Abstract

Posttraumatic Stress Disorder

Acquisition, Recognition, Course, and Treatment

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Posttraumatic stress disorder frequently follows a chronic course and can be associated with other psychiatric conditions, such as anxiety, depression, and substance abuse. PTSD is frequently comorbid with significant effects on emergency services and rescue workers.

TABLE I. Lifetime prevalence of exposure to trauma and risk of PTSD

In a new window

In this window

The introduction of PTSD has been recognized as a distinct psychiatric disorder since the DSM-III in 1980. PTSD are shown by gender and trauma in Table I. The lifetime prevalence of exposure to trauma and the risk of developing PTSD is higher in men. The prevalence of PTSD is higher in men with many other psychiatric disorders, a higher prevalence of PTSD occurs in studies. The risk of developing PTSD has been shown to vary according to the type of trauma experienced and PTSD is a chronic disorder. However, as might be expected, it is not uncommon for PTSD to occur in the community. Significant effects of modifiable, life-time prevalence and disability psychiatric disorder associated with a chronic course and can be associated with other psychiatric conditions, such as anxiety, depression, and substance abuse.
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The Course of PTSD

Pathological Development

When faced with a traumatic experience, a large proportion of the population will experience a brief acute response to stress. However, a smaller proportion will experience persistent PTSD, either alone or in combination with major depression. Previous resilience to stressors decreases the likelihood that PTSD will develop in response to stress. Indeed, both vulnerability and protective factors increase the individual's resilience in the transitional phase from acute stress response to PTSD. In this stage, the acute reaction will stabilize in some individuals, whereas there is progressive decompensation in others.

Some of the symptoms of PTSD are considered to be reflections of the adaptive mental processes involved in the assimilation and integration of new information that results from exposure to trauma. PTSD symptoms representing part of a normal survival instinct in individuals exposed to trauma and the pathological development of PTSD only follow if the response that leads to resolution of the trauma is disrupted in some way. The main conclusion of biological research into PTSD is that exposure to one or more traumatic events triggers a chain of mental and biological events, which ultimately lead to prolonged PTSD.

An examination of the longitudinal course of PTSD in a study of rape victims showed that 91% of rape survivors had all clinical symptoms of PTSD 1 week after the traumatic event, thereby suggesting that such a clinical picture probably constitutes a normal reaction. PTSD declines to 15%–25% 9 months after the event. Following this period, the proportion of rape victims expressing symptoms of PTSD declines to 10%. PTSD is an enduring and chronic disorder that can last for any length of time. In the National Comorbidity Study (NCS), median duration of PTSD among those who did not receive treatment was 3 years. However, this estimate does not take into account the real possibility that people may experience PTSD more than once in their lives. Indeed, a great many people report exposure to at least one lifetime traumatic event, although a community epidemiologic survey in the USA that used DSM-IV criteria reported that 90% of respondents...
whether PTSD develops.

In a prospective study of traumatic stressors (such as loud noises) and determinants over 100 weeks, PTSD became obvious among 4 people with PTSD. PTSD patients therefore continue to read and link their recollection to the noise as an event that triggered their PTSD. Consecutive groups showed a normal pattern of reaction to the noise. Figure 3: The reaction to loud noises (Isaacson, 1989) is similar to PTSD survivors. The auditory startle response of all the trauma survivors is normal at 1 and 4 months after the trauma, suggesting that this is a critical period during which the central nervous system continues to identify and classify the loud noises as threatening in the context of traumatic stress. Although PTSD patients show a normal pattern of reaction to loud noises, they also report that their normal pattern of reaction is different from the normal pattern of skin conductance to loud noises. Figure 2: The importance of such physiological responses is clear since, when do not (Figure 2), the importance of skin conductance and electrodermal responses (involuntary) to normal trauma are not, a short time following trauma, the skin conductance and electrodermal responses (involuntary) to normal trauma are not, a short time following trauma, the skin conductance and electrodermal responses (involuntary) to normal trauma are not.

Similarly, in a study of trauma survivors the physiological response of heart rate in mean=7.2% beats per minute, SD=9.9% (r=0.10); in mean=7.0% beats per minute, SD=9.5% (r=0.25); but not after 1 and 4 months, mean=7.0% beats per minute, SD=9.5% (r=0.10), and 1 week later (mean=7.0% beats per minute, SD=9.5% (r=0.10), the electrical QRS interval (mean=9.5% beats per minute, SD=7.9% beats per minute) versus mean=7.8% beats per minute, SD=9.9% beats per minute, mean=7.8% beats per minute, SD=9.9% beats per minute.

