Posttraumatic stress disorder (PTSD) is a maladaptive response to a traumatic event, which is currently underdiagnosed and undertreated. It is probable that several myths that surround PTSD, for example, that it is almost solely related to combat situations and that it is a “normal” response to a traumatic situation, have contributed to poor recognition of this disorder. The misconception regarding combat and PTSD is reflected in the history of the names given to the disorder—“shell shock,” “soldier’s heart,” “combat neurosis,” and “operational fatigue.” However, in the late 1980s, it was realized that PTSD is related to all types of traumatic events, including rape, physical attack, severe automobile accidents, and natural or human-made disasters. Consequently, the terms for the disorder were changed to “traumatic neurosis” and later to “posttraumatic stress disorder,” and the defined spectrum of events related to PTSD was expanded accordingly.1,2

Keywords: posttraumatic stress disorder; epidemiology; comorbidity; treatment; SSRI; tricyclic antidepressant; MAOI; augmentation treatment

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Interpretation of symptoms, which we would now consider indicating a diagnosis of PTSD, as a “normal response” to traumatic events has further impeded progress in the field. Based on extensive epidemiological studies, it is becoming increasingly clear that the vast majority of individuals who are exposed to a traumatic event will later adapt and continue with their lives. Only a small percentage, which partially depends on the severity and the duration of the trauma and partially on additional factors, will develop a pathological fixation on the traumatic event, ie, PTSD.

According to the 4th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), there are three subtypes of PTSD: (i) acute; (ii) chronic; and (iii) with delayed onset. These subtypes are defined according to when the symptoms appear in relation to the key traumatic event and their duration, although all subsets require a minimum duration of 1 month. Symptom duration of less than 3 months that appear within 6 months of the trauma is diagnosed as acute-form PTSD. Chronic PTSD corresponds to duration of symptoms of more than 3 months, and delayed-onset PTSD corresponds to an onset of at least 6 months after initial traumatic exposure (and may begin up to several decades later).

**Epidemiology**

It has been estimated that at least one third of the population will be exposed to a severe trauma during their lifetime.\(^{3,4}\) Since 10% to 20% of individuals exposed to severe trauma will develop PTSD,\(^{3}\) according to this figure, the prevalence of PTSD in the general population will range from 3% to 6%. This estimation has been confirmed in several studies carried out in the United States,\(^{6,7}\) but not in others.\(^{8,8}\)

The type and magnitude of the trauma on the one hand, and the characteristics of the individual on the other, are all factors associated with the probability of developing PTSD. Personal characteristics that have been associated with higher risk of developing PTSD include high neuroticism scores,\(^{9}\) preexisting depression and anxiety\(^{a}\) (especially social phobia), early history of adversity, and exposure to traumatic events in childhood (childhood separation from parents, childhood abuse, sexual assault, and parental divorce in early childhood).\(^{a}\)

It also seems that, at least in relation to assaultive violence, the female gender is associated with higher risk.\(^{8}\) Other predictors include socioeconomic status; individuals from lower socioeconomic levels may be more prone to develop PTSD.\(^{6}\)

The association between the type of trauma and the differential risk of developing PTSD has been investigated in a number of epidemiological studies. Kessler et al,\(^{6}\) in data deriving from 5877 persons 15 to 54 years of age from the larger National Comorbidity Survey (NCS) of 8098, found that PTSD was associated with 65% of rape cases in males (although the number of times this particular event occurred was very small) and with 49.5% of rape cases in women, with 38.8% of combat-related events, and with 21.3% of women who were faced with criminal assault. Breslau et al\(^{4}\) also report that the highest risks of developing PTSD following civilian traumatic events were associated with rape (49.0% ±12.2%), followed by being badly beaten up (31.9%± 8.6%), and other kinds of sexual assault (23.7%±10.8%).

**Definition and diagnosis of PTSD**

The diagnostic criteria for PTSD are listed in both the DSM-IV and the International Classification of Diseases, 10th revision (ICD-10). The criteria are essentially the same, with the exception that no time requirement is stipulated in the ICD-10. As the authors believe that the element of time is critical in this disorder, the DSM-IV seems to be a more appropriate diagnostic system, and, indeed, has been applied much more widely in studies.

