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ACTUAL NEUROSIS AND PTSD
The Impact of the Other

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The authors argue that posttraumatic stress disorder (PTSD) is not an automatic consequence of a trauma. A review of empirical research demonstrates that there must be subjective mediating factors. On the basis of a conceptual reasoning, the existence of an *actual-neurotic* structure prior to the traumatic event is put forward as a precondition for the development of PTSD. Freud's theory on *actual neurosis* is interpreted as the impossibility to process the arousal coming from the drive in a symbolic way. The reason for this impossibility is sought in the failure of the primary caretakers in presenting the child with the necessary symbolic tools for drive regulation. The therapeutic implications of the presence of an actual-neurotic structure are important, because it can lead to a failure of free association. Suggestions for a more fruitful approach are formulated.

Keywords: trauma, actual neurosis, PTSD

The central question of this article is deceptively simple: What makes a trauma traumatic? This problem has dogged the clinical world from the outset (Freud & Breuer, 1895/1955) and has resurfaced recently in discussions on false memory (Offer, Kaiz, Howard, & Bennett, 2000). The focus seems to be on the debate over the real or factual character of the trauma. This is quite surprising in light of contemporary research demonstrating that even severe traumas do not automatically lead to a long-lasting psychopathology. There must be another factor involved to account for the fact that although some victims develop a posttraumatic stress disorder (PTSD), a significant number do not.

Our hypothesis is twofold. First, we argue that a traumatic incident leads to the development of PTSD if the victim has a preexisting psychological structure that can be understood as Freud's *actual neurosis*. Second, we assume that this *actual-neurotic* structure is based on early child–caretaker interactions and that it can be diagnosed as such prior to a trauma or PTSD. The relevance of our two hypotheses is mainly

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therapeutic. Indeed, the characteristics of the actual-neurotic structure have important implications for treatment. A possible diagnostic relevance is discussed in our conclusion.

Our argument is based on contemporary research data in combination with our reading of Freud (on actual neurosis), Lacan (on the central position of the Other), and contemporary attachment theory (Fonagy, Gergely, Jurist, & Target, 2002).

Empirical Research

At first sight, the diagnosis of PTSD seems to be an easy one—the etiology is quite clear, and it is generally expected that a traumatic experience must have serious effects on the psychological functioning of the victim. However, when we look at the empirical research data, a different picture emerges.

There are two excellent review articles on PTSD. Paris (2000) searched the Medline and PsycINFO databases for all English-language articles published between 1990 and 1999, identifying approximately 1,000 studies concerning etiology and epidemiology. Lee and Young (2001) focused their review on research concerning differential diagnosis, comorbidity, and epidemiology of PTSD.

The results are more than convincing: "A large body of research indicates that trauma is a necessary but insufficient condition for the development of PTSD" (Paris, 2000, p. 175). Whereas exposure to trauma is widespread—the majority of the Western population experiences a traumatic event during the course of life—only a minority of those exposed are likely to develop a clinically significant psychopathology. The conditional probability of developing PTSD following any trauma appears to vary from approximately 1% to 9% (according to Paris, 2000, p. 176) and 8% to 24% (according to Lee & Young, 2001, p. 155). Consequently, we must conclude that there is no direct connection between trauma and the development of PTSD. Moreover, a trauma in itself is not sufficient for the subsequent development of PTSD. In most cases, the victim experiences an acute stress disorder (according to the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* [*DSM–IV*], 308.3; American Psychiatric Association, 1994) immediately after the event, with a minimum duration of 2 days and maximum duration of 4 weeks, but the subsequent evolution to PTSD is rare (Harvey & Bryant, 1999).

Yet another research finding also indicates that the importance of the trauma is relative. Against all expectations, no connection can be found between the objective severity of the traumatic event and the development of psychopathology. This holds true even for long-term physical and sexual abuse of children: "Such research has consistently shown that exposure to child abuse increases the risk for developing a wide range of psychological symptoms, but that only a minority of exposed persons are likely to develop clinically significant psychopathology" (Paris, Andrews, & Valentine, 2000, p. 176).

