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ACTUAL NEUROSIS AS THE UNDERLYING PSYCHIC STRUCTURE OF PANIC DISORDER, SOMATIZATION, AND SOMATOFORM DISORDER: AN INTEGRATION OF FREUDIAN AND ATTACHMENT PERSPECTIVES

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Starting from a contemporary critique of the DSM-IV, *this paper argues that the diagnostic categories of panic disorder, somatization, and undifferentiated somatoform disorders can be understood as belonging to a common type of psychopathology—i.e., the Freudian* actual neuroses. *In addition to their strong clinical similarity, these disorders share an etiological similarity; and the authors propose a combination of Freud's focus on this type of patient's inability to represent an endogenous drive arousal with the post-Freudian focus on separation anxiety. An etiological hypothesis is put forward based on contemporary psychoanalytic attachment theory, highlighting mentalization. Concrete implications for a psychoanalytically based treatment are proposed.*

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INTRODUCTION

In the Western world, current psychiatric and psychotherapeutic practice and research are primarily based on the classification system provided by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) of the American Psychiatric Association (APA 2000). Whereas its first two versions were clearly inspired by psychoanalytic concepts, the later versions, starting with the third edition in 1980, changed to a more empirical approach, based on a description of clinically observable patient characteristics (behavior and complaints). As a result, in its current (fourth, further revised) version, a multitude of disorders are listed in a coordinate manner.

Notwithstanding its popularity, a diagnostic process based on the *DSM* is a questionable undertaking. From a scientific point of view, the value of the system itself is problematic. Up to now, there has been a manifest lack of evidence supporting the reliability and validity of the classificatory system as a whole, as well as of several of the categories and axes implied (Maleval 2003; Widiger 2003). From a psychoanalytic point of view, even more important is its manifest neglect of psychic processes, and, consequently, its limited usefulness for psychotherapeutic intervention (see Shedler 2002). It is our argument that a priori theoretical clustering of disorders based on their dynamic unity provides us with better diagnoses and more useful therapeutic implications.

In this paper, we will focus on panic disorder, on the one hand, and somatization disorder and undifferentiated somatoform disorder, on the other hand—disorders that in recent years have become increasingly prominent in clinical practice (Kaplan, Sadock, and Sadock 2000). In *DSM* terms they are not clustered, but in our opinion they are indeed connected.

Our argument puts forward two theses. First, we believe that there is a common, underlying psychic structure of panic disorder, somatization, and somatoform disorder, and that this structure can best be understood on the basis of the Freudian concept of the *actual neuroses*. Second, we claim that such a psychic structure can be considered a deficiency in the psychic processing of the drive,

which results, in a broader context, from a failure in the relationship between subject and significant other.

We will first outline the respective disorders in terms of their current psychiatric and psychological formulation. Next, we will relate them to Freud's category of the actual neuroses, and, in particular, the anxiety neurosis. In the third section, we will explore Freud's etiological theory. In section four, we discuss important post-Freudian clinical findings and combine them with the theory of actual neurosis. The next section proposes an etiological hypothesis based on current ideas in attachment theory. In integrating the previous sections, we map out the typical failure of psychic representation and the characteristic way of relating to others associated with the disorders in question. Finally, in our conclusion, we discuss the diagnostic importance of actual neurosis from a broader point of view and the therapeutic implications of this specific type of pathology.

PANIC, SOMATIZATION, AND SOMATOFORM DISORDERS

Contemporary thinking on panic disorder dates back to a discovery by Donald Klein (1964) concerning the pharmacological treatment of schizophrenia. One group of schizophrenic patients, characterized by the absence of delusions and hallucinations and the presence of acute anxiety attacks, failed to respond to the then-current pharmacology. This particular kind of acute anxiety was identified as the so-called panic disorder. In subsequent years, panic disorder became an object of study beyond the field of psychosis. In line with this broader orientation, the *DSM-IV-TR* (APA 2000) describes panic disorder (*DSM* codes 300.01 and 300.21) as a mental condition characterized by recurrent panic attacks, a pattern of worrying about these attacks, and consequent behavior changes.

A panic attack itself is defined in the *DSM* as "a discrete period of intense fear or discomfort" (APA 2000, p. 432), with bodily symptoms such as palpitations, pounding heart, or accelerated heart

rate; sweating, trembling or shaking; sensations of shortness of breath or smothering; feelings of choking, chest pain, or discomfort; nausea or abdominal distress; dizziness, unsteadiness, lightheadedness, or fainting; chills or hot flushes; and paresthesias (numbness or tingling sensations), on the one hand—and psychic symptoms, on the other hand, such as derealization (feelings of unreality) or depersonalization (being detached from oneself), fear of losing control or going crazy, and fear of dying.

Most of the symptoms on this list are notably of a physical nature and consist of uncomfortable or painful sensations. This can be further substantiated through reference to the discussion concerning the somewhat paradoxical *nonfearful panic disorder* (or NFPD), which has not yet been included in the *DSM*. Patients associated with NFPD meet criteria for panic disorder, but do not report subjective fear or anxiety (Beitman, Thomas, and Kushner 1992). In such cases, the psychic symptoms listed in the *DSM* description are absent.

Also important to note is the distinction *DSM-IV* makes between panic disorder with agoraphobia (300.21) and panic disorder without it (300.01). Agoraphobia is described as:

> . . . anxiety about being in places or situations from which escape might be difficult (or embarrassing) or in which help may not be available in the event of having an unexpected or situationally predisposed panic attack or panic-like symptoms. Agoraphobic fears typically involve characteristic clusters of situations that include: being outside the home alone; being in a crowd or standing in a line; being on a bridge; and traveling in a bus, train, or automobile. [APA 2000, p. 433]

This coupling of panic attack with agoraphobia arises from the recognition that the two are often found to be closely associated in clinical practice. The many studies devoted to this association invariably come to the same conclusion: agoraphobia is a consequence of a previous panic attack (Clum and Knowles 1991).

