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Review Article

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A Brief Nosological History of PTSD

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Abstract

The modern conceptualization of what we now call posttraumatic stress disorder (PTSD) originated with the European neurologists Jean-Martin Charcot and Hermann Oppenheim, who treated victims of railroad and industrial accidents in the late nineteenth century. Both Charcot and Oppenheim regarded the disorder as stemming from an acute fright or emotional shock. Charcot used the term "traumatic hysteria" to describe a condition of the mind that occurred in mentally defective individuals and stemmed not from the physical effects of the traumatic accident but rather from the idea his patients had formed of it. Oppenheim rejected the hysterical nature of the condition but rather considered that the acute emotional shock induced by the traumatic event injured the nervous system. Both DSM-I and DSM-II failed to recognize these earlier insights, and they did not dignify the condition with a diagnosis. The introduction of the PTSD diagnosis into DSM-III represented a breakthrough in that it a) recognized a common syndrome or pathway shared by victims of disparate traumatic events, b) formulated specific criteria for its diagnosis, c) did not imply a pre-existing mental defect, and d) did not regard the condition as necessarily temporary. By introducing the requirement that the traumatic event induce an acute response of "intense fear, helplessness, or horror," DSM-IV re-introduced Charcot's and Oppenheim's original insight into the causal role of acute emotional shock, but this requirement has now been dropped from DSM-5.

Keywords

Stress disorders; Post-traumatic; Classification; Trauma; Hysteria; Neurotic disorders; Stress; Psychological; Etiology (all MeSH terms)

Early Conceptions

Aspects of what is now termed posttraumatic stress disorder (PTSD) appears in the classical Greek (e.g., Iliad) and Roman (e.g., Aeneid) war literature, illustrating the longstanding strong (but not exclusive) association of this disorder with military combat. After the American Civil War, the psychological problems of combat veterans were called "soldier's heart." In the latter half of the 19th century, the focus shifted to transportation and industrial accidents. So-called "railway spine" was attributed to spinal concussion caused by physical jarring.

Charcot, Janet, and Traumatic Hysteria

Over time, emphasis shifted from the heart and the spine to the mind and the brain, as the key pathological sites of the consequences of psychological trauma. Writing in the 1880s, the French neurologist Jean-Marie Charcot publicized the new diagnostic category "traumatic hysteria" [1]. Charcot posited that intense fright mediated through unconscious mental processes could precipitate physical symptoms [2]. In a revolutionary view of psychological trauma, Charcot regarded traumatic hysteria as stemming not from the physical effects of the traumatic accident (e.g., a railway crash), but rather from the idea his patients had formed of it [3].

A student of Charcot's, Pierre Janet emphasized the mental effects of "vehement emotion," as stimulated by traumatic life events. He proposed that traumatic mental contents could be isolated in the mind according to the hysterical mechanism of dissociation, in which state they could not be integrated into ordinary consciousness, expressed themselves as pathological "automatisms," and thwarted the capacity of the individual to adapt [4]. Some current theories of posttraumatic psychopathology, particularly that resulting from childhood sexual abuse, still place strong reliance on dissociation. Importantly, both Charcot and Janet regarded hysteria and dissociation as reflecting underlying constitutional defects. Additionally, their theories were entirely psychological.

Oppenheim and Traumatic Neurosis

In 1889, the Jewish German neurologist Hermann Oppenheim published a treatise on "traumatic neuroses" [5] based upon his clinical observations of railway, factory, and construction accident victims. Although Oppenheim considered that accident-induced brain lesions could play a role in these persons' pathology, like Charcot he considered that emotional shock alone, even in the absence of physical impact, was capable of causing traumatic neurosis. Unlike Charcot and Janet, however, Oppenheim emphasized the pathogenic effects of emotional shock and terror not upon the mind but rather upon the nervous system. Oppenheim opposed Charcot's and Janet's view of traumatic psychopathology as a hysterical phenomenon, which he believed overly emphasized wishes, fears, and other intrapsychic processes. Moreover, the hysteria formulation suggested that the disorder could be cured by psychological interventions, especially suggestion, whereas Oppenheim's emphasis on the effect of psychological trauma on the nervous system was interpreted as implying incurability. Oppenheim distinguished traumatic neurosis as a specific, narrow diagnostic category from a broader category of ill-defined "post–traumatic neuroses," which included hysteria, neurasthenia, and other miscellaneous entities, in addition to traumatic neurosis [6].