These findings point in the direction of our first hypothesis: Whether PTSD develops is not so much determined by the trauma in itself; rather, there must be *mediating factors* of vulnerability and resilience. The question remains as to the nature of these factors. The most obvious answer, in this genetically biased era, suggests a hereditary factor. Behavioral genetics research, based on twin studies, has demonstrated an important genetic contribution to the predisposition to develop PTSD (Kendler, Neale, Kessler, & Heath, 1993; True, Rice, Eisen, & Heath, 1993). However, it is important to view this in the context of two other findings. First, the same behavioral genetics research shows that this genetic disposition is not specific but pertains to a spectrum of diagnoses rather than to one particular diagnosis such as PTSD. Second, environmental factors determine about half of

the variance of almost all personality dimensions. Moreover, there is evidence that traumatic environmental factors themselves are the cause of long-term neurobiological changes (Paris, 2000, pp. 177–178). In other words, it is the ever-complex interplay between nature and nurture that determines the final result.

This interplay brings us to the empirical findings of neuropsychological research (Brewin, 1998; Van der Kolk, 1994; Van der Kolk & Fisler, 1995). Studies on the *memory functioning* of patients with PTSD consistently show that certain representations of the traumatic event cannot be stored in declarative or narrative memory (functioning via the hippocampus); instead they are initially organized only at the sensorimotor and affective levels, in what is called implicit, procedural memory (based in the amygdala). Possible connections between both memory systems are very indirect. The implication is that these representations cannot be remembered as such in a normal, associative way. They can be constructed only retroactively, meaning that a narrative can be built around the traumatic experience, in which the explicit memory is then also involved. This gives a totally different, and much less important, complexion to the discussion of false versus repressed memory. We are left with a catch-22 situation: Anyone who claims to remember a traumatic event hasn't experienced one; anyone who claims not to remember one may have experienced one (Verhaeghe, 2004).

The new question then becomes this: Why is it that a patient diagnosed with PTSD is unable to process the traumatic incident in a normal, associative way? Where does this failure in symbolic processing come from? In our view, this failure, occurring after the trauma, to process information on a symbolic level is the core element of PTSD. When such an elaboration has taken place and the trauma can be remembered in a normal, associative way, it is not a trauma in the PTSD sense of the word. As we will see, the psychological processing of stressful events happens in early development through the mediation by the Other.[1] If this Other, for one reason or another, is not able to provide this mediation, then the processing becomes impossible. A painful illustration in this respect is found in Holocaust survivors. As can be expected, the offspring of survivors of the Holocaust show an increased propensity for developing PTSD when compared with the children of other parents. The curious thing, however, is that these children are also shown to experience *more* posttraumatic stress symptoms than their parents (Yehuda, Schmeidler, Giller, Siever, & Binder-Brynes, 1998). Is it possible that these parents—victims themselves—function in such a way as to be unable to provide their children with the necessary psychological tools to process traumatic experiences? The question has no easy answers, but studies on the impact of the social environment—the Other as a sociocultural discourse—indicate that the presence or absence of social support clearly influences whether PTSD does or does not develop (Brewin, Andrews, & Valentine, 2000; Lee & Young, 2001, p. 156; Paris, 2000, pp. 179–180).

Moreover, some research indicates that this social support could very well be refused by the patients themselves. In studies on the connection between preexisting personality factors and PTSD, the most frequently mentioned personality factors are neuroticism and antisocial behavior or impulsivity (Breslau, Davis, & Andreski, 1995; Williams, 1999). We return to this refusal in our discussion, as it has certain implications.

On this note, we return to our two hypotheses. A traumatic event leads to PTSD on the

[1] Here we are using the Lacanian concept of the "Other." For Lacan, the Other is both the concrete other (particularly the mother and father) and language. It is through the interaction between the subject and the Other that identity is developed (Verhaeghe, 1998).

basis of a preexisting psychical structure, which accounts for the fact that the traumatic incident cannot be processed in a normal, representational way. The question now is whether it is possible to understand this psychical structure as Freud's actual neurosis and, if so, what the impact of the Other is in the development of this actual neurosis. In the next part of this article, we argue that the basis for the development of PTSD must be sought in an actual-neurotic structure in which the Other is the central mediating factor.

PTSD as Actual Neurosis

If we look at the clinical description of PTSD in the *DSM–IV,* the similarities between this clinical picture and certain characteristics of the Freudian actual neurosis (a diagnostic category described by Freud that is nowadays practically forgotten; Hartocollis, 2002) are striking. We proceed with a short description of this actual neurosis, leading to a discussion of its congruences with PTSD.