In somatization disorder (300.81), as well, discomforting bodily symptoms are prominent. In his classic definition, Lipowski

(1988) defines somatization as the "tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for them" (p. 1359). *DSM-IV-TR* (APA 2000) adds that, in order for a problem to be considered a somatization disorder, the following complaints should be present: four pain symptoms related to at least four different sites or functions (e.g., head, abdomen, back, joints, extremities, chest, rectum, etc.); two gastrointestinal symptoms other than pain (e.g., nausea, bloating, vomiting other than during pregnancy, diarrhea, or intolerance of several different foods); one sexual symptom other than pain (e.g., sexual indifference, erectile or ejaculatory dysfunction, irregular menses, excessive menstrual bleeding, vomiting throughout pregnancy); and one pseudoneurological symptom not limited to pain (conversion symptoms such as impaired coordination or balance, paralysis or localized weakness, difficulty swallowing or lump-in-the-throat aphonia, urinary retention, etc.).

In the case of undifferentiated somatoform disorder (300.82) —a problem sometimes referred to in the current literature as "medically unexplained symptoms," or MUS (De Gucht and Fischler 2002)—medically unexplained physical complaints are once again central. More precisely, chronic fatigue and gastrointestinal and/or genito-urinary symptoms constitute this category.

As one compares the *DSM* descriptions of somatization and somatoform disorders with that of panic disorder, striking similarities appear. First of all, in each case, bodily symptoms are predominant. These symptoms cannot be medically explained, but neither are there clear psychological causes. Several of these symptoms overlap in both groups of disorders (e.g., bodily pain, nausea, and abdominal problems). Concerning psychic symptoms, both similarity and difference can be discerned. The similarity is that the symptoms are a quasi-direct expression of bodily discomfort, accompanied by worrying. The difference can be found at the level of the specific content of the psychic symptoms. In the first case, preoccupation with physical illness stands at the fore, whereas in the second case, anxiety is central. Nevertheless, em-

pirical research concludes that there is a high correlation (up to 40%) between somatization and anxiety disorders (Fink 1995). Conversely, anxiety can be absent in panic disorder, as in the nonfearful variant.

Thus, the most obvious similarity in all these disorders is that the psychic symptoms are poorly elaborated. Current research concerning the etiology of both categories of disorders is mainly neurobiological and cognitive-psychological. The results remain inconclusive, coinciding with the description of anxiety in panic disorder as "occurring spontaneously."

We believe a third line of research is more promising, as it focuses on the absence of psychic elaboration—referred to as *alexithymia* (Sifneos 1973). The word literally means "no words for feelings," and refers to a deficit in the cognitive processing and regulation of emotions (Bagby and Taylor 1997). Research demonstrates a high prevalence of alexithymia in patients with panic disorder (from 47% to 67%), as well as in patients with medically unexplained symptoms or MUS (from 33% to 55%) (Taylor 2000). A closer analysis (De Gucht 2001) concludes that a significant correlation exists between alexithymia and the number of reported MUS manifestations, and that, when compared to control groups of healthy people, patient groups show a significantly higher presence of alexithymia.

Although studies indicate temporal stability of alexithymia both in clinical and in nonclinical populations (Salminen et al. 1994), it remains unclear whether alexithymia is a causal factor or an accompanying side effect of the disordered state. The question of why some people develop alexithymia while others do not remains unanswered as well. Only occasionally in the literature do we find a vague reference to the early infant–caregiver relationship (Kraemer and Loader 1995) as a possible factor. Moreover, the concept of alexithymia can be criticized for its static character—it merely describes a mental state—and for its hypothetical link to a biological defect (McDougall 1980).

COMPARISON WITH THE FREUDIAN ACTUAL NEUROSES

If one considers the symptomatic similarities between the separately categorized *DSM* disorders under discussion with the Freudian theory of the mind, and especially with Freud's category of the actual neurosis, a common ground between the categories can be discerned.

From 1894 onward, Freud discussed a differential diagnostic distinction, which he retained throughout the course of his *oeuvre*. First, he distinguishes the "Neuro-Psychoses of Defence" (Freud 1894, 1896a) or psychoneuroses. These are disorders whose cause can be found at the level of psychic elaboration—representational and defensive—of infantile sexuality. The accompanying symptoms are signifying, and the typifying characteristic for this group is a defense against an inner conflict concerning sexual desire.

Then Freud introduces the category of the *actual neuroses*. Their cause is similarly located at the level of the drive, but specifically relates to the patient's present life, not the past. Symptoms are limited to bodily phenomena—unprocessed anxiety and somatic anxiety equivalents—and have no defensive significance (see Freud 1892-1899, 1895, 1896b).

At this point in our discussion, it is necessary to consider the drive concept, a problem present in Freud's thinking from the outset, years before the concept itself was introduced (Freud 1905). One problem that bothers him from the start concerns the inner rise in tension, the famous "Q"-factor—i.e., the energetic flux that arises from within the body and therefore cannot be escaped. Later on, this becomes a central characteristic of the drive, namely, the pressure (*Drang*) or excitation (*Erregung*) (Freud 1915, p. 122). This pressure has to be abreacted and the necessary condition for that is its binding via representations. In cases of psychoneurosis, the representation has been distorted by defense mechanisms; in cases of actual neurosis, the step toward representation has not been successful and the innervation remains on the level of the

body. Following this line of reasoning, we will use the terms *pressure, tension, excitation,* and *arousal* as synonyms for the drive.

The categories of psychoneurosis and actual neurosis, however, are not to be regarded as mutually exclusive. In stating that psychoneurotic symptoms hardly ever appear without actual ones, but "the latter can appear without the former," Freud (1910, p. 218; see also Freud 1925, p. 26) legitimizes the fact that actual neurotic pathology is a domain of study on its own. Even more so, he considers the actual neuroses as the nucleus and first stage of the psychoneuroses, particularly within the relationships of *neurasthenia–conversion hysteria; anxiety neurosis–anxiety hysteria;* and *hypochondria–paraphrenia* and *paranoia.* The actual neuroses "provide the psychoneuroses with the necessary 'somatic compliance'; they provide the excitatory material, which is then psychically selected and given a 'psychical coating'"; along the way, "the nucleus of the psychoneurotic symptom—the grain of sand at the centre of the pearl—is formed of a somatic sexual manifestation" (Freud 1912b, p. 248).

