adverse emotional states of anxiety and depression are likely to cause cognitive impairments, especially attentional deficits (e.g., Erickson et al., 2005; Mialet, Pope, & Yurgelun-Todd, 1996; Rief & Hermanutz, 1996). Findings also show that subjects' emotional distress distracts attention and interferes with encoding processes, resulting in poor learning and memory (e.g., Christopher & MacDonald, 2005; Krames & MacDonald, 1985; Mialet et al., 1996; J. T. O'Brien, Sahakian, & Checkley, 1993; Roberson-Nay et al., 2006; Rose & Ebmeier, 2006). Accordingly, given RCTN's assumption that neurotic disorders are adopted when patients are faced with an intolerable level of stress, the encoding of KSI is likely to be disrupted at the decision-making time at the onset stage.
2. *Directed forgetting:* Studies in which subjects are instructed to forget given information, demonstrate that people have the ability to forget information by deliberately disrupting the encoding process (e.g., Anderson, 2005; Golding, 2005; Gottlob, Golding, & Hauselt, 2006; Hourihan & Taylor, 2006). Thus, given patients' interest in being unaware of their self-involvement, they can hinder the encoding of KSI by intentionally directing their attention away from this information.
3. *Symptom distractibility:* Research has shown that encoding can be disrupted by distractive factors irrelevant to the memory task (e.g., Tremblay, Nicholls, Parmentier, & Jones, 2005; Wolach & Pratt, 2001). In this regard, the behavioral change is so dramatic and unusual that it is likely to consume the patient's entire attention. Attention becomes so intensely focused on these extremely powerful and distractive stimuli that the encoding of rival stimuli, such as KSI, is seriously disrupted. This suggestion is consistent with Van Pelt's (1975) theoretical position, which noted that neurotic symptoms cause "*superconcentration of the mind*, [inducing] a state of what may be called *accidental hypnosis* . . . . A patient suffering from a psychoneurosis behaves in every way *as though under the influence of a posthypnotic suggestion*" (pp. 28–29; see also Bliss, 1980, 1983, 1984; Frankel, 1974). A clinical example demonstrating the overwhelming effect that neurotic symptoms might have on the individual's attention can be seen in DID, when patients altered

their personalities. As noted by Bliss (1980), a DID patient "creates personalities by blocking everything from her head, mentally relaxes, concentrates very hard . . . clears her mind, blocks everything out and then wishes for the person, but she isn't aware of what she's doing" (p. 1392).

An additional example demonstrating the powerful distractive effect of neurotic symptoms can be seen in Leonard's (1927) autobiographic account during his initial panic attack:

> Then on the tracks from behind . . . comes a freight-train, blowing its whistle. Instantaneously diffused premonitions become acute panic. The cabin of that locomotive feels right over my head, as if about to engulf me . . . . The train feels as if it were about to rush over me . . . I race back and forth on the embankment. I say to myself (and aloud): "it is half a mile across the lake—it can't touch you, it can't; it can't run you down . . . . I rush back and forth on the bluffs: My God, won't that train go, my God, won't that train go away!" I smash a wooden box to pieces, board by board, against my knee to occupy myself against panic. (pp. 304–307)

Thus, Leonard (1927) was so intensively preoccupied by symptom-related behaviors, including extreme catastrophic thoughts, exaggerated motor behaviors and loud shouting, to such an extent that they severely disrupted the encoding of KSI. Imagine that a person is briefly exposed to some information such as a telephone number given by an operator, while preoccupied by behaviors such as those of Leonard. He certainly would have difficulty memorizing this information.

4. *Brief encoding period:* Clinical evidence indicates that the ultimate decision regarding the choice of a specific neurotic disorder is made spontaneously, in the absence of prior planning. For example, Malamud (1944) described the case of an unhappily married man whose wife became pregnant, despite an agreement that they would not have children. On the way to the hospital to visit his wife and newborn child, the man was slightly injured in a car accident and immediately developed hysterical blindness. Obviously, the patient did not have an opportunity to contemplate the knowledge of self-involvement, as the decision to develop the symptom was made spontaneously (see also Leonard, 1927, for an additional case of spontaneous decision). Hence, given studies showing that exposure time is a crucial factor in encoding (e.g., Memon, Hope, & Bull, 2003; Shapiro & Penrod, 1986), and because the exposure time to KSI at the onset stage is brief, patients may not have sufficient time to encode this anxiety-provoking information.

In summary, the above four factors should substantially weaken the encoding of KSI and may even cause the patient to be totally unaware of

this knowledge. It is difficult to say at this stage of inquiry whether all these factors have an effect on every case, but the disruption of KSI may occur even if only one of these factors exerts its influence.

Retrieval-Inhibiting Mechanisms

The encoding-disrupting factors only weaken the memory of KSI but may not be sufficient to cause the forgetting of this information. A complete unawareness is obtained due to several retrieval-inhibiting mechanisms, specified below.

1. *State-dependent memory and environmental context:* Numerous studies on state-dependent memory indicate that retrieval becomes difficult when the individuals' conditions during retrieval differs from that of the original learning situations (e.g., Blaney, 1986; Emmerson, 1986; Houston, 1991; Kellogg & Dare, 1989; Lang, Craske, Brown, & Ghaneian, 2001). Similarly, altering the individual's emotional state facilitates forgetting (e.g., Bower, 1981; Clark, Milberg & Ross, 1983; Eich, 1995; Eich, Macaulay & Ryan, 1994; Pearce et al., 1990). Because symptom adoption relieves emotional distress, the radically different emotional state after symptom adoption hinders the retrieval of KSI. State-dependent memory should be especially effective in inhibiting KSI among DID patients because the alternate personalities tend to be extremely different in terms of tastes, preferences, emotion, behavior, gender, and age (Putnam, Guroff, Silberman, Barban, & Post, 1986; Spanos et al., 1985). Changes in environmental context also have been shown to reduce recall and recognition performance (see review Smith & Vela, 2001). Several symptoms, such as agoraphobia, dissociative fugue, and compulsive cleaning, generate significantly different environments from those prior to symptom adoption and thus would also inhibit the retrieval of KSI.
2. *Suppression:* Experimental evidence indicates that people can recruit a cognitive-neuropsychological mechanism by which awareness is prevented for unwanted memories (e.g., Anderson & Green, 2001; Anderson et al., 2004; Geraerts & McNally, 2008; Levy & Anderson, 2002, 2008). These studies demonstrated that when participants were instructed to intentionally forget earlier learned items, subsequent memory performance was impaired. Hence, patients can decrease the retrieval of KSI by intentionally suppressing thoughts about this anxiety-provoking knowledge, and by avoiding situations that may remind them of it. For example,

after the adoption of the symptom, Leonard (1927) never returned to the place where he experienced his panic attack, and a patient with dissociative fugue (Masserman, 1946) succeeded in maintaining a state of unawareness for her symptom by avoiding people who questioned her about her past. This theoretical approach is consistent with the psychoanalytic concept of suppression, which was seen as conscious efforts to forget undesirable material (e.g., A. Freud, 1936).

3. *Hypnotic amnesia:* Research pertaining to hypnotic amnesia has shown that this phenomenon is the consequence of active distractive maneuvers through which the subject deliberately ignores relevant target cues and attends exclusively to other matters (e.g., Bowers & Woody, 1996; Spanos, 1986, 1996; Wagstaff & Frost, 1996). Hence, given the powerful distractive values of the neurotic symptom even after the encoding stage, patients with neurotic disorder can induce an amnesic-hypnotic state by deliberately focusing on the neurotic behavior, thereby blocking the retrieval of KSI.

Thus, the weak memory resulting from the encoding-disrupting factors and the retrieval-inhibiting mechanisms discussed in this section, are capable of causing a total state of unawareness immediately after the initial display of the symptom.

Preservation of Unawareness: Self-Deceptive Beliefs

Although the memory-inhibiting factors remain in effect as long as the symptom persists, they may be insufficient for maintaining a prolonged state of unawareness of symptom selection. Given the rational nature of the human race, patients may become preoccupied with questions posed by themselves or by others regarding the causes of their radical behavioral changes. This is consistent with Nisbett and Wilson's (1977) suggestion that people search for explanations when they are unaware of the etiology of their behavior (e.g., see case studies, Kraines, 1948, p. 183; Leonard, 1927, p. 308). Obviously, such self-probing can threaten the state of unawareness. As demonstrated below, clinical observations indicate that patients address this threat by developing either a self-deceptive belief of illness or a self-deceptive belief of denial, depending on the specific information to which they are exposed immediately after the creation of unawareness, which hinders the self-inquiry as to the underlying causes of their behavioral change.

Self-Deceptive Belief of Illness

After the production of unawareness, most patients with neurotic disorder are exposed to two types of evidence. One type is the external observation of a deviant behavior (e.g., panic attack), and the second type is the internal experience of loss of control. As a result of this information, patients diagnose themselves as physically or mentally ill, thereby attributing the deviant behavior to forces beyond their control. Most subjects will also develop a self-deceptive explanation regarding the nature of their illness, based on their culturally internalized beliefs. Every society has beliefs concerning the etiology of deviant behaviors, such as psychoanalytic thought in Western societies (e.g., Spanos, Weekes, Menary, & Bertrand, 1986) or spiritual possession in non-Western cultures (e.g., Claus, 1979; Kua, Sim, & Chee, 1986; Mischel & Mischel, 1958), which the individual absorbs through socialization (e.g., see Ravenscroft, 1965) or other channels of information (e.g., reading or TV; see Leonard, 1927, pp. 324, 410; McAndrew, 1989, p. 316). As noted by Nisbett and Wilson (1977), people have beliefs concerning the causal factors of various situations, which they acquired from their culture and employ to account for these events. Thus, Spanos et al. (1986), claiming that the prevalence of DID in Western societies is due to Freudian influence, noted that, "In our culture many people learn to conceptualize interpersonal problems as resulting from an 'unconscious process' and as stemming from early negative experiences with parents" (p. 300).