There are four main diagnostic criteria, or characteristic features, of PTSD. These are: exposure to a traumatic event, reexperiencing, avoidance, and increased arousal. According to the DSM-IV, only extreme traumatic stressors, in contrast with general stressful experiences, have been linked etiologically to PTSD. Such traumatic events

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**Selected abbreviations and acronyms**

| Abbreviation | Definition |
|--------------|------------|
| CBT          | cognitive-behavioral treatment |
| 5-HT         | 5-hydroxytryptamine (serotonin) |
| MAOI         | monoamine oxidase inhibitor |
| NCS          | National Comorbidity Survey |
| PTSD         | posttraumatic stress disorder |
| SSRI         | serotonin selective reuptake inhibitor |
| TCA          | tricyclic antidepressant |

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Pharmacological aspects
are defined as situations in which “the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others ...” (DSM-IV, p 427). As per this definition, very severe humiliation, or any other type of disappointment or intense stress, does not fulfill the criteria for a traumatic event. On the other hand, it has been recognized in the DSM-IV that an individual does not need to be exposed to a trauma that is “outside the range of usual human experience,” as previously defined by DSM-III. Moreover, the DSM-IV has added an important element to the diagnosis: the emotional response, which is characterized as “intense fear, helplessness, or horror”; DSM-IV, p 428), and hence, the diagnostic criteria in DSM-IV is more stringent in this regard.

The second feature of PTSD is reexperiencing (Criterion B). The PTSD patient is emotionally stuck in the traumatic event, even many years after it has occurred, and constantly reexperiencing it in various ways: flashbacks; stressful recollections; recurrent, distressing dreams; acting or feeling as if the traumatic event were reoccurring or experiencing intense psychological distress or physiological reactivity following exposure to internal or external cues that symbolize or resemble the event. An additional maladaptive mechanism used by patients diagnosed with other anxiety disorders, including patients with PTSD, is avoidance. Avoidance is listed as Criterion C in the DSM-IV’s definition of PTSD. Patients with PTSD attempt to avoid any stimuli associated even in a peripheral way with the trauma, including smells, feelings, thoughts, activities, places, or people. This avoidance often expresses itself as “emotional anesthesia,” ie, “markedly diminished interest or participation in significant activities,” “feeling of detachment,” a “restricted range of affect,” and a “sense of a foreshortened future.” Sometimes amnestic or dissociative symptoms (which may also be interpreted as avoidance) appear in response to the extreme reexperiencing, and are thought of as another maladaptive mechanism that originally evolves to buffer the individual from painful recollections.

The fourth feature of PTSD (Criterion D) is increased arousal. Patients are constantly “on alert,” have difficulty in falling or staying asleep, suffer from irritability or outbursts of anger, have difficulty concentrating, and experience hypervigilance and exaggerated startle response. For many of the patients and their families, this group of symptoms is particularly difficult as the families need to maintain a very calm environment while the patients are concerned about losing control.

An additional criterion relates to the functional impairment of the symptoms, described as causing severe impairment in social, occupational, and family areas of life. Comorbidity with other mental disorders is prevalent in PTSD. A recent epidemiologic survey indicated that approximately 80% of PTSD patients meet criteria for at least one other psychiatric diagnosis. The most common disorders experienced concurrently with PTSD found in the US National Comorbidity study are major depression (48.5 in women and 47.9 in men), other anxiety disorders (more than one third), and substance abuse (found in one third of women and half of all men). Depression seems to be a common disorder found in comorbidity with PTSD as evidenced by additional studies of different populations. Since symptoms such as guilt, ruminations, decreased concentration, anxiety, and outbursts of anger are parts of other, more familiar disorders, the diagnosis of PTSD may be overlooked. Many times such patients may be misdiagnosed with depression, sleep disturbance, personality disorder, substance abuse, malingering, or even schizophrenia.

Two studies of psychotic female inpatients demonstrate this point. These studies indicate that patients with a history of childhood sexual abuse were more likely to have intrusive, avoidant/numbing, and hyperarousal symptoms than their nonabused counterparts; a full 66% of these women met the diagnosis for PTSD, but had never been diagnosed.

It has further been suggested that the high levels of comorbidity may point to the possibility of several different subgroups of PTSD. An example of such a grouping is development of psychological or behavioral problems before, concurrent with, or after exposure to the traumatic stressor. An alternative approach suggests that the picture may be more complex, that associated psychiatric disorders are not purely comorbid, but “interwoven with the PTSD.”