In the course of his career, Freud focused primarily on the psychoneuroses, leaving the actual neuroses relatively unexamined, despite the fact that he continued to confirm their existence (Freud 1925, 1926). The reason for this is pragmatic: he did not further examine cases of actual neurosis, since he considered them to be unresponsive to his psychoanalytic treatment (Freud 1910, 1912b, 1925).[1] After all, as the symptomatic superstructure and associated fantasmatic development are completely lacking, there simply isn't anything to analyze. Despite this, he did describe this group thoroughly, first distinguishing between two types of actual neuroses: anxiety neurosis and neurasthenia (Freud 1895, 1896b). Later, he added hypochondria to this list (Freud 1912b, 1914). In each case, the focus is on the drive arousal and on the damming up of libido, along with the impossibility of psychic elaboration. A primary excitatory excess and distress is pivotal to all. And all three actual

[1] It should be noted, however, that this opinion did not prevent Freud (1912b) from granting that "analytic treatment can have an indirect curative effect on 'actual' symptoms" (p. 249).

neuroses are consequently to be considered different manifestations of a similar underlying process.

The category of actual neurosis Freud stresses most is *anxiety neurosis*, to which he can lay claim to both the discovery and naming (Freud 1895, pp. 92-99; 1898). As a matter of fact, he detached it as a separate entity from James M. Beard's larger conception of *neurasthenia*, first described in the 1880s.[2] In cases of anxiety neurosis, Freud distinguished seven clinical characteristics:

1. General irritability: Inability to tolerate excitation.
2. Anxious expectation: A quantum of anxiety in a free-floating state ready to be linked up with any suitable ideational content.
3. Anxiety attacks: Sudden feelings of anxiety without any associated idea, or accompanied by the interpretation that is nearest to hand, such as ideas of death, a stroke, or of a threat of madness, often accompanied by disturbances of one or more bodily functions—spasms of the heart, difficulty in breathing, outbreaks of sweating, ravenous hunger, and the like.
4. A continuum of rudimentary anxiety attacks and their somatic equivalents (not always experienced as anxiety). As equivalents of anxiety attacks, Freud includes disturbances of the heart action; disturbances of respiration; attacks of sweating, tremor, and shivering; attacks of ravenous hunger; diarrhea; locomotor vertigo; congestions; and paresthesias.
5. Waking up at night in a fright (*pavor nocturnus*), usually combined with somatic equivalents of anxiety.
6. Vertigo: The patient is disrupted by sensations of the ground rocking, of the legs giving way, and of finding it impossible to stand up. Although the legs tremble and

[2] For an extensive discussion of neurasthenia and its post-Freudian counterparts, we refer the reader to Hartocollis (2002).

feel as heavy as lead, or the knees give way, this vertigo never results in a fall.

7. Two groups of typically associated phobias: The first relates to general physiological dangers (fear of darkness, thunderstorms, etc.) and to typical moral overscrupulousness and forms of doubting mania. The other group includes agoraphobia. For both kinds of phobias, Freud emphasizes the distinction from psychoneurosis: the affect is always anxiety and does not originate in a repressed idea.

In comparing Freud's clinical descriptions of the actual neurosis with the *DSM* categories we focus on here, our conclusion is that similar problems are surfacing under different labels.

In our interpretation and that of many others (e.g., Compton 1998; de Poderoso and Linetzky 2000; Taylor, Bagby, and Parker 1999), the Freudian anxiety neurosis shows a remarkable overlap with the *DSM* descriptions of panic attack and panic disorder, meaning that the traits listed in the *DSM* can easily merge into Freud's description. The only *DSM* characteristics not explicitly mentioned by Freud are derealization and depersonalization, but these can be understood as a reaction to the failed psychic elaboration of the arousal coming from one's own body. The lack of psychic elaboration determines the dissociative character of these phenomena.

However, the fact that the *DSM* description can merge into the Freudian category does not mean that the reverse is true. Freudian thinking clearly adds an understanding of the dynamics of the phenomena at hand. In the case of anxiety neurosis, for example, Freud stresses the interconnected nature of the problem as he observes that somatic anxiety equivalents can take the place of anxiety, with the absence of experienced anxiety in the patient as a result. In this respect, Freud somewhat foresaw the development of the contemporary concept of nonfearful panic disorder or NFPD (Beitman, Thomas, and Kushner 1992).

Turning now to somatization and somatoform disorders, we find that the overlap with Freud's clinical descriptions is once again remarkable (see also Taylor, Bagby, and Parker 1999, p. 117n). Besides the fact that neurasthenia has been classified as an undifferentiated somatoform disorder in the *DSM-IV*, the actual *DSM* criteria are included in Freud's description of anxiety neurosis (especially the somatic anxiety equivalents). The most conspicuous detail in the *DSM* description of somatization disorder, however, is the presence of conversion in the list of somatic complaints. From a psychoanalytic-therapeutic point of view, we would exclude this symptom from the list, because different dynamic processes underlie the phenomenon. Conversion symptoms are signifying neurotic symptoms that give expression to an underlying conflict; these are clearly to be differentiated from somatization or actual neurotic bodily phenomena (Taylor 2003). The latter are direct consequences of the drive and the associated anxiety; consequently, they are nonsignifying (Freud 1916-1917, pp. 387–388).

Green (1977) made a very apt comparison between (psycho) somatic reactions and expulsion via action, on the one hand, and conversion symptoms and parapraxis, on the other. The latter are constructed in a symbolic fashion; the first are merely discharges to ward off psychic reality. (However, this is a distinction that one can only make based on a theory of psychic functioning, which is absent in the *DSM*.)

We can conclude that at the level of the clinical pictures of the disorders discussed, there are clear similarities between the classical Freudian description of the actual neuroses (especially anxiety neurosis) and contemporary panic, somatization, and somatoform disorders. One reason these similarities are undoubtedly so predominant is that cultural change has little or no effect on the bodily phenomena we discuss. Cultural changes will, however, affect the meanings people attribute to these phenomena, and hence the descriptions of the psychoneuroses.