One exceptional case study that described the development of a self-deceptive belief of illness is Leonard's (1927) autobiographical account of his panic disorder. Observing a dramatic behavioral change and a total loss of control, Leonard initially thought that he had been stricken with sunstroke (p. 308), but soon rejected this diagnosis. Continuing the self-diagnostic evaluation, he arrived at the conclusion that he suffered from a more serious problem when he stated that, "I know I am in a critical condition" (p. 308). Apparently, in an attempt to intensify the distractive value of the symptom, Leonard exaggerated his condition to such a state that when he arrived at his parents' home, he lay down, shaken with terror, saying in a low voice, "Father and mother, this looks like the end. I guess I am dying" (Leonard, 1927, p. 308). Further, Leonard utilized his broad knowledge of psychoanalysis to develop a self-deceptive explanation regarding the nature of his illness:

> I was my own physician . . . . I knew I was a "case." I knew my terrors were phobic . . . . I even knew they were somehow infantile in ultimate causes. A matter of infantile regression. The source of that conviction is psychologically noteworthy . . . I was . . . already familiar with abnormal psychology—even as I have since read,

> first and last . . . the chief men in the field, their books and technical journals in half a dozen languages . . . even to eight German volumes of Freud. (pp. 323–324)

Thus, Leonard (1927) developed a self-deceptive explanation that his panic attack was "a matter of infantile regression" (p. 323). In fact, Leonard was no different from patients originating from less-developed countries, who employed concepts from their culture, such as spiritual possession, to develop a self-deceptive explanation for preserving a state of unawareness when adopting bizarre behavioral deviations (e.g., see Csordas, 1987; Grisaru & Witztum, 1995; Kua et al., 1986; Ravenscroft, 1965; Seltzer, 1983; Varma, Bouri, & Wig, 1981; Wijesinghe, Dissanayake, & Mendis, 1976).

Due to the increasing popularity of medical models (e.g., Reich, 1982; Telch, 1988), even among the general populace, patients from Western cultures may focus on a belief of an organic cause. For example, in a case study of obsessive neurosis reported by McAndrew (1989), a woman relating her extreme suffering from having aggressive thoughts toward her beloved children, raised the possibility that the etiology of her symptom might have been "premenstrual syndrome" (p. 316). She arrived at this conclusion after seeing a TV program about a woman with premenstrual syndrome who had stabbed her children. In another case, O'Kearney (1993) described a 24-year-old woman in her final year of medical school who suffered from obsessive ruminations of extreme aggressive thoughts. The patient reported that she had suffered intrusive thoughts at a younger age as well, and linked the latest onset of ruminations to minor illnesses such as "a short duration gastric upset" (p. 358). Likewise, conversion disorder patients utilize the high prevalence of organic illnesses and physical injuries that are present prior to their disorder (e.g., Blanchard & Hersen, 1976; Jones, 1980; Merskey & Buhrich, 1975; C. R. Miller & Forbes, 1990; Volkmar, Poll, & Lewis, 1984; Whitlock, 1967) to convince themselves that their symptoms are caused by biological factors. As noted by Weinstein, Eck, and Lyerly (1969), patients with conversion disorder may stress "their injuries in the belief that they were etiologically significant" (p. 337; also see case studies by Brady & Lind, 1961; Grinker & Spiegel, 1945; Malamud, 1944).

Another example of self-deceptive explanations of organic nature concerns the strong relationship between panic disorder and adverse physiological sensations, which patients interpret as a sign of heart disease (e.g., Clark, 1986; Eifert, 1992; Margraf & Ehlers, 1991). Although cognitive theory views this as a genuine misinterpretation (e.g., Beck, 1988; Clark, 1986, 1988), RCTN claims that these misleading interpretations can be seen as a deliberate and rational maneuver through which patients hinder self-probing, and thereby preserve unawareness of KSI.

An additional important source that patients tend to utilize for developing or strengthening a self-deceptive explanation of illness is therapy. Patients' participation in therapy and the selection of a specific intervention may be partly motivated by the need to develop a convincing self-deceptive explanation. To these ends, a therapist may endorse the patient's deceptive self-diagnosis, and provide a wealth of "scientific" information that can facilitate the development of a self-deceptive explanation. As noted by S. Fisher and Greenberg (1977), research evidence consistently showed that

> A patient's belief interpretations and his consequent anxiety reduction do not depend on the accuracy of the interpretations. Investigators have found that individuals will enthusiastically accept bogus interpretations as accurate descriptions of their own personalities. (pp. 364; see also Grünbaum, 1984, pp. 135, 211, 265; Marmor, 1962, p. 289)

In this context, it is worth mentioning investigators' claim that DID may be produced in the therapist's office, where patients are "implicitly encourage[d]" (Spanos et al., 1985, p. 364) to develop this disorder (see also Jaroff, 1993; Read & Lindsay, 1994; Rieber, 1999). In such cases, patients also internalize psychoanalytic beliefs that the symptom is the result of repressed childhood abuse, and may confabulate abuse memories (e.g., Acocella, 1999; Lilienfeld et al., 1999; Read & Lindsay, 1994; Spanos, 1994, 1996). Consequently, when patients decide to adopt the therapists' suggestion as a coping strategy in response to an unbearable current stressful situation, they are likely to focus on the implicit belief that the other personality is the result of a "repressed" childhood trauma, thereby hindering self-probing regarding the true causes of their symptoms.

From this standpoint, it may be that the large amount of studies indicating that false memories can be implanted in patients' cognitive systems (e.g., Ceci & Loftus, 1994; Gutheil, 1993; Lindsay & Read, 1994; Loftus, 1993; Loftus & Hoffman, 1989; Mantell, 1988; Zaragoza & Lane, 1994) are not only the result of the therapists' suggestive efforts to "confirm their hunches" (Loftus, 1993, p. 530). Rather, it may well be that patients' falsification is partly the result of their self-deceptive maneuvers to preserve their state of unawareness.

Given RCTN's assumption that the self-deceptive belief serves an important psychological need, one would expect that patients would refuse to change or abandon a therapy that serves this need, even if they realize this intervention has been ineffective. Indeed, hypochondriacs are reluctant to accept referral to psychiatric services and request to be treated by physicians, even after medical intervention proved to be ineffective for their problems (see Salkovskis & Warwick, 1986; Warwick & Salkovskis, 1990; see also case study by Davison & Neale, 1986, pp. 148–150). Similarly, patients may spend years in psychoanalytic therapy, despite the lack

of therapeutic success (see Erwin, 1980; Gross, 1978). As noted by Leonard (1927), who spent several years in psychoanalytic therapy with no satisfactory results despite

> a thousand circumstances that would have robbed many men of faith . . . I have even now not lost all faith in the method, nor all faith that it may yet work out in this case. But the *fact* [emphasis added] is that it has not. (p. 414)

Self-Deceptive Belief of Denial

As stated, most patients will develop a self-deceptive belief of illness, based on the information to which they are exposed. However, how can we explain cases in which patients deny any possibility that there is something wrong with them? For example, how can we account for a patient who is anorexic who "insisted that she looked fine and that there was nothing wrong with her being so skinny" and states that "I enjoy having this disease and I want it. I cannot convince myself that I am sick and that there is anything from which I have to recover?" (Bruch, 1978, p. 2).

From RCTN's standpoint, patients who deny that their behavior is deviant, who are mainly anorexics (e.g., Gottheil, Backup, & Cornelison, 1969; Vandereycken, 2006a) and certain subjects with OCD (e.g., compulsive cleaners, see Foa, 1979; Kozak & Foa, 1994; Rachman & Hodgson, 1980), are no less logical and reality-oriented than patients in the "illness" group. As patients who develop a self-deceptive belief of illness, those who deny their illness derive this inference from external and internal evidence to which they are exposed after the creation of unawareness. The external evidence concerns the observation of socially accepted behavior (e.g., dieting and cleaning). Often, the symptom is an integral part of the individual behavioral repertoire for which he or she was even praised before its exaggeration to bizarre levels. Even after onset, the social reinforcement is likely to continue at least during the initial stage. For example, Branch and Eurman (1980) found that at first, families and friends admire sufferers of anorexia for their appearance and self-control (see also Porzelius, Berel, & Howard, 1999).