From the very beginning of psychoanalysis, Freud introduced a differential diagnostic distinction that he would retain throughout his entire oeuvre. On the one hand, he described what he called *Abwehr-Neuropsychosen,* or psychoneuroses of defense (Freud, 1894/1962a, 1896/1962c). The origin of these disorders is situated in the psychical field, that is, in the representational and defensive elaboration of infantile sexuality. The symptoms associated with psychoneuroses carry a meaning that can be interpreted and understood in this context. The central notions here are conflicts concerning sexual desires and defense against this inner conflict. On the other hand, Freud distinguished the *Aktualneurosen*. Their origin is also sexual but in a completely different way. The typical characteristic of the actual neuroses is the absence of a symptomatic superstructure and its associated representational development with regard to sexuality. Symptoms are limited to somatic phenomena, possessing no additional meaning. The central clinical phenomenon is automatic anxiety and the somatic equivalents of anxiety (Freud, 1895/1962b, 1896/1962d, 1985).

In the further course of his career, Freud would be chiefly concerned with the first group of psychopathology. He did not elaborate the group of actual neuroses, in spite of the fact that he affirmed their existence until the end of his oeuvre. The reason for this lack of attention was largely practical: This group was not suited to his psychoanalytic treatment at that time. After all, because the symptomatic superstructure and representational development are absent in their case, there would be simply nothing to analyze. As we will see, another main problem concerns the therapeutic working alliance and the transference: Because of their history, these patients do not easily accept supportive measures.

Nevertheless, this didn't prevent Freud from describing this group in detail and from formulating a number of hypotheses with regard to their etiology. Inside the field of actual neuroses, he made a distinction between neurasthenia and anxiety neurosis. Later, he added hypochondria as well (Freud, 1914/1957a, pp. 82–85). In all of these cases, the determining cause is connected with an inner tension or pressure from the drive,[2]

[2] For Freud, the drive is a border concept between the somatic (source and pressure) and the psychic (object and aim; Freud, 1905/1953b, p. 168). We are aware of the fact that we use the concept of the drive in a way that must sound unfamiliar to many colleagues. Our use of it is based on a combination of the classic Freudian notion, Lacan's interpretation of this part of Freudian theory (especially his Seminar XI, in which he elaborated the drive as one of the four fundamental

combined with the impossibility of elaborating this drive psychically. This results in a primary anxiety and/or somatic equivalents of anxiety (Geyskens, 2001).

When one compares Freud's description of the actual neurosis with the description of PTSD in the *DSM–IV,* a number of similarities immediately appear. First, for both actual neurosis and PTSD, the central clinical phenomenon is *anxiety*. That is why PTSD is classified under the heading of the anxiety disorders in the *DSM–IV*. The nature of this anxiety is quite typical: There is no psychical processing of this anxiety. The fear, helplessness, or horror described in the *DSM–IV* criteria of PTSD closely resembles the anxiety of the anxiety neurosis and the panic attacks in Freud. As a matter of fact, the *DSM–IV* description of panic disorder is almost exactly the same as Freud's description of anxiety neurosis (American Psychiatric Association, 1994; Freud, 1895/1962b, pp. 92–97; Verhaeghe, 2004).

Second, and directly related to this, for both categories of patients it is nearly impossible to produce a normal—that is, an associative—representation and meaningful elaboration of the underlying cause: the drive, in the case of the actual neurosis, and trauma for PTSD as described in Sections B and C of the *DSM–IV* criteria (American Psychiatric Association, 1994). In Section B, we encounter the repetition compulsion and intrusive reexperiencing (instead of recollecting and remembering); Criterion C3 (an "inability to recall an important aspect of the trauma") represents the core problem of the disorder: The traumatic experience is not inscribed within the psychic apparatus and therefore cannot be associatively elaborated. For actual neurosis, this absence of meaningful elaboration is also salient.

These two characteristics already show that, in both cases, the process of psychical representation by way of a secondary elaboration not only is missing but apparently is even inherently difficult.[3] That is why our third reason for placing PTSD on the side of actual neurosis is not at all surprising: In nearly all cases of PTSD, one finds phenomena of *somatization* (Lee & Young, 2001, p. 152). The reality of the trauma is inscribed on the

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concepts of psychoanalysis), and contemporary attachment theory (Fonagy et al., 2002). In post-Freudian theory, the focus is mainly on the goal and the object of the drive. In Lacanian theory, the source and the pressure are taken into account as well, as Lacan considers these the basic cause of psychic development. Contemporary attachment theorists talk about the bodily arousal and the infant's need to elaborate a mentalization around this arousal. Their reasoning is easy to combine with the Lacanian theory of identity formation, because there, too, the starting point is the "real" part of the drive (i.e., the source and the somatic arousal) that must be processed through the symbolic order with the help of the Other. In the rest of the article, we use *pressure, tension,* and *arousal* as synonyms. In Lacanian theory, the somatic part of the drive belongs to the order of the Real (as opposed to the Symbolic and the Imaginary), meaning that it is originally not psychologically processed. In this article, the word *real* is used with this meaning (Verhaeghe, 2001).