It has been reported that psychophysically induced traumatic survivors exhibited in the emergency room, who subsequently went on to develop PTSD, had higher heart rates at the conclusion of the study. PTSD.

**Figure 1: Rate of PTSD Symptoms in Rape Victims**

- Had exposure to at least one lifetime traumatic event.
Delayed and Chronic Forms of PTSD

Delayed onset, 40% were delayed help-seeking, 33% were exposure of subclinical PTSD, individuals who presented for treatment within 6 years of the Lebanon war, 10% were considered.

The onset of PTSD can be delayed for years. In a large study by Solomon [8], looking at

Develped by another event

retrieved as another event PTSD and the rest were mainly PTSD patients who recovered and were then

diagnosed. This is confirmed in the study by Shaley et al. [9], where 3.1% of patients were only

13% were retrieval of rocused PTSD, and the remaining 4% had other psychopathology.

Delayed onset, 40% were delayed help-seeking, 33% were exposure of subclinical PTSD.

Develped by another event, 40% were delayed help-seeking, 33% were exposure of subclinical PTSD, individuals who presented for treatment within 6 years of the Lebanon war, 10% were considered.

There are two important questions for the clinician to address when trying to recognize the

Related Mental Imaging

FIGURE 2. Physiological Responses to Trauma.
Most cases of PTSD recover within 1 year, and after 6 years recovery without treatment is unlikely. However, up to 70% of patients with PTSD have a chronic condition. Chronic PTSD is characterized by the persistence of symptoms despite treatment and may be associated with a number of comorbid conditions, including depression, alcohol or drug abuse, and chronic medical conditions. Secondary comorbid conditions are related to factors such as trauma history, family history, post-traumatic stress disorder (PTSD), and the complexity of the PTSD reaction. Chronic PTSD is linked with abuse of alcohol and drugs, and increased risk of suicide. The frequency of comorbid disorders with PTSD is shown in the table below.

### Table 2. Frequency of Comorbid Disorders With PTSD in Men and Women

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>74%</td>
<td>79%</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>68%</td>
<td>75%</td>
</tr>
<tr>
<td>Drug Abuse</td>
<td>54%</td>
<td>59%</td>
</tr>
<tr>
<td>Medical Conditions</td>
<td>35%</td>
<td>38%</td>
</tr>
</tbody>
</table>

Disability Associated With PTSD

The chronic form of PTSD is often debilitating. The disability associated with PTSD includes work impairment, change in life trajectories, impaired social relations, marital instability, and perpetuation of violence. This not only represents a burden to the individual but to society as well.

In a study based on the analysis of the NCS data, which examined the effects of mental disorders on work impairment, self-reported work loss (defined as missing a full day of work), and work cut back (either missing part of a day or working less efficiently than usual) during the previous month, those with a diagnosis of PTSD had significantly higher work impairment compared to those without PTSD. The amount of work impairment associated with PTSD was the same as that associated with major depression but less than that associated with panic disorder.

In the NCS data, among those with PTSD, there is an increased risk of making suicide plans (odds ratio [OR] = 2.4; 95% confidence interval [CI] = 1.7–3.3) and an increased risk of attempting suicide (OR = 6.9; 95% CI = 3.4–10.7) for patients suffering from PTSD. In addition, patients with PTSD were more likely to have complex ongoing traumas that occurred in childhood, such as physical or sexual abuse, significant interpersonal relationships, and the process of mastering basic educational skills.
Recognition of Patterns with PTSD

The effects of stress scale, which measures the level of stress, family and social/economic disability, and the vulnerability to

Functional impairment of the patient with PTSD, as seen on the Symptom Checklist-90-R. Similarly, in a study of PTSD among

A study of the quality of life with PTSD reported greater impairment as baseline for subjects with

use of healthcare resources than non-morally-ill patients and encouraged considerable

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References

Conclusions

Introduction of PTSD

Recognition of Patterns With PTSD

Abstract

TOP
Correlative levels associated with PTSD

In interpersonal violence, particularly common among patients who were exposed at an early age to chronic trauma, PTSD prevalence is more chronic and disabling than other cases of PTSD. Furthermore, PTSD after a traumatic event is usually associated with other psychiatric disorders. Although PTSD diagnosis is not included in DSM-IV due to the fact that the vast majority of patients with complex PTSD are not included in the trauma diagnosis, this response has been revealed for the creation of a separate diagnosis to characterize this response. The PTSD prevalence is so distinct that no other group of depression has been disclosed, and the symptom profile is more complex and/or chronic than any other anxiety disorders. PTSD is often complicated by secondary depression (60%-80% of patients), and it is important to note that PTSD is linked with several psychiatric disorders, such as depression, anxiety, and medication

in a study of patients with irritable bowel syndrome of 18 (36%) of 22 patients were diagnosed with PTSD, high anxiety, and depression. In a study of patients with irritable bowel syndrome of 22 (36%) of 60 patients were diagnosed with PTSD.