The second type of evidence that motivates patients to deny that their behavior is deviant is an internal experience of sense of control. As noted by Bruch (1978) regarding anorexia, keeping weight and body size under control provides patients with a considerable sense of achievement and gives them a life goal, which replaces the emptiness and the helplessness. Weight loss constitutes a source of self-worth and helps combat feelings of inadequacy (Garner, Vitousek, & Pike, 1997; Vitousek & Hollon, 1990). A similar view was expressed by Edelstein (1989), who noted that anorexia satisfies patients' compulsive need to master their vital drive of hunger (see

also Huebner, 1993). A sense of control may also be important in OCD, in which the symptoms provide patients with a measure for overcoming their fear of loss of control over their impulsive behaviors (e.g., Denys, de Geus, van Megen, & Westenberg, 2004; Horowitz, 2004, pp. 169–186; McAndrew, 1989). An additional internal factor that should intensify patients' tendency to deny that their behavior is deviant, to such an extent that they may even take pleasure in their behavioral change (see Bruch, 1978), is emotional relief resulting from the distractive value of the symptom. As described by a father of a patient who is anorexic, "when she is not eating and her weight is falling, she appears in good spirits" (Bruch, 1973, p. 90).

Because patients deny that their behavior is deviant, they must invent self-deceptive explanations to rationalize their abrupt behavioral change. For example, Garner (1986) noted that "the apparently bizarre eating patterns and the resolute refusal of adequate nourishment become plausible given the anorexic patient's conviction that thinness is essential for her happiness or well-being" (p. 302). Similarly, in a case study of a 14-year-old compulsive cleaner, Sherman (1938) noted that

> he realized that he washed more than other boys, but that in his case there were real reasons. He believed that his skin was of such a texture that it retained dirt and germs, and therefore was forced to wash and scrub himself. (p. 19)

In some cases, patients employ excessive praying that, like cleaning, they rationalize as a preventative measure against potential danger (e.g., accidents or terror activities; see Rofé, 1989, pp. 273–275) or view as part of a religious ritual (Rachman & Hodgson, 1980, p. 19).

Furthermore, as in the illness group, which focuses on suitable evidence to validate its self-deceptive beliefs, patients with OCD harboring false beliefs may selectively attend to evidence that seemingly supports their self-deceptive belief of denial. For example, Neale, Oltmanns, and Davison (1982, pp. 1–16) noted that a patient with a belief that something terrible would happen to her children if she did not strictly observe her bizarre compulsive counting rituals, was most often confirmed. This is because something unfortunate invariably happened to one of the children within a few days of any relaxation of her manifestation of the symptom.

> The fact that minor accidents are likely to occur at a fairly high rate in any family of four children did not diminish [her] conviction that she had been directly responsible because of her inability to observe the numerical rules. (p. 2)

Given RCTN's claim that self-deceptive belief of denial is not arbitrary, but is based on adequate information, one would expect that people with anorexia will abandon this belief in favor of a self-deceptive belief of illness when bodily conditions are seriously deteriorated. Indeed, as sup-

ported by Greenfeld, Anyan, Hobart, Quinlan, and Plantes (1991), this tendency to adopt a belief of illness is greater among patients with greatest weight loss and longest inpatient stays. Moreover, when anorexic behavior is accompanied by bulimic symptoms, there is a greater tendency to choose the strategy of illness, rather than denial (see Pryor, Johnson, Wiedrman, & Boswell, 1995). This seems to be so because binge eating is socially unacceptable and is associated with the sense of loss of control (*DSM–IV–TR*; APA, 2000).

Advocates of irrational approaches to psychopathology related to the denial of the neurotic behavior as a lack of insight reflecting reality distortion among both anorexia nervosa (e.g., see Melamed, Mester, Margolin, & Kalian, 2003; Vandereycken, 2006a, 2006b) and certain types of patients with OCD (e.g., Kozak & Foa, 1994). From the perspective of RCTN, however, the patients in "denial" are no less realistic or rational than the patients with "illnesses." To the extent that neuroses are a deliberate choice, both types of diagnosis are logical inferences from the specific external and internal observations to which patients are exposed immediately after the creation of unawareness. From this perspective, both groups of subjects have a different but equal level of insight regarding their deviant behavior. As described in Table 1, although patients with illnesses are aware of their symptomatology, they are unaware of the motivational cause (stressor) and the mechanism that controls their behavior (the self). In contrast, the denial patients are unaware of the symptomatology and cause of their symptoms, but are aware of the mechanism.

Discussion

Like psychoanalysis, RCTN maintains that the necessary condition for understanding neurosis is to clarify the mechanism that controls patients' unawareness regarding the underlying causes of their neurotic behavior. However, while psychoanalysis suggested the concept of an autonomous, unconscious entity to account for this phenomenon of unawareness, RCTN claims that the conscious controls both the manifestation of the symptom and the psychological processes by which patients become unaware of their KSI. From this theoretical standpoint, contrary to psychoanalysis, un-

Table 1. Level of Awareness in Illness Versus Denial Group

Group	Symptomatology	Cause	Mechanism
Illness	Aware	Unaware	Unaware
Denial	Unaware	Unaware	Aware

awareness plays no etiological role in the development of neuroses, as it follows, rather than precedes, the adoption of neurotic symptoms.

To some extent, RCTN's theoretical approach is similar to cognitive theory, as it too emphasizes the critical importance of patients' beliefs in the development of psychiatric disorders (e.g., see Beck, 1988; Clark, 1986, 1988). However, according to cognitive theory, the belief is irrational, stemming from impaired thinking processes that directly and automatically cause the display of the symptom (e.g., panic attack). The strength of this position is the bulk of research demonstrating the therapeutic efficacy of CBT, which supposedly re-educates patients to think rationally. The weakness of this position is its difficulty to explain why only a minority of individuals develop irrational thought patterns (e.g., Rachman, 1990) and why symptoms may occur in the absence of catastrophic cognitions (e.g., see Bouton, Mineka, & Barlow, 2001).

In contrast to the cognitive theory, RCTN claims that although patients' beliefs seem irrational, they develop through rational psychological processes, as shown earlier. According to this theory, subjects consciously and rationally choose to display the symptom, and the belief provides a deceptive rationale that prevents self-probing and thus preserves unawareness of KSI. Furthermore, once developed, the self-deceptive belief becomes an additional distractive tool that patients employ to block the accessibility of stress-related thoughts without necessarily displaying the symptom. In certain ways, the belief is more effective than the symptom itself, because it is always available and demands far less expenditure of energy.

Because the major function of the self-deceptive belief is the preservation of unawareness of KSI, it can be expected that any therapeutic intervention that disrupts this function, would motivate patients to abandon the symptom. This disruption can occur either by presenting patients with antideceptive information that invalidates the belief, or by producing curative beliefs that supposedly purify its noxious effect. As the next section demonstrates, this theoretical position enables us to integrate therapeutic efficacies of various techniques, such as CBT, religious therapy, and psychoanalysis.

THE INTEGRATIVE THERAPY MODEL OF NEUROSIS

Perhaps the most troublesome problem that challenges traditional theories of psychopathology is their inability to integrate findings relating to the success or failure of different therapeutic interventions into a single theoretical framework (e.g., see reviews, Feixas & Botella, 2004; Lampro-

poulos, 2000, 2001). In the absence of such an integrative theory, investigators in the field focused on empirical evaluations of therapeutic techniques, neglecting to address the underlying mechanism of change. Based on meta-analyses and research findings, one group of investigators concluded that all therapies have similar efficacies, and suggested that a number of common factors, such as therapeutic alliance, empathy and support, emotional catharsis, and gains in self-understanding, may be responsible for positive outcomes (e.g., see Feixas & Botella, 2004; Lampropoulos, 2000; Luborsky et al., 2002; Reisner, 2005). This approach, however, was criticized on both methodological and conceptual grounds (e.g., Chambless, 2002a; Lampropoulos, 2000, 2001). A second approach lists a number of empirically supported therapies (ESTs), claiming that they have been found to be more effective than other therapies for specific disorders (e.g., see Chambless, 2002b; Reisner, 2005).

The basic goal of every scientific paradigm is to identify and understand the mechanism of the investigated phenomenon. As such, psychopathology cannot give up the ultimate goal of identifying the mechanism of therapeutic change. Indeed, a third approach strongly criticizes the ESTs on methodological grounds, theoretical basis, and for a lack of genuine therapeutic improvement (e.g., Herbert, 2003; Tryon, 2005; Westen, Novotny, & Thompson-Brenner, 2004). Proponents of this approach claim that the central task of psychotherapy should be clarifying the principles and mechanisms underlying the therapeutic changes. As noted by Rosen and Davison (2003), psychotherapy "should work toward the identification of empirically supported principles of change" (p. 303), rather than focusing on empirical evaluations of effective therapies. In the same way, Tryon (2005) noted that "[the] effectiveness of efforts to persuade clinicians to adopt ESTs may depend substantially on the extent to which science can explain why ESTs work and thereby provide clinicians with an empirically supported explanatory context in addition to effective interventions" (p. 68). Indeed, it would be unfortunate if psychotherapy were to remain merely a collection of therapeutic techniques without striving to identify the underlying mechanism of therapeutic change. As noted by Power and Brewin (1997),

> Some therapists simply bury their heads in the sand and continue with their favorite techniques. Others, like ourselves, are puzzled and are asking if there are common mechanisms that apply across different therapies that might explain both their effectiveness and their (sometimes) lack of effectiveness? (p. xi)

The critical importance of this issue is acknowledged by various theories of psychopathology, and "the absence of empirical support about these theories . . . does not diminish the need for an explanatory context" (Tryon, 2005, p. 68). RCTN offers such an explanatory platform for the differential efficacy of interventions in the realm of neurosis. As shown

below, the available therapeutic interventions that emerge from traditional theories, such as CBT, punishment-reinforcement therapy (PRT), psychoanalysis, and drug therapy, achieve their therapeutic success by affecting the same mechanisms that, according to RCTN, maintain the neurotic symptoms.