Oppenheim's concept of traumatic neurosis met strong opposition from the German psychiatric community at the time, less for theoretical or clinical reasons than for economic, political, and ideological ones. The majority saw the proposition that emotional shock could cause incurable changes in the nervous system as legitimizing accident victims' illegitimate claims for compensation. It was feared that such a consequence could dent the treasury, deplete the labor force, and even undermine the work ethos and strength of the German nation. These concerns were multiplied by the numbers

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of psychiatric casualties emerging from World War I, to the point that an epidemic of pension neurosis was feared. The debate came to a head in a 1916 meeting in Munich of German psychiatrists and neurologists that focused on combat psychopathology and associated policy implications, at which Oppenheim's traumatic neurosis diagnosis was overwhelmingly rejected [6].

Several issues raised during the Munich debate persist to this day. There is still concern that the validity of the PTSD diagnosis is undermined by compensation seeking [7]. A variety of New Age therapies are reported to produce miracle cures of PTSD patients, suggesting the role of suggestion. In contrast, a current movement [8] to change the name posttraumatic stress "disorder" in veterans to posttraumatic stress "injury" [9] directly reflects Oppenheim's theory. His theory is also receiving vindication in the neuroimaging laboratory, where enduring effects of psychological trauma on the brain are being discovered [10].

Freud too regarded traumatic neurosis as a fright-induced psychological phenomenon, in contrast to psychoneurosis, which he believed was caused by repressed sexual desires. Freud introduced the concept of repetition compulsion [11], as illustrated in recurrent traumatic nightmares. The American psychiatrist Abraham Kardiner coined the term physioneurosis following World War I to emphasize the contribution of the autonomic nervous system, hyperarousal, and the startle response to the traumatic neuroses of war [12]. Kardiner's approach strongly influenced the current concept of PTSD. Following World War II, Grinker's and Spiegel's study of psychiatrically ill military aviators emphasized failed adaptation to stress [13].

Introduction of the Word "Stress" into the Nomenclature

The word "stress" found its way into the posttraumatic nomenclature in 1952 in the first edition of the Diagnostic and Statistical Manual (DSM-I) under the category "Gross Stress Reaction," which was the closest thing to PTSD in that manual. This was defined as a syndrome in response to an exceptional physical or mental stress, such as a natural catastrophe or battle. However, biological research has now made it clear that the classical biological notion of stress, which relies upon excessive activation of the hypothalamic-pituitary-adrenal cortical axis, does not characterize PTSD [14]. Incorporation of the generic and poorly defined term "stress" into the naming of the disorder we are concerned with, has weakened emphasis on the pathogenic role of acute emotional shock.

A Transient Condition

DSM-I included Gross Stress Reaction under the category of "Transient Situational Personality Disorders." This diagnosis was applicable to persons, otherwise normal, who experienced "extreme emotional stress, such as in combat or in civilian catastrophe." It constituted an advance insofar as it recognized the pathogenic potential of extreme events. Importantly, however, this disorder was regarded as transient and reversible, although it was considered possible that the condition could progress to a "neurotic reaction," in which case this diagnosis was only temporary.

In 1968, DSM-II changed the terminology to "Transient Situational Disturbance," which was a category reserved for more or less transient disorders of any severity that occurred in individuals without any apparent underlying mental disorders and that represented an acute reaction to overwhelming environmental stress. Because the condition was transient, it could not qualify for disability payments. It was stipulated that if the patient had good adaptive capacity, their symptoms usually receded as the stress diminished. If, however, the symptoms persisted after the stress was removed, the diagnosis of another mental disorder was indicated. The only place where combat was mentioned in DSM-II was as an example of Adjustment Reaction of Adult Life, to wit, "Fear associated with military combat and manifested by trembling, running and hiding."