This conclusion is already worthwhile in itself because it permits us to combine three different diagnoses into one larger category. Nevertheless, from a psychoanalytic point of view, we need

to go further. Instead of accepting the mere descriptive stance of the *DSM*, we must ask questions concerning the etiology as well. In this respect, Freud's theory gives us a good start, but it clearly has its limits. An examination of post-Freudian theory will be much more enlightening here, as it will permit us to address the question of the other. Moreover, it will become clear that it is possible to give the idea of actual neurosis a broader scope, and that a psychoanalytically inspired treatment of this disorder is indeed possible.

FREUD'S THEORY ON CAUSATION

Differences between psychiatric and psychoanalytic understanding of the phenomena most clearly emerge when considering their origin. In discussing Freud's theories on these origins, we want to distinguish clearly between his metapsychological theory on causation and his thoughts on the direct etiology. We believe his reflections on direct etiology were strongly influenced by common theories and preoccupations of his time (such as those of Beard and Krafft-Ebing; see Hartocollis 2002), views that nowadays seem outdated. His metapsychological theory on causation, however, maintains its relevance as a starting point for contemporary causal considerations.

In his metapsychological formulations, Freud locates the dynamics of the actual neuroses in the interplay between a somatic-sexual factor that serves as an endogenous source of excitation and the subject's failure to psychically master this excitation and thus discharge it associatively via a representation. As a result of this failure, excitation exerts a "toxic effect on the body" (Freud 1895, 1912b, 1926). Freud's reflections on causation converge in the idea that, in the case of actual neurosis, the drive is handled in a nonrepresentational way: quanta of somatic-sexual excitation are not coupled to psychic representations, as a result of which excitation remains present in a "free-floating" or "automatic" state. Whereas endogenous excitation obtains a representational coating in psychoneuroses (see Freud 1912b) and finds its symbolic ex-

pression via classically analyzable symptoms, the representational process is short-circuited in cases of actual neuroses, as a result of which excitation reaches excessive proportions and is expressed in bodily phenomena.[3]

The direct etiologies linked by Freud (1925) to these metapsychological formulations concern patients' "abusive" sexual practices (i.e., the way in which they handle somatic-sexual excitation). In the case of anxiety neurosis, Freud points more precisely to the role of an inhibited sexual life: one of abstinence, imperfect or interrupted coition (Freud 1895, 1896b), or, in the case of neurasthenia, "(immoderate) masturbation or spontaneous emissions" (Freud 1896b, p. 150; see also Freud 1912b).

Freud paid little further attention to the causal mechanisms of the actual neuroses in his later work, which has resulted in this part of his theory being nearly forgotten by post-Freudians. This also undoubtedly has to do with the specific direct etiologies that Freud attributed to the actual neuroses (Hartocollis 2002). Nowadays, sexual abstinence and masturbation do not have the same significance that they had in the early 1900s. Our main argument is that these are merely a particular expression of a much broader metapsychological etiology (i.e., the failure to psychically process endogenous excitation via representations) (Freud 1898, pp. 90-115, 1905, p. 204). As such, abstinence points to inhibition (Vanheule 2001), whereas masturbation can be interpreted as a reaction of discharge (i.e., the expulsion via action that Green [1977] points to).

From the perspective of Freud's broader theory, it is not surprising that he links the direct etiology to sexuality. After all, he qualified somatic excitation as sexual (see, e.g., Freud 1895, p. 111). For him, it was "an expression of libidinal instinctual impulses (*libidinöser Triebregungen*)" (Freud 1926, p. 110). In this context, the sexual nature of excitation does not refer specifically

[3] This focus notwithstanding, Freud also indicates that direct changes at the level of bodily excitation can have an effect on actual neurotic symptomatic manifestations. A starting point for reflections on this topic can be found in Freud 1895, p. 111.

to genitality, but more broadly to the way quanta of endogenous excitation are processed. Neurobiological research, too, shows how the increase of stimuli can be linked to more than one bodily system (McNally 1994). It is this mechanism of arousal that Freud (1895, 1905, 1925) qualifies as sexual.[4]

We now turn to the failure of psychic representation that Freud indicated: this is the psychodynamic basis for panic disorder and somatization, which we interpret as belonging to Freud's actual neuroses. We believe that the failure of this psychic representation needs to be studied in the context of psychoanalytic therapy, which Freud neglected to do in his later work. More specifically, we think that the two most prominent factors in psychoanalytic treatment should be taken into account: the patient's style of free association and his/her position in transference. What needs to be examined is the patient's relation to representation and verbalization, as well as the concomitant style of relating to significant others. After all, in current literature, we find clear indications that, at both levels, patients with the disorders under discussion take a specific stance.

THE "NEW" PATIENT: SEPARATION ANXIETY AND LACK OF SYMBOLIZATION

In 1975, Green put forward the idea of a malaise in psychoanalysis. One of the causes for this crisis has to do with what he describes as changes in the post-Freudian patient. He reads these changes from a generic interpretation of the borderline state and psychosomatics.

Some twenty-five years later, Hartocollis (2002) argued that, in the second half of the twentieth century, psychoanalysts from dif-

[4] This opens a discussion of whether there is an original sexual energy that can later become desexualized or neutral, or whether it is the other way around. In our reasoning, we prefer the thesis that psychic development starts at the confrontation with a somatic arousal that must be answered, both in a psychological and a physical way, and for which the intervention of the other is necessary (Freud 1892-1899, pp. 317-322). The sexual drives will be grafted onto these original self-preservative instincts (Freud 1912a, pp. 180-181).

ferent backgrounds studied patients with predominant actual neurotic problems and, in doing so, they pinpointed the poor or even absent psychic representation in domains of functioning where an analyst would classically expect to find conflict and defense, and therefore representation. Nevertheless, neither Green nor Hartocollis presents a clear etiological reasoning in relation to these comments. It has been observed that such patients tend to deny any relationship between their problems and an emotionally significant situation, event, or thought; it seems to be difficult for them to express conflictual situations or even to experience these situations in a psychic way (see Milrod et al. 1997).