CBT

Cognitive theory of anxiety disorders such as OCD (e.g., Salkovskis, 1999; Taylor, 2002) and panic disorder with agoraphobia (e.g., Austin & Richards, 2001; Beck, 1988; Clark, 1986, 1988, 1996; Taylor, 2000), attributes the cause of these disorders to irrational thinking, whereby patients catastrophically misinterpret harmless stimuli, such as bodily sensations in panic disorder and dirt in OCD. CBT treats these disorders by confronting patients with evidence that is inconsistent with their belief, either through in vivo exposure or cognitive information, which supposedly educates patients to interpret the stimuli rationally (e.g., Beck, 1988; Clark, 1988; Gelder, Clark, & Salkovskis, 1993; Hoffart, 1993; Salkovskis, Hackmann, Wells, Gelder, & Clark, 2006; Taylor, 2000). In accordance with this approach, investigators found that reducing the patients' dysfunctional beliefs or strengthening their perceived self-efficacy in coping with the symptom resulted in effective therapeutic outcome with these disorders (e.g., Bouchard et al., 2007; Casey, Oei, & Newcombe, 2005; Emmelkamp, 2002; Emmelkamp, van Oppen, & van Balkom, 2002; Salkovskis et al., 2006).

Although patients with neurotic disorder manifest beliefs that seem irrational, these cognitions do not necessarily reflect any inherent deficit in their thought processes. As shown before, according to RCTN, these cognitions are a self-deceptive maneuver by which patients preserve their unawareness. Therefore, CBT does not reeducate patients to relate to harmless stimuli in a rational manner, but rather this mode of therapy can be seen as a sophisticated maneuver designed to sabotage the patients' self-deceptive belief. Patients are well aware prior to their neurosis that these stimuli are harmless, and their cognitive system remains intact after the development of the symptoms. Once they are re-exposed to a stimulus that challenges their self-deceptive beliefs (e.g., dirt or adverse bodily sensations), patients must interpret it as catastrophic and display their symptom. Failure to do so will sabotage their self-deceptive belief. CBT exposes patients to the belief-challenging stimuli, but either prevents the presentation of the symptom (e.g., prevention of washing following exposure to dirt) or increases patients' awareness of the senseless nature of their

neurotic responses (e.g., exposure to CO_2 in panic disorder). This evidence sabotages self-deceptive beliefs and motivates patients to abandon the symptom, as they lose the rationale that enables them to be unaware of KSI in subsequent presentations of the symptom. Thus, from RCTN's standpoint, CBT is a therapeutic intervention that compels the patients through rational means to abandon their pathological coping mechanism.

Given RCTN's assumption that neurotic symptoms are adopted to ward off intolerable levels of stress, patients are reluctant to abandon the symptom as long as this stress persists. Accordingly, evidence shows that the dropout rates among panic disorder and agoraphobia patients may reach up to 20% to 38% (e.g., Arthus, Cottraux, Mollard, Guerin, & Bouvard, 1997; Barlow, Gorman, Shear & Woods, 2000; Clum, 1989; Keijsers, Kampman, & Hoogduin, 2001). Similar findings were found with patients with OCD (e.g., Abramowitz, 2006; O'Conner, 2005; Schruers, Koning, Haack, Luermans, & Griez, 2005; Stanley & Turner, 1995; Tundo, Salvati, Busto, Di-Spigno, & Falcini, 2007). Although the dropout rates were attributed to reasons such as financial difficulties, time commitments, and schedule difficulties (see Taylor, 2000, pp. 187–188), the possibility remains that these "reasons" are mere excuses to rationalize the termination of therapy. Another indication of resistance is when patients conjure deceptive excuses to ward off the therapist's effort to disarm them of their coping mechanism. For example, following exposure, patients with agoraphobia (e.g., Hoffart, 1993) and OCD (e.g., Foa, 1979) may attribute the incompatible evidence of not being afraid of their anxiety-provoking stimuli to the presence of supporting figures (e.g., therapist or relative). Therefore, formal therapy usually includes unaccompanied exposure as well (e.g., see Hafner, 1983; Hoffart, 1993; Ollendick, 1995). Sometimes, however, the patient invents a self-deceptive excuse that is more difficult to challenge directly. For example, Hoffart (1993) described a patient with agoraphobia who successfully exposed himself to various parts of the city without experiencing anxiety, yet maintained his deceptive self-diagnosis of illness by attributing the contradictory evidence to chance. Hoffart concluded that "in the future, he would be just as likely as before to experience overwhelming panic attacks in similar situations" (p. 85).

Although CBT is an effective intervention, its efficacy is limited. Many patients fail to respond to therapy, seek further therapy, develop problems unrelated to the symptom, or undergo relapse (e.g., Brown & Barlow, 1995; Hafner, 1976; Hoffart, 1993; O'Kearney, 1993; O'Sullivan & Marks, 1991). Moreover, only a small percentage (21–27%) of those who completed therapy demonstrated high-end state functioning, defined as a clinical cure or functioning within a normative range (e.g., P. L. Fisher & Wells, 2005; N. S. Jacobson, Wilson, & Tupper, 1988). This limited improvement may be much lower in light of Westen et al.'s (2004) critique, especially con-

sidering their claim that subjects in EST studies were not randomly assigned to different experimental groups. Instead, patients were "screened to maximize homogeneity of diagnosis and minimize co-occurring conditions that could increase variability of treatment response" (p. 632). From RCTN's standpoint, the limited efficacy of CBT stems from the therapy's failure to address the underlying stressor that motivated the development of the neurotic symptom. Indeed, studies have shown that coping skills training, which increase the patient's ability to deal with the initial stressor, enhances the efficacy of CBT (e.g., Biran, 1990; Chambless, Goldstein, Gallagher, & Bright, 1986; Chernen & Friedman, 1993; Craske, Burton, & Barlow, 1989; Hiss, Foa, & Kozak, 1994; O'Kearney, 1993; Richards, Klein, & Austin, 2006).

In conclusion, in line with cognitive theory, RCTN views patients' belief as a crucial factor in maintaining the neurotic symptoms, such as panic disorder and OCD. However, contrary to the cognitive model, the belief does not reflect irrational thinking. Instead it constitutes part of patients' cognitive maneuvers in maintaining the unawareness of KSI. Accordingly, although cognitive theory maintains that CBT re-educates patients to think rationally, RCTN claims that this intervention merely sabotages patients' self-deceptive beliefs.

PRT

According to RCTN's rational approach, neurotic symptoms are adopted based on a cost-benefit analysis. Therefore, it is expected that punishment-reinforcement therapy (PRT), which increase the cost of the symptom or reinforce patients for normal behavior, will increase the tendency to abandon the symptom. Indeed, research on both conversion disorder (e.g., Blanchard & Hersen, 1976; Donohue, Thevenin & Runyon, 1997; Goldblatt & Munitz, 1976; Gooch, Wolcott, & Speed, 1997; Mizes, 1985; Teasell & Shapiro, 1994) and anorexia nervosa (e.g., Bemis, 1987; Blue, 1979; Okamoto et al., 2002; Touyz, Beumont, Glaun, Phillips, & Cowie, 1984) has shown that behavioral therapy, which either removes certain benefits associated with the symptom (e.g., withdrawal of attention, monetary rewards, or hospital privileges) or increases its cost through punishments (i.e., electric shock and bed confinement), facilitates positive therapeutic outcomes.

The efficacy of PRT is demonstrated through a case study by Tucher and Long (1985), in which a 22-year-old soldier developed hysterical blindness in one eye after experiencing difficulty in adjusting to military stress. The behavioral treatment consisted of verbal reinforcements for the

patient's reports of visual improvement, and aversive consequences that included withholding hospital privileges (e.g., time off ward, use of civilian clothes, etc.) until the subject's vision improved. The patient was also given a rationale for improvement when instructed to wear an eye-patch over his good left eye to "exercise and train" his defective right eye. The subject was told that most people with visual problems improve within 3 days when wearing an eye patch, with complete recovery by the fifth day. The patient reported almost normal vision on the fifth day of treatment and remained so at the 3-day follow-up.

From RCTN's standpoint, PRT can be seen as a symptomatic intervention, which does not normally address the underlying stressor that motivated and maintained the symptom. Therefore, to the extent that patients lack suitable coping mechanisms, this therapeutic intervention may lead to symptom substitution or relapse, as shown in both conversion disorder (e.g., Blanchard & Hersen, 1976) and anorexia nervosa (e.g., see Wilson, 1999). However, these adverse therapeutic effects may be prevented if patients are given coping skills training. For example, Blanchard and Hersen (1976) reported that extinguishing the utility value of conversion symptoms (e.g., sympathy and attention) resulted in symptom substitution in patients who encountered new stressful situations. Effective and long-lasting therapy occurred only when the extinction procedure was accompanied by coping skills training. In this regard, the authors noted,

> It appears that because of a faulty learning history this type of patient has not developed the requisite social skills needed to cope with both the usual and more unusual stresses encountered in life. Therefore, training or retraining in social skills becomes a needed ingredient in a comprehensive therapeutic regime. (p. 127)

In conclusion, the utility of punishment-reinforcement therapy emerged out of the behaviorist experimental studies with animals. However, while learning concepts seem adequate for addressing behavioral changes in animals, it seems that the efficacy of this method with neurotic patients can be equally accounted for by RCTN's cost-benefit principle.