Thus did the first two editions of the DSM preserve the prejudices against traumatic neurosis that were manifest or implicit in the 1916 Munich meeting: An extremely traumatic life event cannot cause lasting psychopathology; persons who develop lasting psychopathology after a traumatic event must have been defective to begin with; soldiers who become mentally ill as a result of combat are weaklings and cowards, and they are undeserving of compensation. Against this background, the importance of the introduction of the PTSD diagnosis into DSM-III in 1980, which at the time was met with considerable resistance from the psychiatric establishment, cannot be overestimated.

Synthesis of Event-Defined Conditions into a Unitary Disorder

The inclusion of PTSD in DSM-III represented a breakthrough that had been urged by Vietnam veterans and their advocates, including clinicians who treated them, for the recognition, treatment, and entitlement to service-connected disability, of the adverse psychiatric consequences of their combat service. Previously the syndrome had been classified according to the type of precipitating event, e.g., "post-Vietnam syndrome" [15], or "rape trauma syndrome" [16]. Persuasive evidence of an identifiable, often non-transient syndrome, or so-called final common pathway [17], which was shared by victims of disparate traumatic events, formed the clinical scientific basis for the creation of the unitary diagnosis.

Introduction of Diagnostic Criteria

Aside from the inclusion of PTSD, the DSM-III was unique in its incorporation of specific diagnostic criteria for the diagnoses it comprised. The purpose of these criteria was to improve reliability, i.e., diagnostic agreement among clinicians, and to remedy the old criticism that when three psychiatrists evaluated the same patient, they arrived at five different impressions. This movement originated with the desideratum that researchers working at different sites, all claiming to study a certain mental disorder, were in fact studying the same condition, i.e., they were recruiting similar subjects. To achieve this, "research diagnostic criteria" [18] for the diagnosis of specific mental disorders in subject candidates were formulated for common use by investigators. When the clinical values of this approach became appreciated, it was incorporated into DSM-III.

The Chinese Menu; Category vs. Dimension

The approach adopted in DSM-III and subsequent DSM editions has sometimes been referred to as a "Chinese menu." The characterization dates back to bygone days when customers ordering at Chinese restaurants were required to select two dishes from column A, one dish from column B, etc. This approach persists in DSM-5, insofar as a minimum of two (rather than one or three) symptoms will be required to satisfy two of the four PTSD symptom clusters.

The result is an obvious arbitrariness. It may be that this arbitrariness results from the attempt to convert what is in nature a continuum or spectrum of posttraumatic psychopathology into a categorical (present vs. absent) classification (i.e., PTSD). Analogies exist in other areas of medicine. Whereas a patient either does or does not have tuberculosis, conditions such as hypertension or type 2 diabetes are more appropriately regarded as matters of degree, i.e., dimensional. The little research that has been done suggests that this is also true for PTSD [19]. However, converting PTSD to a dimensional disorder would require a nosological revolution. Insurance companies want a diagnosis, not a dimension, that they can decide whether or not to cover, or compensate. Clinical investigators want to know whether a subject should be assigned to a PTSD or non-PTSD research group.

Failure to Recover

In DSM-III-R, the requirement was added that the disturbance last at least one month following the traumatic event before PTSD could be diagnosed, in recognition that many individuals may show the PTSD syndrome shortly after the traumatic event but get over it fairly quickly. Thus was the element of failure to recover incorporated into the PTSD definition.

Acute Stress Disorder

Partly out of the consideration that PTSD could not be diagnosed until a month had elapsed since the stressor, DSM-IV added a new condition, Acute Stress Disorder (ASD). ASD could be diagnosed between two days and four weeks following the traumatic event, but it could last no longer than four weeks. The definition of the requisite traumatic event, and the requirement of a response of fear, helplessness, or horror, were the same as in PTSD. The ASD syndrome was conceived as a combination of the kinds of re-experiencing, avoidance, and hyperarousal symptoms typically found in PTSD, as well as a required element of dissociation. Skepticism that dissociation is an essential element of the short-term (or even long-term) pathological response to psychological trauma has led to dropping dissociative symptoms from required to merely possible in the DSM-5 ASD diagnosis.