A recent publication aptly summarizes this thesis in its title: "When Words Fail: Psychosomatic Illness and the Talking Cure" (Kuriloff 2004). Several concepts refer to this same phenomenon of poor psychic processing: unmentalized experience (Mitrani 1993, 1995); bad mentalization (Marty 1980; Smadja 1990); operational thinking[5] (Marty 1980; Marty and de M'Uzan 1963); borderline states of analyzability (Green 1975), in which the patients' structures of meaning and their capacities for symbolization are hampered or even nonexistent; or McDougall's (1972) concept of the *anti-analysand*. Alexithymia, too—a construct first introduced by a psychoanalyst (Sifneos 1973)—has been linked with Freud's actual neuroses (Taylor, Bagby, and Parker 1999; Vanheule, Desmet, Meganck, and Bogaerts 2007; Weinryb 1995). The question remains: What causes the absence of psychic representation in these patients?

This question brings us to this kind of patient's typical style of relating to significant others. Reports on the psychoanalytic treatment of patients with panic disorder show evidence for the occurrence of negative life events implying separation, either emotional or physical, from a significant person in the patient's life *before* the onset of the disorder (Busch et al. 1991; Milrod 1998). Research-

[5] Marty (1980) situates this kind of mental functioning, as well as actual neuroses, in the context of so-called *essential depressions*, in which positive depressive symptoms are absent, the value attributed to representations is dramatically diminished, and somatic disorganization is at the fore.

ers such as Faravelli and Pallanti (1989) also conclude that such patients have been confronted significantly more often than control subjects with the death or severe illness of a relative or friend in the year prior to symptom occurrence. These findings indicate the presence of combined separation and trauma.

The importance of taking into account the role of significant others is also confirmed by the frequent appearance of a combination of panic disorder and agoraphobia, as mentioned by both Freud and by the *DSM*. We find the perspective of Klein and Gorman (1987) to be the most interesting one; they present the following progression as typical: the spontaneous occurrence of the first panic attacks is followed by help-seeking behavior, whereupon chronic expectation anxiety appears, followed by the final development of avoidance behavior. We find this sequence particularly noteworthy because here the focus is not on the phobic object to be avoided—the *agora*—but rather on the underlying motivation of the patient in adopting this behavior. In our interpretation, a panic attack always contains a reaction to an internal, unmanageable arousal, as a consequence of which a number of patients appeal to a significant other. The so-called agoraphobia is an expression of this need for the other and the avoidance of all situations in which this other could be absent—in short, separation anxiety.

Similar observations have been made in regard to patients with a somatization or somatoform disorder; the intensity and frequency of the somatic phenomena always increase when the patient feels abandoned by significant others (Blaustein and Tuber 1998; McDougall 1980). During the treatment, separation anxiety tends to arise every time a session nears its end or whenever the analyst goes on holiday (Mitrani 1993, 1995).

Obviously, this combination of separation anxiety and lack of symbolization demands further explanation. Two psychoanalytic authors, Green and Mitrani, have produced milestone papers in this respect.

Following Khan, Green (1975) argues that the post-Freudian patient has changed in such a way that a change in the analyst is needed as well. He understands this patient from a generic inter-

pretation of the borderline state, which he compares to Freud's actual neurosis. He describes four mechanisms of defense as characteristic for them. On one side are the mechanisms of psychic short-circuiting in which drive impulses are internally channeled in somatic reactions or are externally expelled via action: on the other side are the two basic psychic defense mechanisms of splitting and decathexis. The net result of these four defenses is a blindness to psychic reality, combined with a lack of secondary elaboration by means of a representational system. The accompanying anxiety is not castration anxiety, but a combination of separation and intrusion anxiety. There is no repression; the pathology is not situated on the level of desire and conflict, but has everything to do with the lack of thought formation.

Even when such a patient produces an extreme associative release, obsessively compulsive thinking, and dreams and fantasies, these do not belong to normal symbolization. On the contrary, they belong to an *action model,* as a primitive system of defense against the massive quantity of affects. For Green, the combination of separation/intrusion anxiety and lack of symbolization finds its etiological ground in what he calls *blank psychosis,* and in a regression to what he considers a fundamental psychotic kernel.

Mitrani (1995) discusses the same problem from a different perspective. Unlike Green, she states that unmentalized experience is not a matter of regression; these experiences have never been symbolically processed. She defines them as:

> . . . elemental sense data, internal or external, which have failed to be transformed into symbols (mental representations, organized and integrated) or into signal affects (anxiety which serves as a signal of impending danger, requiring thoughtful action), but which are instead perceived as concrete objects in the psyche or as bodily states which are reacted to in corporeal fashion (e.g., somatic symptoms or actions). [p. 70]

Mitrani understands such unmentalized experiences as Freud's actual neurotic anxiety equivalents, although with a different eti-

ology. In her reading, a premature separation from the primary caregiver results in the combination of primordial terror and the inability to mentalize this experience. She refers to Bion's *alpha function* of the mother (i.e., the function of receiving and transforming the infant's *beta elements* [raw sensory data] into meaningful thoughts [*alpha elements*] that allow a differentiation between the internal and the external and between fantasy and reality). The failure of this function is then repeated during the therapy, leaving the therapist with a countertransferential experience of "changelessness and numbness" (Mitrani 1995, p. 104).

In her many clinical examples, Mitrani stresses the separation anxiety the patient feels during the actual sessions and his/her need for holding. For example, one patient managed several times to be hospitalized for a physical condition only during weekends or on holidays because of an otherwise unbearable sense of separation from the therapist. More subtle is the patient who buries the therapist under a "barrage of words" at the end of the week (Mitrani 1995, p. 92). Interpreting these words has no effect until the therapist understands their function as a protection against the terror of being abandoned. In our reading, this barrage of words is similar to the crying appeal of the baby to the mother when confronted with distress and the possibility of her absence.