Religious Therapy

As stated, the self-deceptive belief preserves the unawareness of KSI, thereby enabling patients to benefit from the distractive value of both the symptom and the belief. When the symptom is associated with a specific stimulus in reality (e.g., dirt in OCD), the best therapeutic intervention for disconfirming the belief is CBT. However, when patients attribute their symptoms to supernatural forces, the therapy of choice should be a religious figure, such as a rabbi, priest, or shaman (e.g., see Hoffman, Laub, &

Zim, 1990; Kua et al., 1986; Seltzer, 1983; Wijesinghe et al., 1976; Witztum, Grisaru, & Budowski, 1996). According to RCTN, a religious therapist produces a counterbelief (e.g., when the symptom is attributed to an evil spirit, the therapist conducts certain religious rituals that expel this force), which sabotages patients' ability to employ the belief for preventing awareness of KSI. For example, Pattison and Wintrob (1981) reported a case of a young Mexican woman who complained of nightmares and nightly visitations from her fiancée following his death, and believed she was possessed. In the treatment session, the therapist invited the fiancée's ghost into the house and explained to it that, being dead, it had to leave the patient alone and find an appropriate place for the dead. The ghost was then dismissed out the door and the patient's symptoms subsided rapidly. Similarly, Csordas (1987), describing a number of successful religious therapy cases, mentioned a case of a university professor who suddenly developed dissociative fugue, claiming spiritual possession, following a conflict with her husband. Although she had spent 2 years in psychoanalysis prior to the symptom, she refused psychiatric treatment and demanded religious therapy. The psychiatrist, at a loss, advised her to seek a prominent member of a certain cult in Brazil, and, on filling the cult's religious obligations, she completely recovered from her symptoms.

In another example of Western therapists utilizing the services of religious figures to facilitate the therapeutic process, Hoffman et al. (1990) described three case studies in which therapists enlisted the services of a rabbi, resulting in a successful therapeutic outcome for religiously observant patients. In one particular case, a 14-year-old Orthodox Jewish boy had suffered the loss of a friend and was subsequently preoccupied with thoughts of her spirit coming to harm him. The rabbi succeeded in relieving the patient's emotional difficulties by denying the existence of spirits in the Holy Land and by encouraging the patient to say a special prayer every day, "in order to thwart unwanted and frightening thoughts and imaginings" (p. 181).

Psychoanalysis

According to classic psychoanalysis, true recovery from neurosis necessitates the lifting of repression, that is, gaining insight into the underlying causes of deviant behaviors (e.g., Bergmann, 1992; Blum, 2003; Breuer & Freud, 1895; Eagle, 2000). However, studies challenge the existence of both Freudian repression (e.g., McNally, 2003; Piper et al., 2000; Rofé, 2008) and the unconscious (e.g., Greenwald, 1992; Kihlstrom, 2000, 2002, 2004; Kihlstrom, Barnhardt, &Tataryn, 1992; G. O'Brien & Jureidini, 2002;

Rofé, 2008). Hence, although psychoanalysis was found to be an effective intervention (e.g., Grant & Sandell, 2004; Leichsenring, 2005; Leuzinger-Bohleber, Stuhr, Ruger, & Beutel, 2003), this efficacy is probably the result of factors other than repression or the unconscious. The same conclusion is applicable to psychodynamic interventions, which were found to be effective for neuroses such as eating disorders (e.g., see review Fonagy, Roth, & Higgitt, 2005), panic disorder (Milrod et al., 2007), and DID (e.g., Kihlstrom, 2005; Kluft, 1995). A similar position was expressed by Grünbaum (1984, 1986, 2007) and Jopling (2001) who attributed the efficacy of psychoanalytic/psychodynamic therapy to a placebo effect.

From RCTN's perspective, there seem to be two main reasons for the efficacy of psychoanalytic interventions. The first relates to cases where therapy leads patients into believing that their symptom is the consequence of repressed childhood traumas. Although in some cases the repressed trauma was confirmed by independent sources (e.g., see Brenneis, 2000; Cheit, 1998; Kluft, 1995; Martinez-Taboas, 1996), most investigators question the authenticity of these supposedly forgotten traumas (e.g., Gardner, 2004; Kaplan & Manicavasagar, 2001; Lynn, Lock, Loftus, Krackow, & Lilienfeld, 2003; Pope & Hudson, 1995; Pope, Oliva, & Hudson, 1999). Moreover, there are no empirical studies to confirm that the therapeutic efficacy of psychoanalysis results from lifting of repression (see review by Rofé, 2008). Hence, it is difficult to accept the psychoanalytic explanation for the effectiveness of their therapeutic method. An alternative explanation is that psychoanalytic therapy in such cases is comparable with religious therapy. A psychoanalyst who is endowed with the reputation of being capable of undoing the noxious effects of the unconscious can lead a patient to believe that he is now aware of these evil forces and can therefore function normally. This claim may be applicable to DID, which is viewed by the sociocognitive model as role playing (e.g., Lilienfeld et al., 1999; Spanos, 1994, 1996), and as a Western form of spiritual possession (see Spanos, 1994, 1996). Considering Spanos et al.'s (1985; see also Spanos et al., 1986) suggestion that patients with DID implicitly conceptualize their symptoms as a reflection of unconscious forces, it seems that just as with religious therapy, psychodynamic interventions are successful with these patients (e.g., Boyd, 1997; Coons, 1994; Kluft, 1995; Putnam & Loewenstein, 1993; Waiess, 2006) mainly because they succeed in sabotaging patients' self-deceptive beliefs. Because patients believe in the utility of psychoanalytic therapy, the psychoanalytic ritual, conducted by a prestigious therapist, produces a new belief, that the patient was cured of the noxious components of the unconscious. Consequently, because patients can no longer rationalize the symptom by attributing it to forces beyond their conscious control, they are inclined to abandon it. This claim is consistent with Sampson's (1992) statement that analysts' attitudes are

important to the therapeutic process, to the extent that they disconfirm the pathogenic belief of the patient.

> If the patient unconsciously perceives the analyst's behavior and attitudes as disconfirming the belief he is testing, the patient will make progress. In this way, direct experiences with the analyst sometimes may lead to significant analytic progress even without interpretation. (pp. 519–520; see also Spanos, 1996; Spanos et al., 1985)

Accordingly, "even admittedly inexact interpretations have been noted to be of therapeutic value!" (Marmor, 1962, p. 289).

The second reason for the efficacy of psychodynamic interventions is that in many cases, psychoanalytic therapists make patients aware of their current stressors or intrapsychic conflicts (e.g., see Jenike, 1998; Milrod et al., 2007; Wolitzky & Eagle, 1999). Taking into account RCTN's position that the primary function of neuroses is to ward off/repress stress-related thoughts from attention, awareness of the stressor would diminish the repressive value of the symptom, and thus motivate patients to reconsider the continued maintenance of their deviant behavior. The abandonment of the symptom may aggravate patients' depression and result in relapse if the original stressor remains at the same intensity level. A genuine recovery should occur when the stressor is removed, or when the patient is capable of resolving the problem with the therapist's support and encouragement.

A clinical example demonstrating this theoretical position can be found in a successful psychoanalytic intervention with a 28-year-old married woman suffering from agoraphobia (Wolitzky & Eagle, 1999). The patient's husband had taken a new job about 50 miles from the city where the couple and her parents lived. The couple planned to move to another city and thus sold their old apartment and temporarily moved with their 3-month-old baby into her parent's house. The patient's mother suffered from kidney disease, and from the age of eight it had been her job to change the filters for her mother's dialysis machine. According to the authors, "the anticipated move to the new city appeared to precipitate the outbreak of agoraphobic symptoms" (p. 209). At the initial stage of therapy, the husband agreed to change his plan and seek a job in the city where they lived. Although this caused some emotional relief, it did not affect the patient's agoraphobic symptoms, which persisted at the same intensity. The symptoms gradually disappeared when the patient became aware of her conflict pertaining to separation from her sick mother. Soon afterward, the patient moved with her husband and their baby into a new apartment across the street from her parent's house. A follow up of 5 years showed that the patient remained symptom free.

From RCTN's standpoint, the patient's agoraphobic symptoms fulfilled two psychological functions: (1) The husband's decision to move to a new city exacerbated the patient's emotional distress and the symptoms

constituted a rational coping means that compelled him to reconsider his decision and (2) the symptoms intensively preoccupied the patient's attention, thus enabling her to become unaware of her intrapsychic conflict regarding the separation from her mother. Once the therapy succeeded in generating insight into this conflict, the symptom lost its repressive value and motivated the patient to eventually abandon it. Awareness, however, did not change the patient's apprehension that separation might endanger her mother's life. Nevertheless, this did not aggravate the patient's emotional distress, and relapse did not occur because she resolved this problem optimally by separating from her mother, while staying in the close vicinity.