**Treatment approaches**

Treatment can either be applied to “seal over” the distress of the patient or do exactly the opposite, to “uncover the pain,” which can then facilitate resolution of the traumatic experience in conjunction with psychologically oriented therapy. Accordingly, it has been...
noted that serotonin selective reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) are useful in helping the patient to “put their fears away,” while cognitive-behavioral treatment (CBT) helps patients via stress inoculation, training, and exposure\(^\text{19,20}\) to better cope with the traumatic event.

**Psychological treatment**

The effect of different courses of psychological treatment are only beginning to be systematically reviewed. A combined approach to treatment is generally considered to be beneficial, especially in the acute stages\(^\text{21}\). CBTs are the most developed, and have been most rigorously tested; they include a variety of treatments such as exposure procedures, cognitive restructuring procedures, and anxiety management programs (for a review, see Foa and Meadows\(^\text{20}\)). Further methodologically sound research is needed to follow up on the encouraging preliminary research.

**Psychopharmacological treatment**

The aim of pharmacotherapy is to reduce symptoms of intrusion and generalization of the trauma, lower the degree of avoidance and numbing behavior, reduce hyperarousal, and decrease impulsivity and dissociative symptoms\(^\text{22}\).

While attempting pharmacological intervention for patients with PTSD, careful listing of the main symptoms is advisable, and the therapeutic effect of medications should be evaluated according to the specific changes in those symptoms. In addition, patients should be made aware that it may take as long as 10 weeks, or even longer, to attain the maximal beneficial response. Emerging data indicate that antidepressant medications may have more prominent roles in the treatment of this disorder, namely, selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants (TCAs), and monoamine oxidase inhibitors (MAOIs).

**Serotonin selective reuptake inhibitors (SSRIs)**

SSRIs are currently the most widely investigated agents, and have been studied in several large, multinational, double-blind, placebo-controlled studies. Based on studies with sertraline and fluoxetine, and on additional positive open studies with other SSRIs, namely fluvoxamine and paroxetine, it is becoming increasingly clear that SSRIs are effective in the treatment of PTSD. Moreover, the symptomatic changes are related to the core symptoms of PTSD and not merely to unspecified changes. The doses used in these studies were 40 mg for fluoxetine, 100 to 150 mg for sertraline, 150 to 300 mg for fluvoxamine, and a mean dose of 40 mg for paroxetine.

**Tricyclic antidepressants (TCAs)**

Two double-blind studies with amitriptyline and imipramine showed these drugs to be superior to placebo in PTSD by a difference of 35% in number of improved patients\(^\text{23-25}\). Doses used were 150 to 250 mg of amitriptyline and a mean dose of 225 mg for imipramine. However, as PTSD patients have low tolerance for side effects (related to their hyperarousal cluster of symptoms), TCAs have not been widely used in PTSD. It is of interest to note that there appears to be an inverse relationship between the intensity of exposure to trauma and the success of treatment with TCAs.

**Monoamine oxidase inhibitors (MAOIs)**

In a study that compared imipramine with phenelzine, at a mean dose of 68 mg, and placebo\(^\text{23}\), a better rate of improvement was demonstrated in the phenelzine group (68%) than in the placebo group (28%). Moreover, phenelzine-treated patients showed better treatment retention than those treated with imipramine (7.4 weeks vs 5.6 weeks for imipramine and 5.5 weeks for placebo) and also improved more on globally assessed symptoms (phenelzine: 44%; imipramine: 25%; placebo: 28%). However, it is important to note the attendant risks, namely, hypertensive crisis, if the dietary restrictions (low tyramine diet) associated with this medication are not kept. In a disorder where impulsiveness and the abuse of alcohol are often present these risks may be even higher.

If the patient is not responding, or if the response is partial, one possibility is to switch from one group of medication to another, eg, switching from SSRIs to another group, such as TCAs or MAOIs, or else, to make a switch within the same group, eg, switching from one SSRI (or one TCA) to another.
Although the available data are very limited, another alternative may be to switch from SSRIs to nefazodone, since the side-effect profile of this medication is very favorable and its mode of action quite different. Nefazodone potently antagonizes 5-HT2 receptors while also inhibiting both serotonin and norepinephrine reuptake. The recommended dose for depression is 200 mg twice a day, which would probably be suitable for PTSD as well, providing double-blind studies are able to demonstrate the efficacy in this condition.