Delayed PTSD

DSM-III included a "delayed" subtype of PTSD which was originally motivated by observations that Vietnam veterans often did not report symptoms until they had returned from combat and resumed living in civilian society, referred to at the time as "delayed stress syndrome." Under this subtype, symptom onset did not occur until at least six months after the trauma. Subsequent research has indicated that the delay is more often in the recognition than in the onset, and truly delayed PTSD has been found to occur in only a small minority of PTSD cases. The DSM-5 criteria include a PTSD subtype "With Delayed Expression," but this only requires that the diagnostic threshold be exceeded more than six months after the event; the onset and expression of some symptoms may be immediate. In DSM-III, PTSD was considered to become chronic if it lasted at least six months. DSM-IV changed this to three months. The DSM-5 criteria dispense with the chronic qualifier altogether. The DSM-5 criteria add two new PTSD subtypes: PTSD in Preschool Children, and PTSD with Prominent Dissociative (Depersonalization/Derealization) Symptoms.

Symptoms and Syndrome

Of all the ingredients of the PTSD diagnosis, the least changed from DSM-III to DSM-5 has been the clinical description of the syndrome. Of the 12 PTSD symptoms that originally appeared in DSM-III, 11 are still included in DSM-5, although not necessarily within the same clusters. Conversely, of the 20 PTSD symptoms in DSM-5, 12 had already appeared in DSM-III. In DSM-5, the PTSD symptoms are categorized into four clusters: intrusion, avoidance, negative alterations in cognitions and mood, and alterations in arousal and reactivity. The first two clusters originally derived from a theory that psychologically traumatized persons alternate between attempts to process traumatic memories and attempts to avoid them [20], but this temporal alternation within the individual has not received empirical validation. Another way of interpreting the co-occurrence of these first two clusters derives from two-factor learning theory, in which intrusions and distress occur, at least in part, in response to trauma-related cues according to classical (Pavlovian) conditioning. Then, because the emotional responses to such cues are are aversive, avoiding them is rewarded according to operant conditioning. Alterations in arousal and reactivity may reflect the phenomenon of sensitization, which is not cue-dependent. Negative alterations in cognitions and mood, which include what previously was referred to as psychic numbing, are closely related to depression, which has high comorbidity with PTSD.

Definition of the Causal Event

Over the four iterations of the PTSD diagnostic criteria since 1980, namely DSM-III in 1980, DSM-III-R in 1987, DSM-IV in 1994, and DSM-5 in 2013 (DSM-IV-TR did not alter the diagnostic criteria), the most conspicuous changes have been in the definition of the required causal traumatic event. DSM-III required "a recognizable stressor that would evoke significant symptoms or distress in almost everyone." DSM-III-R required "an event that is outside the range of usual human experience … that would be markedly distressing to almost anyone." DSM-IV required "an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others." The DSM-5 criteria require "exposure to actual or threatened a) death, b) serious injury, or c) sexual violation." The evolution of these changes over the several DSM editions have reflected the development of thinking that a) events that are capable of causing PTSD in some persons do not necessarily cause symptoms or distress in others persons; b) qualifying events, e.g., accidents, unfortunately too often may fall within the range of usual human experience; and c) the critical ingredient in such events is threat.

Classification by Cause

It has been pointed out that PTSD is nearly unique in the DSM in that, whereas nearly all disorders are defined in terms of phenomenology, PTSD is defined in terms of cause, namely the traumatic event. However, up until DSM-5, PTSD was classified not according to cause but according to phenomenology, namely anxiety. DSM-5 removes PTSD from the Anxiety Disorders and draws upon causality to classify it under a new category: "Trauma- and Stressor-Related Disorders" [21].

The Question of Pathogenesis

DSM-IV was unique in that the PTSD criteria included not only a cause but also a pathogenesis. Criterion A.2 required that the causal

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traumatic event act on the individual to produce an internal response (extreme fear or helplessness) that resulted in PTSD's subsequent symptoms. DSM-5 has eliminated this criterion (which was already eliminated from the ICD-10 criteria in 1994). Whether this represents a step forward or backward remains to be seen. This step could be regarded as abandoning the insights of Charcot and Oppenheim regarding the critical pathogenic role of emotional shock, undoing the narrow definition of traumatic neurosis that Oppenheim strove to promote, and diffusing PTSD into a broad condition or group of conditions with mixed or uncertain pathogeneses. As a distinct medical disorder, PTSD must have a pathogenesis. If this is not found in the effect of emotional shock on the nervous system, then where is it to be found? Eventually the answer may come from biological research.

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