Drug Therapy

RCTN's main thesis is that patients are motivated to adopt and maintain the neurotic symptom mainly because its powerful distractive value alleviates the level of emotional distress, resulting from stressful life events or intrapsychic conflicts (Rofé, 2000, 2010). Accordingly, it is expected that therapeutic interventions that reduce patients' emotional distress, such as drug therapy, would yield positive therapeutic outcomes. Further, given RCTN's assumption that stress is the motivational cause of neurosis, one would also expect that the discontinuation of medication would increase the risk of relapse. In addition, because CBT appears to be the most effective intervention for sabotaging patients' self-deceptive beliefs in anxiety disorders, a combined treatment of drug therapy and CBT should yield better therapeutic results than drug therapy alone. Finally, as RCTN postulates that neurotic disorders have a similar etiology, the same drug should be effective for a variety of neurotic disorders. In support of these hypotheses, studies have shown that:

1. Antidepressants have beneficial effects on nearly all neurotic disorders, including panic disorder and agoraphobia (e.g., Furukawa, Watanabe, & Churchill, 2006; Mavissakalian & Ryan, 1998; Westenberg, 1996), OCD (e.g., Abramowitz, 1997; Cottraux, Bouvard, & Maud, 2005; Dell'Osso, Nestadt, Allen, & Hollander, 2006; Dougherty, Rauch, & Jenike, 2004), DID (e.g., D. D. Miller, 2000; Putnam & Loewenstein, 1993), eating disorders (e.g., Bacaltchuk, Hay, & Mari, 2000; Ferguson, & Pigott, 2000; Vaswani, Ramesh, Kalra, & Sagar, 2005), and conversion disorder (e.g., Pu, Mohamed, Imam, & El-Roey, 1986; Voon, & Lang, 2005).
2. Discontinuation of antidepressants (e.g., Doyle & Pollack, 2004; Mavissakalian & Guo, 2004; Mavissakalian & Ryan, 1998) or antipanic medication (see review Taylor, 2000) increases the risk of

relapse among patients with panic and agoraphobic disorders. Similar effects were found regarding the discontinuation of drug therapy for patients with OCD (e.g., Bystritsky, 2004; Koran, Bullock, Hartston, Elliot, & D'Andrea, 2002; Marks & O'Sullivan, 1988; Pato, Murphy, & DeVane, 1991; Ravizza et al., 1998), and patients with bulimia disorder (see Kotler & Walsh, 2000). In contrast, continuous use of antidepressant medication among patients with panic disorder (Mavissakalian & Perel, 1999), OCD (e.g., Fineberg, Pampaloni, Pallanti, Ipser, & Stein, 2007; Ravizza et al., 1998), and bulimia (e.g., Kotler & Walsh, 2000; Peterson & Mitchell, 1999) reduced the risk of relapse.

3. Some evidence seems to indicate that a combination of antidepressants and CBT has superior results in treating panic disorder and agoraphobia than each therapy alone (e.g., Bakker, van Balkom, & van Dyck, 2000; Barlow et al., 2000; Feusner, Cameron, & Bystritsky, 2005; Hajjar, 1989; Marchesi, Cantoni, Fonto, Giannelli, & Maggini, 2006; Weissman, 2002). Similar findings were obtained for OCD (e.g., Cottraux et al., 2005; Franklin & Simpson, 2005) and for bulimia (see Kotler & Walsh, 2000; Peterson & Mitchell, 1999). However, further research is needed to clarify this issue because some studies show that the combination of medication and CBT is equally effective as CBT alone for the aforementioned disorders (e.g., Foa & Franklin, 1999; Furukawa et al., 2006; Kaye, Klump, Frank, & Strober, 2000; Mitte, 2005; van Oppen, van Balkom, de Haan, & van Dyck, 2005; Wilson, 2005).
4. A variety of studies have shown that the same drug may be effective for disorders of different symptomology. For example, clomipramine, fluoxetine, fluvoxamine, monoamine oxidase inhibitors, and tricyclic antidepressants are effective in the treatment of both OCD (e.g., Abramowitz, 1997; Demal, Zitterl, Lenz, Zapotoczky, & Zitterl-Eglseer, 1996; Park, Jefferson, & Greist, 1997; Piccinelli, Pini, Bellantuono, & Wilkinson, 1995; Van Balkom et al., 1994) and agoraphobia and panic disorders (e.g., Alexander, 1991; Black, Wesner, Bowers, & Gabel, 1993; Burrows, Judd, & Norman, 1993; Gelder, 1992; Klerman, 1992; Westenberg, 1996). Similar results were reported for eating disorders (e.g., Hoffman & Halmi, 1993; Wolfe, 1995).

In conclusion, drug therapy should not be seen as an intervention that uproots the source of patients' "illness." Rather, this therapy mainly reduces patients' emotional distress, and therefore, the discontinuation of medication increases the risk of relapse. This theoretical position is also strengthened by the fact that the same single pharmacological agent is

effective for several neuroses. RCTN can also account for why a combination of drug therapy and CBT is more effective than each therapy by itself.

Placebo Effects

Occasionally, the sheer participation in a therapeutic intervention results in a positive outcome. For example, Carl Gustav Jung (1963) informed a middle-aged female patient, who had been suffering from a painful paralyzed leg for 17 years, that he was going to hypnotize her. Without any hypnotic manipulation, the patient immediately fell into a deep trance and talked without pause for over a half hour, resisting Jung's attempts to awaken her. Soon afterward she declared herself cured, threw away her crutches and proceeded to walk. Jung, whose theoretical framework could not account for this unexpected result, announced to his students, "Now you've seen what can be done with hypnosis" (p. 120). However, he noted that "in fact I had not the slightest idea what had happened" (p. 120). From RCTN's standpoint, it seems likely that the patient wanted to abandon her symptom even before she sought treatment. However, she felt uncomfortable to do so after so many years, and resolved this problem through a pseudotreatment intervention by a prestigious therapist. This provided her with a rationale (i.e., a self-deceptive excuse), which justified the behavioral change and prevented the aversive feelings of cognitive dissonance and social embarrassment that she might have experienced by spontaneous remission. As noted by Ullmann and Krasner (1975):

> Once the person has made the act, and performed the new role, he cannot shift back into his prior role . . . . The person may continue to play the role because not doing so would cast doubt on his prior behavior in that role. *This is one reason why a placebo may be effective: It gives the person an "out," saves face and avoids many of the aversive consequences in "giving up" the "sick" behavior* [emphasis added]. (p. 256; see also Mooney & Gurrister, 2004)

RCTN's Integrative Therapeutic Model

Based on the conviction that neurotic disorders are the consequence of factors beyond the individual's conscious control, traditional theories of psychopathology believed that, like the treatment of a physical illness, therapeutic intervention has a curative effect in the sense that it eradicates the underlying cause of the deviant behavior. Although different schools of psychopathology disagree regarding the direct cause of the deviant behav-

ior, all agree that patients have some constitutional psychophysiological deficit which therapies are able to cure. Psychological treatment resembles a biological intervention that destroys a given virus or repairs a dysfunctional organ. Accordingly, psychoanalysis "purifies" the unconscious of some maladaptive, repressed trauma, behavioral therapy extinguishes unfortunate conditioned responses, cognitivists re-educate patients to think rationally, and biological interventions repair some adverse neurochemical changes. In contrast, given RCTN's claim that the neurotic symptom is a pathological coping mechanism that individuals consciously and deliberately adopt when stress exceeds their normal coping abilities, the goal of therapy is to produce appropriate psychological conditions that will motivate patients to abandon their symptom. The optimal psychological state is removal of stress, either directly or indirectly, through the reinforcement of patients' coping skills. For example, Wolpe (1982, pp. 286–287) reported a case of a 26-year-old agoraphobic female, whose symptom disappeared when she left her husband (the stressor) for a month to be with another man, and reappeared when she returned to her husband. When it became clear that the marriage could not be saved, therapeutic efforts were directed at enabling a separation from her husband while simultaneously strengthening her coping skills. When the couple eventually separated, her symptoms completely disappeared, and she remained symptom-free after a 3-year follow-up. It appears that the fluctuation of the symptom depended on the patient's contact with the stressor and was permanently abandoned when she was removed from the stressful situation and was able to function independently.

As already demonstrated, patients can also be motivated to abandon the symptom by affecting those components that, according to RCTN, maintain the neurotic behavior. From this theoretical perspective, available therapeutic techniques can be classified into five major categories.

Sabotaging the Self-Deceptive Belief

As previously theorized, certain techniques, including CBT, religious therapy, and psychoanalysis, obtain their therapeutic efficacy by sabotaging the self-deceptive belief. The efficacy of a given technique depends on the nature of the symptom and the type of belief. CBT may be the most direct form of sabotaging the patient's belief in cases where the symptom is activated by specific stimuli, such as in anxiety disorders. CBT invalidates the self-deceptive belief by confronting patients with inconsistent evidence, such as exposure therapy with response prevention. Psychoanalysis and religious therapy seem to be more useful in sabotaging the belief when the

symptom is implicitly or explicitly attributed to the unconscious (e.g., DID) or supernatural forces (e.g., spiritual possession), respectively. As a rule, the greater the perceived healing power of the therapist, the greater the patient's tendency to modify his or her self-deceptive belief, and hence, to abandon the symptom.