Based on both Green's and Mitrani's conceptualizations, it can be argued that this "new" patient represents a reemergence and expansion of Freud's original concept of actual neurosis. Freud's finding that some patients fail to symbolize the inchoate part of the drive arousal is thus broadened to include a tendency toward failure in relations with others. We believe that the best way of understanding the dynamics of the actual neurosis is to combine into one model the typical failure in representation and the specific style of relating in which separation is considered especially problematic. As we will discuss further in the following sections, support for our thesis is to be found in contemporary psychoanalytic attachment theory and research.

THE ACTUAL NEUROSES REVISITED: THE IMPORTANCE OF THE OTHER

Based on Freudian theory, actual neuroses may be understood as a category unsuitable for a classic psychoanalytic approach. Indeed, compared to what is seen in the psychoneuroses, there is a lack of symbolization, and a transferential relationship seems to be absent. Considered from a different angle, this double lack comes down to the same etiology and presents us with an important basis from which to reconsider the treatment of these patients within a psychoanalytic frame of reference.

The combination of the lack of symbolization, affect regulation, and the other is described in contemporary attachment theory. Whereas its original emphasis was on the infantile attachment style and its determining effects on adult relationships, the goal of attachment has been reformulated as the creation of a symbolic representational system through which affect regulation and the development of a self can come into being. Authors in this field have empirically demonstrated that identity arises through the caregiver's mirroring of what the child internally experiences as "arousal," together with the possibility for regulating affect (Fonagy et al. 2002, pp. 145-251). At moments of arousing alarm, the infant performs attachment behavior, such as proximity seeking and proximity maintaining, in an effort at self-preservation and protection. As a consequence of this appeal to the other, a representational system is created that enables the child to cope with situations of distress on his or her own (Fonagy et al. 2002; Main 2000; Slade 2000). It is in interaction with a caregiver that automatic primary affects can be transitioned into secondary representations. Parallel to this development of the representational system, identity, too, is formed in an interactive process with the caregiver.

The representational system achieved through the other's mirroring action permits the child to gradually acknowledge and master its own bodily arousal. However, for this to occur, the other's mirroring must meet a number of conditions. First, the mirroring

has to be congruent (not identical) with the emotional state of distress of the child. It is the return of one's own experience in a modulated form that makes this experience manageable (Fonagy and Target 1998, 2000). Second, it is important that the child realize that the reaction of the other is not real, but merely a reflection of the child's inner state. This occurs through "marking," which is an exaggerated parental imitation of the child's experienced emotions. The anchoring of the latter to one's own state finally results in the construction of a separate representation: a second-order representation of the primary somatic and affective experience, which has the effect of making this experience manageable. In other words, through the internalization of representations coming from significant others, arousal becomes regulated while identity is being formed at the same time. More specifically, a "physiological self" changes into a "social self" (Fonagy et al. 2002, pp. 203–251).

It is not too difficult to consider this part of attachment theory to be a better explanation for Freud's causal reasoning concerning actual neuroses.[6] Both for Freud and for attachment theory, an internal pressure or arousal functions as a starting point that normally leads to psychic processing via representation. Attachment theory explains how the appeal to the other sets into motion a mirroring process that provides the basis for psychic representation of arousal, as well as for a primary identity through which the child gains both access to this arousal and the possibility of regulating it. In light of Freud's conclusion that, in actual neuroses, the process of psychic representation is lacking, attachment theory permits us to assume that actual neurosis originates from a deficient mirroring process.

[6] In making this claim, we of course acknowledge that constitutional factors, too, play an important role in the etiology of the disorders under discussion. Research indicates that precisely the interplay between genetic determinants and environmental factors, like attachment relationships, strongly determines biochemical abnormalities in the nervous system and/or illness symptoms (see, e.g., Mohammed et al. 2005; Shear 1996). In this paper, we focus on the attachment system since psychoanalytic interventions specifically relate to the patient's representational and relational systems.

In order to specify deficient mirroring as the basis of actual neuroses, some fundamental information can be drawn from empirical studies that have examined the relationship between self-reported attachment styles and panic disorder (Fonagy and Target 2003; Shear 1996), somatization (Ciechanowski et al. 2002; Fonagy and Target 2003; Stuart and Noyes 1999), and somatoform disorders (Stuart and Noyes 1999). Throughout these studies, the anxious-avoidant substyle of insecure attachment has proven to be most typical. Studies starting from implementation of the Adult Attachment Interview have observed that, compared to matched controls, patients with somatoform disorders have considerably more dismissive attachment (Scheidt et al. 1999; Waller, Scheidt, and Hartmann 2004). A common etiological hypothesis that we can distill from these studies, which reported anxious-avoidant and dismissing attachment styles as typical, is that in each case attachment figures are seen as unresponsive. We suggest that this illustrates that the roots of the actual neuroses are a deficient mirroring and symbolization in the relationship between attachment figure and child.

In their discussion of deficient mirroring, Fonagy and colleagues (2002, pp. 192-198) distinguish three pathological mirroring styles. In the first, mirroring is absolutely congruent with the affective state of the child, but marking is absent. As a consequence, the secondary representation of the infant's primary emotional state cannot materialize, resulting in a deficiency of self-perception and self-control of affect. Negative emotions are not modulated but, on the contrary, escalate. Furthermore, the child experiences his/her own negative affect as belonging to the other (projective identification). A history of this type of mirroring is typical in cases of borderline pathology.

In the second case, marking is present; however, the mirroring is not congruent but distorted. Hence, although a secondary representation is constructed, it does not match the constitutional self. The result is a distorted representation of the self state and an alien self. Fonagy and associates (2002) postulate this type of mirror-

ing as causing sexual pathologies in which libidinal excitement is perceived as aggression.

We suggest that this is a fruitful model for understanding pathologies in which discomfort is expressed through physical illness symptoms as well. When attachment figures are preoccupied with physical illness, manifestations of arousal in a child will easily be translated as indicative of illness, and not as affective responses. Discomforting arousal is thus introduced in an objectifying medical discourse and not in a subjectifying discourse that frames arousal as a result of an affected mind, which is exactly what we expect in cases of congruent mirroring. The result is that the subject becomes alienated to a medical discourse for expressing arousal. The subject lacks agency: the mind is framed as powerless in affecting arousal, and relief is only expected from external interventions that address physical functioning. This type of mirroring clearly has a function of solution as it at least tries to grasp the disturbing arousal. The disadvantage of this solution is that it makes the subject strictly dependent on external agents that address the body.