[3] *Secondary elaboration* (*sekundäre Bearbeitung*) or *secondary revision* is a term used by Freud (1900/1953a, p. 499) in connection with the formation of the dream. It indicates the defensive process by which the manifest dream content is made logical and coherent after awakening. In short, the process through which the dream is reconstructed to become a neatly closed story, over and against the original unconscious conflict source of the dream. We broaden the meaning of this term by applying it to the entire process of identity formation. It is no coincidence that Freud considered the function of synthesis to be one of the major ego functions (Freud, 1923/1961a). Here we can make a clear comparison with the contemporary concept of mentalization (Fonagy, 2001, pp. 165–170). We prefer *secondary elaboration* to *mentalization,* because the former term accentuates the closing or smoothing of the conflict between drive and identity via the story or narrative. Fonagy applied the same line of reasoning with regard to dividedness and identity with the term "narrative smoothing" (Fonagy et al., 2002, pp. 12–13; Holmes, 2001).

body itself. For Freud, somatization was also a typical characteristic of actual neurosis. He regarded these somatic symptoms as equivalents of anxiety and distinguished them from conversion symptoms. The latter imply a psychical elaboration of an underlying conflict concerning sexual desire.

The very high comorbidity of PTSD with toxicomania points in the same direction: The patients try to "treat" their problem by making an immediate impact on their body (Jacobsen, Southwick, & Kosten, 2001; Perkonigg, Kessler, Stortz, & Wittchen, 2000). Moreover, in a large number of patients, toxicomania can be considered as an actual neurosis as well (Loose, 2002).

This descriptive resemblance is striking but not sufficient in itself to conclude that the occurrence of PTSD can be related to a *preexisting* actual neurotic structure. To reach this conclusion, it must be shown that such a structure is present in patients diagnosed with PTSD *before* the actual development of PTSD. Such connection was confirmed by a large-scale epidemiological study ($N = 3{,}021$) of risk factors and comorbidity patterns. This study showed that more than 60% of patients diagnosed with PTSD had primary disorders preceding traumatic events. The highest proportions were found for somatoform disorders (64%) and other specific anxiety disorders, especially social phobia (62%) and simple phobia (71%) (Perkonigg et al., 2000, p. 53).

First is the existence of *somatoform disorders* prior to trauma and PTSD. Empirical research into medically unexplained symptoms has confirmed time and again that somatization can be divided in two categories. On the one hand is presenting somatization, where the somatization is seen as a phenomenon that is secondary to psychological distress; on the other hand we find functional somatization, where somatization is considered a primary phenomenon, characterized by medically unexplained symptoms (De Gucht & Fischler, 2002). The group of *presenting somatization* is characterized by the presence of an affective disturbance (anxiety, depression) and the manner in which the patient presents his or her symptoms to the doctor: The medically unexplained symptoms are presented instead of, or in combination with, psychological symptoms. The dimension of the Other is clearly present here. In the group of *functional somatization,* however, there appears to be no connection with psychological problems or this link is explicitly denied. This distinction, however, remains confusing, and the mechanism underlying both categories is still unclear. The current hypothesis is that somatization can be understood as a somatic equivalent of an anxiety disorder or a depression (De Gucht, 2001). Its relation to the old Freudian theory of actual neurosis seems quite evident but is not mentioned in these studies. In search of an explanation of this phenomenon, researchers have found another important aspect, namely, that the personality factor *alexithymia* is a crucial factor underlying medically unexplained symptoms. *Alexithymia*, literally meaning "no words for feelings," is defined as a deficit in the cognitive processing and regulation of emotions (Bagby, Taylor, & Parker, 1994). In the context of somatization, research into the Toronto Alexithymia Scale (Bagby et al., 1994), the most validated instrument for alexithymia, found a significant and stable correlation between the number of medically unexplained symptoms mentioned and "difficulty identifying feelings," one of the four dimensions of alexithymia (De Gucht, 2001). These findings clearly confirm the existence of a stable subjective position characterized by an absence of psychical elaboration of bodily arousal. This allows us to understand the somatoform disorder, existing prior to trauma, in the context of Freud's actual neurosis (Verhaeghe, 2004).