Moreover, PTSD is associated with personality and interpersonal problems, hallucinations, and cognitive impairments, as well.
Responses

Neurobiology of the Acute Stress Response

The hypothalamic-pituitary-adrenal axis plays a critical role in the stress response. When the hypothalamus detects a stressor, it releases corticotropin-releasing factor (CRF) and corticotropin. These hormones stimulate the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary, which in turn stimulates the release of cortisol from the adrenal gland. Cortisol then acts on the hippocampus and amygdala, modulating the hypothalamic-pituitary-adrenal axis and inhibiting the production of stress hormones.

In healthy individuals, the hippocampus and amygdala play a regulatory role in the stress response. However, in individuals with chronic PTSD, both baseline cortisol and stress-induced cortisol levels are elevated. This can contribute to the development of PTSD symptoms.

In those who have suffered a previous assault that was not resolved after the initial trauma, lower levels of cortisol remain, even in those who have subsequently experienced depression. A study by Itzhaki et al. (1977) found that cortisol levels were significantly lower in those who were exposed to subsequent stressors. This is explained by the HPA axis suppression, which inhibits the release of cortisol, thus reducing the stress response.

In healthy individuals, the hippocampus and amygdala are involved in the modulation of the stress response, while in individuals with PTSD, these structures may be dysregulated, leading to an exaggerated stress response.

The diagram illustrates the neurobiology of the acute stress response, highlighting the role of the HPA axis and the hippocampus and amygdala in regulating cortisol levels.
Following low-dose dexamethasone:

FIGURE 5. Enhanced Suppression of Cortisol

issues, social avoidance)

the cascade of alterations that have occurred since the trauma (sleep disruption, characteristic

cognitive reconsolidation to correct already formed neural associations, addressing any number of
disorders, cognitive reconsolidation to avoid forming new associations and similar generalizations;
PTSD becomes apparent such as reduction of nonspecific arousal, reduction of memory-retained
model for PTSD is correct, then possible causes for intervention that may help the patient with

system. This would lead to a cascade of consequences as detailed in Figure 6. If this cascade
downstream of the HPA axis, which would prevent the dampening down of the sympathetic nervous

Thereafter, under stress, this enhanced negative feedback could result in a premature shift
recently shown to enhance suppression of cortisol in PTSD patients (Figueira et al., 2003).
been successfully demonstrated using a low-dose dexamethasone suppression test, which

inhibition, which results in lower cortisol levels. This increased receptor sensitivitly in PTSD has
the cortisol receptors in the pituitary are oversensitive, leading to a greater negative feedback
reduced to risk for PTSD. The most likely explanation for this low cortisol before trauma is that

controlled may be reduced to account for the reduced the trauma, in short, low cortisol levels may be
of PTSD but whose parents had PTSD also had low cortisol levels, indicating that the low

PTSD and no parental PTSD) have normal cortisol levels. Interestingly, children with no trauma
with PTSD whose parents had PTSD have low cortisol levels, while Holocaust survivors with no
children PTSD during the Holocaust were at risk of PTSD whereas when faced with trauma, children

suicidality. Further study of this group showed that only the children of Holocaust
been shown to be a high-risk group for developing PTSD are the children of Holocaust

recorders for developing PTSD are difficult to design. However, one group of patients that have
varieties such as IQ and education, studies to isolate which of these variables are particular risk
under such an exposure to trauma, avoidant personality, genetics, or familial factors, and cognitive

Therefore, prior trauma is definitely a risk for developing PTSD, along with the more common


PsYchoSocIal TreaTment

Pharmacological treatments are being studied. Both kinds of treatment are effective. Therapy, more recently, for example, and desensitization therapy, has been employed.

A number of treatment outcome studies for PTSD have focused on cognitive-behavioral therapy.

In this context, the importance of stress resilience, improving quality of life, reducing disability, and reducing the impact of PTSD becomes a core symptom.

Appropriate treatment of PTSD is essential to reduce the development of secondary disorders. Multidisciplinary teams in order to help prevent the secondary problems can be included to improve the quality of life of the patient. Early intervention is a key factor in improving and increasing both the functioning and symptoms of PTSD.
FIGURE 7: Response to Psychotherapy

A number of treatments can produce beneficial effects. For PTSD, exposure therapy is the most effective, and cognitive behavioral therapy is also recommended in the treatment of PTSD. In addition, medication with selective serotonin reuptake inhibitors (SSRIs) and benzodiazepines is recommended.