**Benzodiazepines**

Regarding the available evidence on benzodiazepines, this group of drugs to have limited efficacy in the treatment of PTSD. Braun et al²⁶ found no significant difference between alprazolam and placebo in a group of 10 patients who had treatment-resistant illness. Propranolol has been administered in open studies of children and adults and was found to improve PTSD symptoms in most of the studies. The role of propranolol is still unclear and needs to be further examined in double-blind studies.

**Augmentation therapies in PTSD**

Although little is known about augmentation strategies in PTSD, one possible approach to treating an individual who is partly responsive or nonresponsive to treatment is in accordance with the symptomatic approach, ie, if the patient is suffering from an outburst of anger, mood stabilizers such as lithium, carbamezapine, or valproic acid might be added.²⁷,²⁸ If anxiety and irritability are present, a buspirone augmentation is an option to be considered.²⁹ Another possibility for augmentation is for a patient who is very agitated. In such cases, small doses of antipsychotics might be administered. The case for the addition of antipsychotics is even stronger if a concurrence of psychosis and PTSD is present. Indeed, there are several case reports demonstrating the efficacy of thioridazine, olanzapine, risperidone, and clozapine.³⁰ Buspirone, a 5-HT₁₅ agonist, and clonidine have been administered either alone or as augmenting agents in PTSD. As buspirone may be associated with decrease of anxiety, it may be administered either as an augmenting agent for SSRIs or TCAs, or as a stand-alone drug (10-20 mg, three times daily).

Clonidine has been reported in open studies to be effective in ameliorating PTSD symptoms, especially the depression component. Although it has also been used as an augmenting agent for imipramine, double-blind studies are needed in order to substantiate this claim. There are a number of case reports with antidepressants such as trazodone, venlafaxine, and bupropion, which in very limited cases under open conditions were reported to be of benefit in improving PTSD symptoms. The doses used were 300 mg for trazodone, 250 mg for venlafaxine, and 300 mg for bupropion.

**Duration of treatment**

Very little is known regarding maintenance treatment of the disorder. It seems, though, that there is a spontaneous decrease in the symptoms of PTSD in the first 6 months following the trauma, which continues up to 4 or 5 years. From this standpoint, one should take into account this spontaneous recovery when applying psychopharmacological intervention during the first 5 years after the trauma, ie, a gradual down-titration of the dose is called for in order to evaluate whether the medication is really needed.

The same basic rules about discontinuation of medication in other anxiety disorders apply here as well, namely, slow and gradual discontinuation, every 6 weeks or so. The down-titration of medication should be in very small doses. Only if by the end of this period there is no sign of symptom exacerbation may the patient proceed to the next titration. In cases of deterioration, a return to the previous dose seems logical, although evidence to confirm this is lacking. For patients who have exceeded the 4- to 5-year period, it seems that long-term administration of medication is often needed, although there is still the possibility of spontaneous remission, as long as 10 years following the trauma.

A special consideration for long-term maintenance treatment of patients with PTSD is in relation to specific dates in the year that may be associated with an exacerbation of PTSD symptoms, such as the anniversary of the trauma, or memorial days for veterans with PTSD. During these periods, specific close monitoring may be appropriate, with the possible addition of nonbenzodiazepine hypnotics and anxiolytics, or alternatively, preparing the patient by increasing the dose of medication that was used throughout the year.
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Conclusion

The prevalence of PTSD ranges from 1.5% to 6% in different studies of different populations. The disorder has severe consequences on the quality of life, not only of the individuals afflicted, but also for their families and significant others. Although it is a prevalent and severe disorder, PTSD is currently underdiagnosed, and consequently undertreated.

The diagnostic criteria for PTSD are comprised of four components: the trauma (including the immediate emotional response); reexperiencing; avoidance (including “emotional anesthesia”); and hyperarousal. In order to identify PTSD patients, specific questions addressing these points need to be included in every mental status examination, especially if elements of depression, anxiety, outbursts of anger, or drug or alcohol abuse are present, as they often appear to be sequelae of PTSD.

Treatment should take a broad approach, addressing familial and occupational issues as well. Currently, SSRIs are emerging as the pharmacological treatment of choice for this disorder, as demonstrated in large double-blind, placebo-controlled, multicenter studies. However, the effect size, though significant, is modest. Clearly, more research and better therapeutic interventions are called for in this unique disorder, which, as per the definition, point to the external stressor as the cause.