Second, there is the presence of anxiety disorders, more specifically social or simple phobia. As stated above with regard to the nature of the anxiety in actual neurosis, the

presence of this anxiety prior to trauma points in the direction of a nonneurotic elaborated anxiety, that is, unelaborated via the Other, just as was found in actual neurosis.

On these grounds, we believe there is sufficient evidence to confirm our first hypothesis: PTSD occurs in those victims who, prior to the traumatic incident, *already had an actual-neurotic structure*. It is precisely because of this structure that they are unable to process the trauma in a psychical, representational way and, as a consequence, develop PTSD.

This brings us to our second hypothesis, namely, the importance of the Other as a mediating factor. To anticipate, our claim is that an actual-neurotic structure arises in the course of the process of identity formation in cases where the Other has failed to fulfill its critical task of mediating—that is, representing and mirroring—the regulation of the drive. This is also why separation anxiety, which can find expression in antisocial behavior (Breslau et al., 1995; Williams, 1999) or social phobias (Perkonigg et al., 2000, p. 53), remains so predominant in these patients (Fonagy et al., 2002).

Actual Neurosis Revisited: The Importance of the Other

Freud's conceptualization of actual neurosis contains no reference to the impact of the Other. If we are to combine the Lacanian conceptualization with contemporary attachment theory, we will find that the Other is central in arousal regulation and identity acquisition. Yet in neither theory do we find the concept of actual neurosis. By combining Freud with these two more recent theories, we will be able to clarify the importance of the Other in the development of the actual-neurotic structure.

From 1948 onward, Lacan developed his theory of identity acquisition wherein the Other plays a central role. In short, his starting point is the assumption that, at birth, the child has no given identity; identity is acquired through identification with the mirror image offered by the Other (Lacan, 1966). This mirroring results in an initial awareness of bodily identity, through which the subject gains access to the psychical experience and processing of the component drives. The subsequent development (called *subject formation*) comes down to identifications with the signifiers presented by the Other—hence Lacan's famous statement "L'inconscient, c'est le discours de l'Autre" (The unconscious is the discourse of the Other). Because he starts from Freud's idea of a nonbridgeable gap between the real part of the drive and the symbolico–imaginary elaboration, identity acquisition, for Lacan, is an unending process (Lacan, 1966, 1978a; Nobus, 1998; Verhaeghe, 1998, 2001).

Attachment theory has recently been taken up again from a psychoanalytic perspective, and with it has come an impressive corpus of empirical research. The main conclusions clearly confirm that the original relationship between caretaker and infant determines later relationships. Furthermore, they show that identity is developed via the caretaker's mirroring of the child's internal experiences with regard to "arousal" (Fonagy et al., 2002; Holmes, 2001). It is through this mirroring that the possibility of affect regulation emerges. The correlation with Lacan's mirror stage is clear, even though these authors do not make this connection. Their work on PTSD and on borderline personality disorder clearly links the development of these disorders to a failure in the original process of mirroring as either inadequate or lacking. It is specifically the idea of inadequate mirroring that is applied to borderline pathology (Fonagy & Target, 2000; Fonagy et al., 2002; Target & Fonagy, 1996). Empirical research has repeatedly confirmed that PTSD and borderline personality disorder are based on an original unresolved attachment style.

The question is what "unresolved" means exactly. Research has also indicated that this clinical group experiences increased separation anxiety (Fonagy et al., 2002; Sabo, 1997).

Both theories have an identical point of departure: An internal rise in tension leads to an appeal to the Other, and it is the Other that lays the foundations for a first identity, via his or her mirroring reaction. Representations from the Other result in a "theory of the mind" that provides the subject with both access to and a means of regulating its own drive. It is particularly relevant from our perspective to examine what happens when such a mirroring fails to occur or takes place in an inadequate way. For us, this forms the basis of the actual-neurotic structure, whose typical characteristic is the continuing, unregulated—real—nature or level of the arousal.

At first sight, there seems to be little connection in Freud between the actual neurosis and (the mirroring by) the Other. However, on a closer look, one can find the nucleus of both Lacan's mirror stage and attachment theory. Freud wrestled from the beginning with the central question of etiology. Both actual neurosis and psychoneurosis start from the same point, namely, the experience of an internal pressure that has not been, or is unable to be, psychically elaborated (see especially Draft G "Melancholia," letters to Fliess; Freud, 1985; Geyskens, 2001; Hartocollis, 2002). In the case of actual neurosis, the arousal continues to be active on the somatic level. In the case of psychoneurosis, however, the transition toward psychical processing has begun, hence the construction of hysterical and obsessional symptoms and their interpretative possibilities. The question is how this transition toward psychical elaboration comes about. Here, we must turn to Freud's unsurpassed *Project for a Scientific Psychology* (1895/1966).