Pharmacotherapy

The principal goals of pharmacotherapy are reducing PTSD symptoms, improving resilience to stress, and preventing retraumatization.

Intrusion to pathological cognitive therapy

Although most have not been well-controlled, a well-designed study by Foa and colleagues (1995) found EMDR was effective in reducing symptoms of PTSD. The use of EMDR in the treatment of PTSD, however, has not been as well studied. In general, the evidence of EMDR in the treatment of PTSD is still not conclusive. It is suggested that EMDR may be effective in reducing symptoms of PTSD, but further research is needed.

Eye movement and desensitization reprocessing (EMDR) is a relatively new therapy for PTSD.

Intervention to pathological cognitive therapy

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sexual assault. Further studies of serotonin are required to demonstrate its efficacy in both
patients, however, 73% of the population was female and 61.9% had suffered physical or
sexual violence (N=0.03), but not on reexperiencing (P=0.01), but on reexperiencing
injury (P=0.02). In addition, serotonin was significantly better than placebo for the symptom clusters
change from baseline for Clinician-Administered PTSD Scale Part 2 (CAPS-2) total score.
PTSD, serotonin treatment yielded a significantly greater improvement than placebo in mean
PTSD-R in the 24-week, double-blind study of serotonin in 187 psychiatric outpatients with DSM-IV-R
PTSD, with placebo treatment in maintenance treatment.

A European study found benefit for fluoxetine in predominantly combat
PTSD patients. However, results in U.S. combat veterans were not as positive as for the general
population. In those new data, similarly, there have been positive placebo-controlled trials for
paroxetine, which are effective in decreasing symptoms of PTSD, and considerable interest has
more recently, placebo-controlled studies have shown that the SSRI’s sertraline and

**Figure 8. Response to MAOI Drugs in PTSD**
Figure 9. Effects of Open-Label Placebo in

With paroxetine (20–50 mg/day) was observed across all domains assessed by the Hamilton Depression Rating Scale (HAMD-D), however, these improvements were not statistically significant when compared with placebo. This new data, which was generated in 12-week, double-blind, randomized, fixed-dose studies also confirmed that treatment benefit was sustained (100'00'0.0>12, P<0.02). Clinical Global Impression (CGIS) score (P<0.00'0.0) and Hamilton Depression Rating Scale (HAMD-D), however, these improvements were not statistically significant when compared with placebo.

In a 5-week, double-blind, randomized, placebo-controlled study of 94 patients, 22
gender differences were statistically significant when placebo after 9 months of treatment.
Paroxetine was well tolerated with an adverse event profile similar to that in other anxiety...

FIGURE 10. Response to Paroxetine in PTSD:

In a new window
In this window

VIEW LARGER VERSION (60K):

Multicenter Trials:

FIGURE 10. Response to Paroxetine in PTSD:

In a new window
In this window

VIEW LARGER VERSION (31K):

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REFERENCES

Can high-risk individuals be identified early following trauma exposure, and then given effective treatments? Can these treatments serve as markers of resilience?

Do the neuroendocrine and other biological alterations found in PTSD correlate with resilience?

How do drugs and psychological treatments compare when combined?

RESILIENCE

Are cognitive-behavioral therapy, exercise, and mindfulness-enhancing techniques helpful?

What factors protect individuals from developing PTSD after trauma, and how might they be promoted?

The following questions remain to be answered, among which are:

- Combined pharmacotherapy with psychological therapy should be considered.
- While PTSD may be successfully treated with psychological but in moderate to severe cases, anticonvulsants may have a useful place.
- Effects in PTSD, with the largest database existing for paroxetine. Other drugs (e.g.,
- feinberg increase attention. Paroxetine, sertraline, and paroxetine have all shown positive
- Exposure therapy has shown promising results, and the utility of pharmacotherapy is now
- recognition and treatment of PTSD may help to prevent the development of secondary mobility.
- With other anxiety disorders, PTSD is associated with substantial impairment. Prompt
- depression as well as comorbid depression, both of which may impair recognition of the condition. As
- becomes disabling and unmitigating, being accompanied by a high incidence of comorbid
- physiological/cognitive and neuroendocrine (HPA
- receiving becoming obvious, including
- variety of factors that determine the likelihood of such
- develop some short-lasting symptoms of PTSD. A
- following exposure to trauma, almost all survivors

CONCLUSIONS
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Harvey MA, Friedman ML, Redcorn RC, et al: Lack of efficacy for Fluoxetine in PTSD.


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