Disruption of Cost-Benefit Equilibrium

In some cases, it is difficult to sabotage the patient's self-deceptive belief. As discussed in earlier in this article, anorexics develop a self-deceptive belief of denial based on their observation of socially accepted behavior (i.e., dieting) and the experience of emotional relief resulting from the distractive value of the symptom. Accordingly, because it is difficult to disprove the patients' strategy of denial, they can be motivated to abandon their symptoms through PRT, which either increases the cost of these behaviors, or reinforces normal functioning. As previously noted, this therapeutic intervention was also shown to be effective in specific conversion disorders, where it is difficult to disprove patients' belief that their symptoms are the consequence of physiological damage.

Drug Therapy

Given RCTN's assumption that neurosis is aimed at reducing patients' intolerable levels of emotional distress, drug therapy, when administered continuously, should motivate them to abandon their symptom. The risk of relapse here may be higher than procedures that sabotage self-deceptive beliefs or PRT. This is because aside from the reduction of emotional distress, which returns to its original level once the drug is discontinued, it does not affect any of the components that, according to RCTN, maintain the symptom.

Lifting of Repression

This type of interventions concerns psychodynamic therapies that increase patients' awareness of their current stressors, thereby lifting/nullifying the repressive/distractive value of the symptom (e.g., see Jenike, 1998; Milrod et al., 2007; Wolitzky & Eagle, 1999). Such insight diminishes the distractive value of the symptom, and thus increases patients' tendency to abandon the behavior. Lifting of repression may yield better long-term

outcomes than the aforementioned interventions because, as claimed by psychoanalytic advocates (e.g., Westen et al., 2004), patients become aware of their stressor that enables therapists to work with the patients toward resolving their psychological problems in a more adoptive way.

Rational Insight Therapy

As stated, unawareness of KSI is crucial for maintaining the symptom because without it, the symptom would lose its distractive value. Hence, a therapy can also succeed in eradicating the symptom by increasing patients' awareness to KSI. In some exceptional cases, such an intervention may yield positive outcomes even at the initial stage of therapy. For example, Symonds (as cited in Merskey, 1979) treated cases of fugue by telling patients, "I know from experience that your pretended loss of memory is the result of some intolerable emotional situation" (pp. 264–265). The therapist further stated that if the patient would tell the whole story he would respect the patient's confidence, even to the point of telling the patient's doctor and relatives that he or she has been cured by hypnotism. Symonds reported that all of his patients admitted to having faked their symptoms, that is, they lost their pathological coping strategy. Obviously, such an intervention may lead to strong resistance by the patient, who is reluctant to share the information he concealed even from himself. It may be that such a therapeutic intervention would be effective at an advanced stage of therapy when therapeutic trust and good rapport have already been established. Research, therefore, is needed to examine this possibility.

A Case Example

John was a 14½-year-old boy who was referred to therapy by his mother. She was concerned about her son who displayed obsessive–compulsive symptoms over a period of 6 months. These included cleaning, checking, and praying rituals. John also displayed excessive bossiness and parent-like behavior toward his two younger brothers, aged 8½ and 11 years old.

The father, a truck driver who received only elementary school education, firmly objected to his wife's current studies at the university. Despite several attempts made by the therapist to encourage the father's participation, he adamantly refused to come to the therapist's office, which was located on campus. The father considered leaving his family, and a few months after John began treatment, he left home and began divorce

proceedings. The mother revealed that John was often subjected to criticism and punishments, particularly by his father. She also reported that both she and the father doted on the middle child, who was more talented than John. There was also tension between John, who had his own bedroom, and his two brothers, who shared a bedroom. In addition, John began attending a new school, and consequently, his schoolwork started to deteriorate. John reacted to these stressors by displaying violent behavior, which according to his mother, culminated in one aggressive incident in which John beat up a neighborhood child so badly that he needed to be hospitalized. Shortly afterward, John started displaying OCD symptoms. Throughout the intake, John appeared highly defensive, and denied that anything was wrong with him. For example, when he was asked about his excessive praying rituals, he replied, noting that the therapist himself was an observant Jew, "You know how bad the situation is in Israel. I have to protect myself and my family from the Arab terrorists! Don't you pray as well? Don't you believe in God?"

Applying the RCTN theoretical framework, it seems that the underlying cause of John's symptoms was stress pertaining to family conflicts and school. This stress generated or exacerbated his aggressive tendencies to the extent that they were difficult to control, rendering him afraid of losing control. The fear of losing control became acute after the hostile incident involving the neighborhood child and it became so intolerable that John needed to adopt OCD symptoms to regain some measure of control. Additionally, the OCD symptoms provided John with a distractive tool to block the accessibility of stress-related thoughts, thereby relieving his intolerable tension.

The first five therapeutic sessions with John consisted of two main components. First, John was given training in progressive muscle relaxation (E. Jacobson, 1938), which he practiced for at least 30 min of each session. He was also asked to practice this technique in a quiet place at home for 45 min every day. Second, John was asked during the third session to vocally repeat the following sentences:

> From now on I will be relaxed and think only positive thoughts about myself.
>
> I must also stop all strange behaviors.
>
> These responses do not help me resolve my problems and only make my mother sad.

He was then given a sheet of paper on which these sentences were written, and was asked to continue the relaxing exercise on a daily basis, to be followed by rehearsal of the sentences. At the end of the session, the therapist warmly praised John for any progress he made, as he did every session. The relaxation training and the suggestions had several aims:

(1) They reinforced John's coping skills by relieving his emotional distress; (2) they enabled the therapist to warmly praise John for his performance, thereby inducing conditions for establishing a good rapport; and (3) they provided a measure of diagnostic assessment of whether common factors relating to the patient-therapist relationship were by themselves sufficient to induce therapeutic change. If that were the case, it would not have been necessary to address the RCTN factors that maintain the neurotic symptoms.

At the beginning of the fourth session, the mother noticed a temporary improvement, but noted that John resumed his old behavior after a few days, and even developed new rituals. Therefore, after developing a good rapport and strengthening the patient's coping skills, therapy began to focus on the two major components, which according to RCTN, maintain John's OCD symptoms. The first therapeutic goal concerned the reduction of environmental stressors associated with both the family and school. The second goal involved getting John to realize that underlying his OCD symptoms was the fear of losing control.

Accordingly, during the next sessions (6–14), John's mother was made aware of the stressors that he was subjected to. These included the fact that he has been denied his status of being the eldest, that he was continuously criticized, and that his two brothers collaborated against him. The normal difficulties of adolescence, together with stress connected with his school transfer, were also emphasized. The mother was encouraged to be less critical and demanding, while displaying more affection toward John. The importance of making a gradual shift in her behavior and not "overdoing it" was explained. Furthermore, in an attempt to weaken the coalition between the two younger brothers, she was advised to consider the possibility of separating the two siblings into different bedrooms, which she readily implemented after 2 weeks.

To improve the relationship between John and his siblings, family meetings were arranged, in which the mother and all three children participated. In these sessions, the distress that the children were causing to each other, and particularly to the mother, was addressed. An agreement was made that John would stop displaying bossy behavior toward the two brothers, who in turn, promised to be friendlier toward him. To address the stress originating from the new school setting, private tutors were arranged to deal with John's difficulty with the new academic requirements, and subsequently, his academic achievements improved. This also increased his feeling of control, as he felt that he could change his own situation and improve it. In addition, the school principal was informed of the hard times John was experiencing, and was asked to be more considerate. As a result of these interventions, John's school performance was further enhanced, he

felt better at home and there was some improvement in the OCD rituals. However, his symptoms still prevailed.

At the eleventh session, therapy focused directly on making John aware of the underlying causes for his symptom adoption, that is, "lifting of repression." As elaborated above, RCTN maintains that once a patient becomes aware that he deliberately employed the symptom as a pathological coping mechanism, this behavior becomes nonbeneficial and thus he is motivated to abandon it. Accordingly, John was led to be aware of his strong aggressive impulses, and that his OCD symptoms were aimed at increasing control over his aggressive tendencies and to solicit sympathy and attention from his parents. He denied this possibility, and no further attempt was made to sabotage his pathological defensive measures.

In this session, however, fear of aggression became the central issue. John was told his fear is perfectly normal, but that the way he chooses to handle it is inappropriate. He was encouraged to discuss this problem with his mother and express his fear of anger verbally. John continued to deny that he had any fear of aggression. He was then asked to recall events that occurred during the past year in which he was exceptionally aggressive, but he could not recall such an event. With John's permission, his mother was then called into the room and was asked to remind him about the incident regarding the neighborhood child. This had a dramatic effect on John: He burst out crying "Nobody loves me, I don't want to go back home." This authentic reaction reflected John's anxiety that he would not be able to cope with his feelings following awareness of the underlying causes of his symptoms, a knowledge that reduced their distractive and control-giving values. Despite this aggressive emotional response, both therapist and mother expressed genuine empathy and support, and John gradually calmed down. The therapist then reviewed with John how he focused on his problems in a self-damaging way. He was warmly praised for the progress he made and was assured that he had the strength to cope with his difficulties in an efficient and productive manner. Thus, from RCTN's standpoint, the process of "lifting of repression" nullified the coping utility of the obsessive symptoms and enabled the therapist to motivate John to adopt a more productive manner of dealing with his stressors.

In a telephone conversation the next day, the mother related that on the way home, John was very sad and solemn. She bought him his favorite treat and tried to cheer him up. At home he behaved quite normally and without any symptoms. The therapist asked John to come to the telephone and praised him for the improvement in his behavior. Therapy lasted two more sessions. In the first session, John was, as before, encouraged to express his anger and use the relaxation skills to control his anger when necessary. The last session was held 3 weeks later, allowing the patient a short break. The main issues discussed were John's feelings toward his

father, who had in the meantime left home, and John's criticism of his mother's relationships with other men. The content and tone of the discussion indicated that John was trying to deal with his stressors in a more direct way.