At the time of the *Project,* Freud was elaborating a theory of the origin of psychical functioning. The point of departure of human development was found in an original experience of unpleasure, which he called *Schmerz,* caused by an internal "need" and whose prototype was hunger and thirst. Freud considers this pain a quantitative accumulation of tension, whose stimuli break through the so-called protective shields, just like in physical pain (Freud, 1895/1966, pp. 298–307). Later, Freud would compare this to the trauma caused by the drive (1926/1961b, p. 170; see also 1915/1957b, pp. 146–147). In other words, pain, drive, and trauma are brought into a single line: The subject originally experiences them as an arousal coming *from the outside* (see also Verhaeghe, 2001).

Because it originates in the subject's somatically immature, infantile body, the usual flight reaction is impossible. The infant's reaction to this unpleasurable experience is prototypical and will prepare a basic form for all later intersubjective relationships. The baby turns to the Other with the cry; the caretaker is to provide the "specific action" to neutralize the inner arousal (Freud, 1895/1966, pp. 317–321; 1926/1961b, pp. 169–172). This intervention from the Other will always consist of a number of acts and words, indicating that the child's appeal has been understood and dealt with. Notice that because of this prototypical basic form, an original physical pain and excitation becomes inextricably bound up with the Other. In other words, *from the very beginning the somatic pressure acquires an intersubjective dimension,* marking the point at which the transition from the somatic to the psychical takes place.

This transition can scarcely be overestimated; it prepares the foundation for a self-identity along with the ability to regulate the drive arousal. First, the child literally receives images and words for its internal state from the Other (see mirror stage and attachment theory). These representations are the basis for identity formation. Freud described such a process of internalization in terms of incorporation and identification,

which are the foundational mechanisms of the ego.[4] Second, the regulation of the drives is in keeping with this: The images absorbed from the Other (in accordance with the pleasure principle) not only give the subject access to its own "arousal" but moreover teach the subject to handle this arousal via the Other. This process culminates in a special kind of identification during the oedipal period, namely the *Ueber-Ich*.

The acquisition of identity and regulation of the inner tension has clear repercussions that manifest themselves in the way anxiety is experienced, for instance. The original experience of unpleasure and anxiety is of a chiefly somatic nature. Freud calls this automatic or traumatic anxiety (1926/1961b, p. 136, p. 148). This is closely connected to what is called nowadays a panic attack. Such an anxiety is meaningless and has a clear somatic quality (e.g., palpitations, respiratory problems, tremor; Freud, 1917/1963, p. 395; 1926/1961b, pp. 132–133). In addition to the actual anxiety, Freud also describes the somatic equivalents of anxiety, nowadays called "somatization" (1895/1962b, p. 94), which, as research has demonstrated, shows a clear comorbidity with anxiety disorders (Fink, 1995).

The connection with the Other ensures that the original anxiety (helplessness with respect to the arousal) shifts toward separation anxiety (because the Other is supposed to provide an answer) and even later to signal anxiety (signaling the potential return of the original anxiety and/or the potential absence of the Other). The physical unpleasure becomes psychic distress the moment the Other fails to turn up. This kind of separation anxiety will be powerfully present at the beginning of development. It diminishes as identity and acquisition of object permanence are securely established. As the object is internalized, its actual presence is no longer needed.

Joining Freud's theory of the development of the ego to his distinction between actual neurosis and psychoneurosis, we can outline the impact of the Other in this distinction much more clearly. In the case of a psychoneurotic development, the pressure resulting from the drive is processed via the mirroring reaction of the Other. On these grounds, the subject acquires a representational identity, a "theory of the mind," whose further oedipal elaboration may lead to the construction of meaningful symptoms based on unconscious fantasies. This transforms the original automatic anxiety into a defensive signal anxiety. The nature of the symptoms will be determined by the specific mechanisms of defense. In case of an actual neurosis, however, the elaboration does not occur; the subject remains stuck in this transition. The effect with regard to the clinical picture of actual neurosis is an absence of "meaningful" symptoms and the preponderance of anxiety-related somatic phenomena, these being expressions of the automatic anxiety.