In a third type of deficient mirroring, which they link up with panic disorders, Fonagy and colleagues (2002, pp. 35, 219) focus on incongruence or absence of mirroring. In cases where mirroring is "absent, not readily forthcoming, or contaminated with the mother's own preoccupation," internal states will remain unlabeled and undifferentiated, and therefore difficult to regulate. We suggest that if marking is also absent or barely present, arousal will manifest itself in a raw and unprocessed manner. This unrepresented arousal is manifested through somatic pathways, especially in panic attacks—evoking psychic helplessness due to the impossibility of mental regulation.

This discussion of pathological mirroring enables us to understand the problematic relation of actual neurotic patients to failures in psychic representation and to separation. In cases where mirroring has been deficient, a person remains dependent on the physical presence of the other. This dependency can find its expression in at least two ways: first through a continuing attempt to acquire identity and to regulate arousal by means of others, and second by

taking the other as a screen onto which parts of self-representation that are experienced as alien and incompatible with the self are projected.

By combining contemporary attachment theory with Freudian theory, we can connect the impact of the relation to the other with the original Freudian division between psychoneuroses and actual neuroses. In the case of psychoneuroses, the pressure from the drive is elaborated by means of a relatively congruent and marked mirroring reaction by the other. This forms the basis upon which the subject acquires a representational identity that permits a psychic processing of the drive. Its further oedipal elaboration eventually gives rise to the construction of signifying symptoms. By these means, the original automatic or free-floating somatic excitation is at least partially mastered by a defensive representation.

In the case of an actual neurotic development, this psychic defense has not adequately been established through mirroring. A short circuit between the other's mirroring function and the subject's inner arousal becomes the cause of the actual neurotic structure. As a result of this failure of the other, the pressure of the drive (Freud's damming up of the libido) continues to function on an unelaborated, bodily level, thereby giving rise to automatic anxiety and/or somatic anxiety equivalents without psychic processing (i.e., somatoform disorders with exclusion of conversion phenomena). Note that Freud (1939) considers such a quantitative factor traumatic (p. 73)—meaning that here again we meet with the combined effect of separation and trauma.

The clear advantage of combining current attachment perspectives and classic Freudian theory is that it enables us to integrate the nonrepresentational style of handling endogenous excitation that Freud and many other analysts have observed in actual neurotic patients who exhibit the problematic style of relating to others (whereby separation, especially, comes to the fore) that was likewise observed. The relational style thus becomes the context within which poor psychic processing needs to be considered.

In a recent study, we found preliminary evidence to support this idea. Starting with data collected in a psychiatric population,

we observed that, compared to depressed patients without alexithymia, alexithymic and depressed patients—i.e., those with an explicit nonrepresentational style—had substantially more somatic depressive symptoms and were more distant in relation to others (Vanheule, Desmet, Verhaeghe, and Bogaerts 2007).

CONCLUSION: DIAGNOSTIC AND THERAPEUTIC IMPLICATIONS

We have argued that panic disorder, somatoform disorder, and undifferentiated somatoform disorder are three different manifestations of Freud's actual neurosis. Based on post-Freudian developments, this category can be applied to a generic reading of borderline states and unmentalized experience. From an attachment perspective, their etiology can be understood as a lack in representational coping strategies concerning arousal and drives, based on an original failure in the mirroring relationship with the primary caregivers. One of the consequences is that the subject becomes extremely dependent on the presence of the other, in the sense that he/she persistently clings to the other and appeals to the other for his/her own identity and arousal regulation—and hence, the presence of separation anxiety that both empirical research and clinical practice have indicated.

Following Freud, classically analyzing actual neurosis is impossible, precisely because there is no symbolic symptom formation. Short-term outcome studies seem to demonstrate that cognitive-behavioral therapy produces the best results (Barlow 1997; Gould, Otto, and Po 1995). Outcome studies in long-term treatments that focus solely on the somatic phenomena present a considerably less rosy picture: residual symptoms, relapse, alcoholism, and co-morbidity with anxiety and affective disorders are the rule for 40% to 80% of patients (Milrod and Busch 1996; Rosenbaum 1997; Shear, Cooper, and Klerman 1993; Shear and Weiner 1997).

These new studies resulted in the surprising discovery that the majority of patients (up to 80%) underwent additional treatment

during the follow-up period after the first treatment that the outcome studies were attempting to measure. Because most of the studies following the first treatment failed to inquire into the possibility of additional treatment during the follow-up period, their results are dubious (de Beurs et al. 1999). A review of empirical outcome studies of mainly cognitive treatments for panic disorder (Bakker 2001) demonstrates that with longer follow-up periods, the risk of relapse is considerable, and that patients run a significantly higher risk of developing depression.

Clearly, a cognitive focus on somatic phenomena and/or anxiety is not enough for treatment to succeed. Even more, the failure of such an approach, which has recently become increasingly clear, leads to a repetition of the original problem. From a psychoanalytic point of view, we can predict that the original failure in the relationship to the primary others will be repeated in all later relationships, including the (counter)transference during treatment. It has been noted that these patients tend to develop demanding relationships in which they assume a dependent role and expect to be cured according to a medical model (Taylor, Bagby, and Parker 1999). Conversely, they typically install a distance between themselves and others so as to avoid relational closeness and conflict (Vanheule, Desmet, Meganck, and Bogaerts 2007; Vanheule, Desmet, Rosseel, Verhaeghe, and Meganck 2007).

Realizing that medical science is inapplicable to such patients (see the information about medically unexplained symptoms in the foregoing sections), the physician experiences such patients as "difficult and frustrating" and refers them to a psychiatrist or psychotherapist (Hahn et al. 1996; Walker et al. 1997). If the latter is expecting a patient with "psychological mindedness" and does not find it, a negative countertransferential reaction is to be expected. This can go quite far: such patients have been designated "therapy resistant" (Lydiard and Brawman-Mintzer 1997; Rosenbaum 1997), and a recent empirical study describes the therapist's predominant reaction as one of contempt (Rasting, Brosig, and Beutel 2005)!