A follow-up 1 year later showed no relapse of John's symptoms. He behaved quite normally and successfully coped with the school's demands. Although the parents were still in the process of divorce, and the relationship between John and his father had become very hostile, John remained in control, did not relapse, and remained symptom free.

The type of intervention presented in this case involves a multidimensional strategy. This includes coping skill training, which helped the patient to reduce emotional distress, while allowing the therapist both to establish good rapport and to assess whether common factors, relating to patient-therapist relationship, can be sufficient for inducing therapeutic change. More important, this case provides a clinical demonstration of RCTN's claim that addressing patients' current stressors, along with insight-oriented therapy, which increases patients' awareness of the underlying causes of their behavior, may yield long-lasting therapeutic benefits. Thus, unlike conventional interventions, therapy focused on removing the stressor in the here and now, the very type of stressors that currently motivated the maintenance of the symptom. Furthermore, although the therapy lifted the patient's repression, insight was obtained by directly breaking through the patient's unawareness concerning his aggression, rather than through hypnosis or other psychoanalytic techniques.

Discussion

Despite tremendous research and theoretical effort, investigators have not been successful in integrating the various therapeutic models into one theoretical framework and have become skeptical over whether this major goal in psychotherapy is achievable (e.g., see reviews Feixas & Botella, 2004; Lampropoulos, 2000, 2001). It seems that RCTN can achieve this goal by making two theoretical shifts. First, RCTN distinguishes between neurotic and nonneurotic deviant behaviors, claiming that all neuroses share a similar etiology, whereas nonneurotic behaviors need to be accounted for using different theoretical concepts. Second, RCTN abandons the axiomatic assumption of traditional psychopathology theories that both the development and treatment of neurosis are controlled by irrational mechanisms (see Rofé, 2000, 2010). Thus, although RCTN remains loyal to the ultimate goal of the integrative movement in psychotherapy, it focuses only on neurotic disorders and claims that the therapies for these disorders can

be integrated into one theoretical framework only when conceptualized in rational-conscious terms.

A successful integrative theoretical approach must not only address the variance in the efficacy of therapeutic interventions, but also explain why different techniques often yield similar outcomes. As noted by Feixas and Botella (2004) in their comprehensive review of psychotherapy integration, "it seems paradoxical that supposedly different (and even contrary) therapeutic models are equally effective" (p. 197). In an attempt to resolve this paradox, these authors devalued the importance of therapeutic techniques, stating that "Techniques do not *do* anything to the client" (p. 198), emphasizing instead the importance of common factors associated with the patient-therapist relationship. Contrary to this approach, RCTN claims that the therapeutic techniques are the key factors inducing therapeutic change. Each technique addresses different components involved in symptom maintenance. Hence, to the extent that different techniques result in similar outcomes in the treatment of neurosis, this should be attributed to the fact that each technique encourages the abandonment of the symptom by affecting different underlying mechanisms that maintain the deviant behavior. Alternatively, similar therapeutic outcomes may also be observed when different techniques affect the same component that maintains the symptom, as when the self-deceptive belief is sabotaged by CBT, psychoanalysis, or religious therapy.

RCTN's therapeutic model equips clinicians with theoretical tools that may facilitate both the therapeutic processes and research effort in this area. RCTN raises awareness regarding the psychological effects of their interventions. Furthermore, it may now be possible to plan studies that can examine the relative effects of the variables that are putatively involved in symptom maintenance and symptom abandonment.

GENERAL DISCUSSION

RCTN presents a new outlook in psychopathology that is antithetical to traditional theories in this field. Contrary to the basic assumptions of such theories, RCTN claims that patients consciously and rationally choose a neurotic symptom as a pathological coping mechanism when confronted with intolerable levels of stress. This radical change in conventional thinking enables RCTN to use numerous studies of informational processing to account for patients' unawareness of the underlying cause of their neurotic symptoms. Moreover, this new conceptualization of neurosis provides a breakthrough in understanding the mechanisms of therapeutic change. Consequently, although investigators have become increasingly pessimistic

regarding the possibility of integrating research and clinical evidence pertaining to the efficacy of various therapeutic interventions (e.g., see Feixas & Botella, 2004; Lampropoulos, 2000, 2001), RCTN is able to account for the efficacy of various interventions by a single set of theoretical concepts.

RCTN shares Freud's (1915a, 1915b) clinical intuition that the key for understanding neurosis is clarifying the mechanism that controls the process of unawareness and the manifestation of the symptom. Both theories also believe that all neurotic disorders have the same etiology, and that patients become unaware of a given stressor, trauma, or conflict through the mechanism of repression. Freud became convinced that unawareness is important for understanding the phenomenon of neurosis through his work with neurotic patients. As noted by Shevrin and Dickman (1980), "the clinical phenomena that led to the assumption of unconscious processes often takes the form of a patient describing a bothersome condition that the patient can neither account for nor control" (p. 422). This view is also shared by Woody (2003), who noted that "the unconscious is invoked to explain behavior that is remarkable or portentous and inscrutable: actions and thoughts that seem otherwise inexplicable—bizarre behavior" (p. 190; see also Erdelyi, 1985; Searle, 1992). However, Freud (1915b) proposed an omnipotent mechanism that has neither received empirical support (e.g., see Greenwald, 1992; Grünbaum, 2002; Kihlstrom, 1999, 2000, 2004; G. O'Brien & Jureidini, 2002; Rofé, 2008) nor can it account for the variability in both the prevalence of neurotic symptoms and therapeutic efficacy. Moreover, the unconscious in the context of the development of neuroses becomes meaningless in light of findings that refute the existence of repression (see reviews Piper et al., 2008; Rofé, 2008), the cornerstone of Freud's theory of neurosis (S. Freud, 1914, p. 16).

Contrary to psychoanalysis, RCTN accounts for the phenomenon of unawareness in conscious and rational terms. Repression is defined as a conscious coping mechanism by which the individual deliberately employs distractive measures to eliminate stress-related thoughts from attention. Accordingly, neurosis is seen as a distractive measure that patients adopt when stress exceeds their normal coping resources (see Lester, 2002; Rofé, 2000, 2010). The most important departure from psychoanalysis is the proposition that it is the conscious, rather than the unconscious, which controls the adoption and the maintenance of the symptom. The sophistication of the conscious also enables patients to be unaware of KSI. Thus, although Freud undermined the importance of the conscious, RCTN sees it as the only mechanism that controls the manifestation and maintenance of the neurotic behavior.

In agreement with cognitive theory (e.g., see Beck, 1988; Clark, 1986, 1988), RCTN claims that patients' beliefs play an important role in both the development and treatment of anxiety disorders, and evens expands it to

the entire spectrum of neurotic disorders. However, while cognitive theory maintains that a belief emerges out of irrational thought processes, without clarifying why only a minority of people develops this pattern of thinking, RCTN attributes the origin of the belief to conscious and rational processes. Furthermore, unlike cognitive theory, RCTN claims that the self-deceptive belief has no direct etiological role in the development of the symptom. Moreover, although RCTN agrees that refuting the belief is critically important for the therapeutic process, from its standpoint, CBT does not educate patients to think rationally. Rather, CBT sabotages the patient's self-deceptive belief by rational means. Thus, while RCTN integrates CBT findings into its theoretical framework, it provides different interpretations of the efficacy of this therapeutic intervention.

After a century of intensive research, along with the continued controversy regarding the mechanism that controls the development and treatment of psychological disorders, it appears that the central task of a new theory in psychopathology is to resolve the difficulties in the field and integrate the findings that have accumulated throughout the years into a single theoretical framework. As noted by Tryon (2005) in his discussion regarding the current status of psychopathology theories, "at some point, the resulting facts need to be placed into an explanatory context" (p. 69). This theoretical position is also accepted by modern approaches in the philosophy of science. Although in the past, the philosophy of science emphasized the importance of new evidence for the acceptance of a new theory, largely due to the influence of Popper (1959; see also Hull, 1943; Whewell, 1847/1967), an increasing number of authors claim today that the theory's ability to explain existing data is even more important than its ability to present new supporting evidence (e.g., Brush, 1989, 1990, 1992, 1993; Haig & Durrant, 2002; Proctor & Capaldi, 2001). As noted by Brush (1989),

> There is even some reason to suspect that a successful explanation of a fact that other theories have already failed to explain satisfactorily . . . is more convincing than the prediction of a new fact, at least until the competing theories have had their chance (and failed) to explain it. (p. 1127)

Thus, although RCTN does not present new data of its own, with the exception of a single case study, it provides a theoretical framework by which unaccountable difficulties in both psychotherapy and psychopathology can be addressed. The new insight into the mechanism by which neurosis develops (Rofé, 2000, 2010) and by which therapeutic change occurs, as elaborated in this article may resolve fundamental difficulties encountered by traditional theories of psychopathology in the realm of neurosis. Most important, it helps integrate the accumulated research and clinical findings into one theoretical framework. Although further research is obviously needed to reach the ultimate understanding of neurosis, it

seems that to achieve this goal RCTN must be included in the panorama of available theoretical approaches.

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