This amalgamation of Freudian ego development with the Lacanian theory of identification and attachment theory confirms our second hypothesis: In the development of an actual-neurotic structure, the impact of the Other is absolutely central. In cases where the Other fails to take on his or her mirroring function in relation to the regulation of the drives, the subject cannot develop a psychical apparatus to handle the arousal in a

[4] Already in his *Project,* Freud had stated that this situation, with its combination of inner unpleasure and appeal to the Other, leaves its memory traces, and this is based on the fact that a situation of this kind occurs repeatedly. Freud talked about facilitations (*Bahnungen*—a more correct translation could be "pathways"), which moreover tend to facilitate a later return to them (Freud, 1895/1966, 1920/1955, p. 26; Lacan, 1978b; Verhaeghe, 2001). In terms of contemporary attachment theory, this means that the child constructs a representational system, a "theory of the mind," in which a self-image and an image of the Other is constructed and, more specifically, revolves around the "arousal" that the child experiences (Fonagy et al., 2002, pp. 36–37).

psychical way. The result is that the processing of the drives remains stuck at a somatic level.

This implies that when one is confronted with a traumatic incident at a later stage, the absence of such a psychical processing leads to the same, original reaction: a combination of automatic or traumatic anxiety, somatic equivalents of anxiety and separation anxiety, forged in reaction to something that is experienced as stemming from the outside, just as was the case with the original "arousal."

Conclusion and Discussion

Our argument for tracing the occurrence of a PTSD back to a preexisting, actual-neurotic structure in which the impact of the Other is of central importance can be summarized as follows. The symptoms are parallel in both: automatic or traumatic anxiety and/or somatic equivalents of anxiety, in combination with the absence of psychical processing. Somatizations, along with social phobia, are already present before the emergence of PTSD. The deficiency in the function of the Other is evident not only in the social phobia but also in the presence of separation anxiety. In attachment research, this is the "unresolved" category.

Freud's notion that a trauma acquires an especially traumatic impact when it is repeated can be understood in this way. The subject's failure to process the original arousal as a result of the absence of an adequately mirroring attitude of the Other is the original trauma. Research has shown that previous "life events" and maternal depression are frequently found here (Murray & Cooper, 1997). This allows us to give an operational definition of what a trauma is and to distinguish it from other shocking emotional events. A trauma is the confrontation with an identity-threatening incident that cannot be elaborated by the victim in a normal, associative and meaningful way. The reason for this impossibility can be understood in the light of a preexisting, actual-neurotic structure.

Actual research will be necessary to confirm this hypothesis, and a number of questions remain. Can people develop PTSD without having suffered early negative attachment issues? Our expectation is that they would develop another disorder, possibly only an acute stress disorder, but this needs to be further researched. Second, do people with an actual-neurotic structure always develop PTSD after having met with a trauma? There is no study to our knowledge that has investigated this question. The closest to our line of reasoning is a very recent research project (Declercq & Palmans, in press) in which 544 subjects working in a high-risk environment were researched for PTSD, adult attachment style, perception of social support, and parental sensitivity. They demonstrated that the risk of PTSD is significantly lower if the victim of a critical incident receives social support, on the condition that the victim perceives this support as such, meaning that social support per se is not necessarily helpful. The perception of social support is determined by the attachment style and the experienced parental sensitivity. To the researchers' surprise, they discovered that two attachment styles operate in a protective way: secure attachment and insecure dismissive–avoidant. Moreover, people with these two attachment styles experience the offered social support as optimal and as a surplus, respectively, and both felt overprotected by both parents. Unfortunately for our purposes, alexithymia and actual-neurotic structure were not taken into account. Nevertheless, on the basis of these results, we can conclude that the absence of parental sensitivity lays the ground for transforming a later trauma into PTSD. We interpret this lack of parental sensitivity as an indication for an actual-neurotic structure in their offspring.

This brings us to another question: What are the "mediating" qualities of the Other—ensuring that identity development does not remain stuck at an actual-pathological level? This question has been largely treated by Fonagy et al. (2002). What they call "mentalization" and "reflective functioning" is not difficult to interpret as the surpassing of the actual-pathological position during the development. Their studies indicate that mirroring of the infant's internal experiences by the primary caretakers in a "marked" way is essential in this respect.