In terms of the dialectics of the therapeutic relation, this amounts to blaming the patient: it is the patient who is the reason for the therapeutic failure, s/he is "resistant." In our reading, the reaction of both the physician and the psychotherapist comes down to a modified repetition of the original failed relationship between the patient and the primary other. In both cases, the inability to cope with the discomforting arousal is foremost. The primary other was unable to present the subject with an adequate representational mirroring of what was going on internally, and both physician and psychotherapist experience the ineffectiveness of their usual intervention strategies.

As clinicians, we need to be aware of the risk of blaming the patient for the sense of failure that inevitably slips into the therapeutic relationship. We suggest that we must address this particular (counter)transferential relationship both as a diagnostic tool and a therapeutic medium. Mitrani (1995) aptly describes the feelings of flatness and numbness experienced by the therapist in confrontation with these patients. In actual neurosis, the first aim of the treatment is the restoration—or even the onset—of the primary relation between the patient and the other. This is the condition necessary for the patient to embed the original bodily arousal into meaningful secondary representations.

According to Green (1975), these "new" patients do not only present a challenge for the countertransference: "It is his [the analyst's] mental functions which are demanded, for the patient's structures of meaning have been put out of action" (p. 38). The analytic setting must be used to make the onset and development of a meaningful object relation possible. Mitrani (1995) defines the goal of the treatment as shifting the body memories into verbal representations. Instead of interpretation, the therapist has to contain these states of the patient, to think and even to suffer them "prior to the formulation and delivery of an interpretation" (p. 94). It is as if the therapist has to give the patient a piece of the therapist's mind in order to make the process of mentalization possible. In our reading, the link to the original mirroring process during the holding and containing relationship between child and primary

caregivers is obvious. It fits with the focus that contemporary attachment theory places on the aim of early attachment as the development of a secondary representational system.

This brings us to the second part of our conclusion, namely, the diagnostic status of what we call *actualpathology*. Indeed, as indicated by Green and Mitrani, the idea of actual neurosis can be understood in a broader way than Freud's original conception and its reappearance in panic disorder and somatoform disorders. After all, at the time when Freud formulated his theory of actual neurosis, *neurosis* was a generic term indicating psychiatric disorders. Following Freud, we postulate that a quantum of drive impulses constitutes the *actualpathological* kernel of all pathology, and, usually, it is the starting point for further development into a psychopathological problem. In this sense, the possibility of a regression to this original kernel is indeed open. Even more so, as Green (1975) correctly states, during a classic analysis of a psychoneurosis, this original level can be identified as well. Nevertheless, for the etiological reasons we have discussed above, in a number of cases, the development is arrested at the *actualpathological* level. As a consequence, arousal and drives have to be coped with at a level prior to symbolization.

In this respect, we do not agree with Green's (1975) understanding of these states as belonging to an original and general psychotic kernel (*blank psychosis*). It seems more appropriate to speak of an actualpathological kernel that is present at the start of psychological development as such. We are convinced that such a kernel can be found in psychosis as well.[7] Let us not forget that the rediscovery of anxiety neurosis as panic attack took place within the study of psychosis. The Freudian psychotic actualpathology is hypochondria, the desperate attempt of the psychotic patient to try to give meaning to what s/he experiences at the level of the

[7] This means that the question concerning the specific etiology of neurosis and psychosis is not answered by this reasoning. The idea we put forward here is that an actualpathological form of psychosis is possible as well. For reasons of clarity and space, we have not elaborated the application to psychosis in this paper; the interested reader is referred instead to Verhaeghe 2002, pp. 443-450.

body. For Freud (1911), hypochondria stands in the same relation to paranoia that anxiety neurosis does to hysteria (p. 57). In this paper, we have argued that panic disorder and somatoform disorder can be categorized under the heading of *actualpathology*. And, given our hypothesis that deficient mirroring is the etiological basis of actualpathology, other manifestations are possible as well.[8]

From a psychoanalytic point of view, it can be argued that psychological development starts at the point where drive and affects need to be processed representationally through the mirroring and holding processes presented by primary caregivers. If this takes place, a further development is set in motion, with the possibility of a further psychopathology (in the proper meaning of the prefix *psycho*). If this further development is lacking, an arousal will be expressed at a much more primitive, body- and action-oriented level, for lack of symbolization. Instead of the classic psychopathological symptoms, we will then meet with what we call *actualpathological phenomena.*

The therapeutic and diagnostic consequences are both obvious and far-reaching. On the descriptive level, the original anxiety (i.e., separation anxiety) reappears in the form of unprocessed anxiety, with panic attacks and somatic anxiety equivalents as the most prominent manifestations. Expulsion via action and somatic acting in can be expected and will result in problems that are structurally different from classic symbolic symptoms. On the transferential level, there will be a combination of a clinging, demanding separation anxiety and an inherent difficulty in entering a meaningful relationship. This transferential result of the original relation to the primary caregivers produces the risk of a countertransferential repetition of the pathogenesis, meaning that the therapist does not produce a meaningful answer and even rejects the patient as therapy-resistant.

[8] In another paper, we have demonstrated how post-traumatic stress disorder—and possibly borderline personality disorder as well—can be understood in terms of an insisting, unprocessed, actualpathological kernel that disturbs the patient (Verhaeghe and Vanheule 2005).

Indeed, the therapeutic goal in such treatments must be defined as the exact opposite of that in cases of psychoneurosis: instead of deconstructing symptoms via interpretation and analysis of the transference, the analyst has to construct a subjectively significant meaning for the actualpathological phenomena via the establishment of a working relationship. A purely cognitivist approach does not work, and the mere presentation of interpretations does not help much either. The primary therapeutic instrument here is the analyst him- or herself, who has to take in the experiences of the patient, internalize them—even, in the words of Mitrani (1993, 1995), *suffer them*—in order to be able to present and represent them back to the patient in such a way that s/he can handle them on a symbolic level.

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