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Puesta al día en la epidemiología, el diagnóstico y el tratamiento del trastorno de estrés postraumático

El trastorno de estrés postraumático (TEPT) es una respuesta patológica, que traduce una mala adaptación frente a un acontecimiento traumático. Este cuadro está actualmente subdiagnosticado y subtratado. Esto se debe en parte a una falta de conciencia acerca de la prevalencia de este trastorno. Se ha estimado que al menos un tercio de la población general estará expuesta a un trauma severo en algún momento de su vida, y de ellos aproximadamente un 10% a 20% desarrollarán un TEPT. En diversos estudios en población general se ha encontrado una prevalencia entre el 3% y el 6% de TEPT, lo que se corresponde bien con el marco de referencia antes enunciado. Tanto el tipo de trauma, como las características personales del individuo afectado se asocian con la probabilidad de desarrollar un TEPT. El Manual Diagnóstico y Estadístico de los Trastornos Mentales en su cuarta edición (DSM-IV) determina cuatro criterios diagnósticos para el TEPT: a) exposición y respuesta emocional al acontecimiento traumático, b) re-experimentar el acontecimiento, c) conductas de evitación y d) aumento de la activación fisiológica. Todos estos criterios se traducen en un severo deterioro del funcionamiento ocupacional, social e interpersonal. La frecuencia de comorbilidad con otros trastornos mentales es alta, especialmente con depresión mayor, trastornos de ansiedad y abuso de sustancias. Se han ensayado diferentes tipos de intervenciones psicológicas incluyendo terapia cognitivo conductual y varios tratamientos farmacológicos. Los inhibidores selectivos de la recaptación de serotonina (ISRS) son actuales los medicamentos más ampliamente investigados y se ha encontrado que sus efectos terapéuticos, aunque modestos, son consistentes. Otros medicamentos como los antidepresivos tricíclicos (AT) y los inhibidores de la mono amino oxidasa (IMAO) han demostrado ser efectivos, pero su uso está limitado por los efectos secundarios indeseables. El TEPT constituye un fenómeno psicobiológico, en respuesta al trauma psicológico, que representa una disrupción neurobiológica de mala adaptación y una disfunción psicológica que requiere de mayor reconocimiento e investigación en el futuro.

Mise au point sur l’épidémiologie, le diagnostic et le traitement de l’état de stress post-traumatique

L’état de stress post-traumatique (ESPT) correspond à une réponse inadaptée et pathologique à un événement traumatisant. Ce trouble est actuellement sous-diagnostiqué et sous-traité, en partie en raison d’une mauvaise appréciation de sa prévalence. On a estimé qu’au moins le tiers de la population générale sera exposée à un traumatisme sévère au cours de sa vie et que 10 à 20% de cette population développera un ESPT. Plusieurs études ont trouvé une prévalence de 3 à 6% d’ESPT dans la population générale, ce qui correspond bien à ce schéma. Le type du traumatisme et les caractéristiques personnelles du sujet impliqué sont corrélés à la probabilité de développer un ESPT. La 4e édition du Manuel Diagnostique et Statistique des Troubles Mentaux (DSM-IV) définit quatre critères diagnostiques : l’exposition et la réponse émotionnelle à un événement traumatisant ; la réexpérience ; l’évitement et l’hyperactivité neurovégétative, ainsi qu’une altération sévère des activités professionnelles, sociales et interpersonnelles. Le taux de comorbidité avec d’autres troubles mentaux est élevé, particulièrement en ce qui concerne les dépressions majeures, les troubles anxieux et l’usage de substances toxiques. Différents types d’aides psychologiques, y compris la thérapie cognitivo-comportementale et un grand nombre de traitements médicamenteux ont été essayés. Les inhibiteurs de la recapture de la sérotonine (IRS) sont actuellement les médicaments faisant l’objet des recherches les plus nombreuses, car ils ont des effets thérapeutiques constants, bien que modestes. D’autres composés, tels que les ant dépressives tricycliques et les inhibiteurs de la monoamine oxydase (IMAO), se sont aussi montrés actifs, bien que leur utilisation soit limitée par leurs effets secondaires. L’ESPT, phénomène psychobiologique en réponse à un traumatisme psychologique, correspond à une dysrégulation neurobiologique inadaptée et à un dysfonctionnement psychologique, nécessite donc de faire l’objet d’une meilleure identification et d’une recherche accrue.