Another very interesting question concerns the possibility of carrying out a *diagnostic screening* to identify potential victims of PTSD in advance. We expect that subjects manifesting increased somatization, in combination with the absence of the possibility of psychical elaboration, run a greater risk for the development of PTSD after encountering a critical incident. Alongside the presence of somatization (see above), a useful indication might be found in the presence of alexithymia as a "trait" factor. We have already mentioned that alexithymia is considered a crucial underlying mechanism in somatization, and because somatization is frequently shown to exist prior to trauma and the development of PTSD, alexithymia could be used as an indication for actual neurosis. The Toronto Alexithymia Scale (Bagby et al., 1994) can be used here as an instrument for screening.

At the level of the *treatment,* there are also clear implications. First and foremost, the previous emphasis on remembering the trauma, as the goal of the treatment, must be completely revised. In trauma, the normal, associative processing and elaboration are absent. Therefore, the patient cannot "forget" the trauma, because a normal memory of it is missing. Instead, therapy must focus on the (re)construction of the traumatic incident. Freud himself was on to this very early. In 1895, he asked whether it is true that the lost memories of the trauma have indeed disappeared, or whether it is not actually more likely that the therapist is confronted here with thoughts *that have never come into being before,* so that the treatment must perform a psychical operation that could not take place at the time of the trauma (Freud & Breuer, 1895/1955, p. 300).

Such a reconstruction comes down to redoing a process that was not originally completed. Moreover, in view of what we developed above, the emphasis should not be placed on an accurate, actual reconstruction but rather on the therapeutic relationship in which this takes place.

We can recognize the same line of reasoning in cognitive–behavioral therapy and, more specifically, in its evolution: The emphasis is no longer placed on systematic desensitization and "flooding" but has shifted toward "imaginal exposure." In our reading, this amounts to the installation of a secondary elaboration by way of, and in the presence of, the Other, in which case it is often the therapist who repeatedly narrates the traumatic experience in detail (Livanou, 2001). This approach, however, does not sufficiently emphasize the transference relationship as the operative factor. It is precisely the success or failure of installing an effective relationship with a mediating Other that decides the outcome.

In this way, the treatment becomes the reestablishment of an original relationship between subject and Other in which this Other must take a supporting and a mirroring position. Because of the lack of meaningful symptoms, classical analytic interpretations are not effective here. This is why Freud believed that the actual neurosis was not suitable for a classic psychoanalytic treatment. The following clinical vignette illustrates this.

Mrs. A, 49 years old and divorced, is referred by her physician because of somatization (hypertension and heart palpitations, breathing problems, dizziness, sleeplessness, extreme tiredness). The first interview reveals that her problems started a year ago, after the loss of her job in combination with and related to sexual abuse on the work floor.

During the 4-month treatment (two sessions a week, psychoanalytic psychotherapy face to face) and in spite of our active questioning and supportive attitude, the patient remains stuck in an endless repetition of her somatic complaints without ever being able to link these complaints herself to the sexual abuse. The abuse appears in nightmares, but she cannot think about it during the day. She presents her life story in a very banal way; her marriage and divorce do not evoke many memories, and she describes her parents in a colorless way. The only important memory seems to be the loss of a grandfather when the patient was 10, but even this does not lead to an associative elaboration. Free association remains impossible, and the treatment is stopped in mutual agreement.

In our reading, this patient suffers from PTSD based on an underlying actual neurosis, that is, anxiety neurosis with somatic anxiety equivalents. As already indicated by Freud, classical psychoanalytic treatment did not work, because there were no symptoms that could be interpreted. A supportive approach did not produce the expected results either. We can reasonably assume that our support could not be experienced as such by this patient (see above, Declercq & Palmans, in press). Since the experience of this therapeutic failure, we have taken a different approach to this kind of patient. Active mirroring of the somatic complaints in combination with support and with an almost immediate interpretation of every negative transferential reaction (especially separation anxiety) has produced much better results. The therapeutic goal with these patients is paradoxical as compared with psychoneurosis. Briefly put, in the latter, we need to reduce the (pathological meaning of their) symptoms. In cases of actual pathology, we need to help them to make their symptoms meaningful. The requisite condition, therefore, is the active construction of a positive (counter)transference.

It would be wrong to focus solely on the mirroring per se, for this can work only in, and through, a securing relationship. It is important to avoid the trap of repeating the original failure or even rejection by the Other. The desire of the analyst is crucial here. For analysis to be possible, the answer to the omnipresent question of the subject, "Veut-il/elle me perdre?" (Can he or she lose me? Does he or she want to lose me?) must unambiguously be "no" (Lacan, 1994, pp. 213–215).

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