1.1 Walking the Line in Defining PTSD: Comprehensiveness Versus Core Features

The inclusion of post-traumatic stress disorder (PTSD) in the Diagnostic and Statistical Manual III (DSM-III) was a considerable achievement that has vastly enriched our knowledge of reactions to traumatic events and our ability to offer appropriate care to survivors. Nevertheless, limitations created by the way in which PTSD belatedly entered the diagnostic canon continue to create problems today. One problem was created by the assumption, subsequently proven incorrect, that PTSD was fully explained by exposure to an event outside the range of usual human experience. This resulted in the stressor criterion, Criterion A, assuming a central role in the diagnosis. The realisation that PTSD can follow more mundane traumatic events such as motor-vehicle accidents that nevertheless have the potential to create intense fear and helplessness, and the confirmation that individual vulnerability is as important in PTSD as in other psychiatric disorders, has led inevitably to subsequent problems in defining exactly what does and does not comprise a traumatic event.

In the late 1970s there was also far less appreciation than there is today concerning the role of stressful life events in the onset and maintenance of many psychiatric disorders. In seeking to introduce a condition that was defined in terms of the aetiological role of extreme stress, it may therefore not have been so evident to those crafting the DSM-III definition that traumatic stressors would produce a range of psychopathological reactions, and that it would be necessary...
to single out those features that made PTSD a unique syndrome. This aspect (i.e. discriminant validity) is key to defining psychiatric diagnoses and guarantees that the putative disorder is not simply a different but overlapping expression of another condition [1]. In contrast, the approach taken in DSM-III appears to have been one of assuming the uniqueness of the syndrome and seeking to provide a comprehensive description of its features. It is ironic in this regard that the ideas behind the PTSD diagnosis borrowed so much from the work of Mardi Horowitz (e.g. [2]), who, in drawing on earlier research on bereavement, has done more than most to emphasise the similarities between stress-response syndromes following a variety of challenging life events (see [3] for further discussion).

Although the strategy adopted in DSM-III was effective in helping mental health professionals to identify the condition in their practice, one legacy has been the complexity of the diagnosis relative to depression and other anxiety disorders. This complexity is not just created by the stressor criterion but by specifying three different groups of symptoms all possessed of different thresholds (PTSD currently requires one reexperiencing symptom, three avoidance/numbing symptoms and two hyperarousal symptoms). Perhaps as a result, PTSD is often still poorly recognised in primary care [4]. It is unlikely that the proposal to increase the number of symptom clusters from three to four, although this is consistent with the results of factor analyses, will alleviate this problem.

It can also be argued that this complexity has to some extent impeded the development of a scientific understanding of the condition. For example, the degree of overlap between the symptoms of PTSD and other disorders has been frequently remarked upon, and has left the field unable to determine whether the very substantial comorbidity associated with PTSD is a real effect or an artefact of similarity in the definition of the various diagnoses. Similarly, the fact that it is possible to receive the PTSD diagnosis on the basis of so many different combinations of symptoms makes it difficult to define the core nature of the disorder and to generalise the results of individual investigations. The lack of core symptoms of the kind found in other anxiety disorders is a major stumbling block to developing adequate biological models of PTSD.

The proposal to introduce new symptoms, for example those addressing the existence of a pervasive negative emotional state and persistent distorted blame of self or others, continues the strategy of comprehensively describing the features of the condition and will further increase heterogeneity. Although these additional symptoms do characterise many cases of PTSD they are also commonly encountered in other disorders such as depression, and hence will not assist in differentiating PTSD from the other disorders that share these features.

It is a source of disappointment that the DSM-5 Work Group has not had a more extensive knowledge base upon which to draw in considering its options. Analyses of the PTSD construct have largely consisted of factor analyses of the existing DSM-III-R and DSM-IV sets of 17 symptoms. There have been few if any systematic attempts to investigate the effect of varying the thresholds of
the different symptom clusters, or to test whether the predictive value of the avoidance and numbing symptoms [5] is due to the higher threshold required. Nor have investigators conducted factor analyses on a broader range of possible symptoms, or used factor analysis to see which symptoms of PTSD, depression or pathological grief are distinctive and which overlap (but for recent exceptions see [6, 7]).

The proposed revisions for DSM-5 will provide a framework in which better research can be done in future. Clustering several stress- or trauma-induced disorders together is helpful in making explicit the substantial similarity between the different conditions that follow these events. As depressive reactions are also a major consequence of stress and trauma, these should probably form a new category of disorders, rather than a subcategory of Anxiety Disorders. The improved focus of the reexperiencing symptom cluster on memories rather than thoughts of the trauma will provide greater homogeneity and exclude individuals who solely report the depressive symptom of rumination. The separation of deliberate avoidance and numbing symptoms is an essential first step towards properly investigating these very different types of response. The addition of new symptoms, including the externalising features of aggressive and risk-taking behaviours, will hopefully provide additional ways of discriminating PTSD from other stress-response syndromes using factor-analytic and other methods.

The proposed revisions should also bring with them a number of clinical advantages. As Friedman notes, there is a tension between formulating Criterion A sufficiently broadly to include everyone who may benefit from trauma-focused treatment and making it narrow enough to define a homogeneous group of patients. The removal of Criterion A2 will allow people who for various very good reasons did not experience peritraumatic fear, helplessness or horror to be diagnosed with the disorder. Also, by introducing the ASD/PTSD subtype of AD, DSM-5 ensures that those not meeting the current Criterion A may nevertheless qualify for a diagnosis and for treatment. The introduction of additional PTSD symptoms to the diagnosis will alert clinicians to important aspects of the condition such as self-blame and risk-taking behaviour that good management needs to take into account. We are still learning much about stress- and trauma-related disorders, and it is to be hoped that the same open-minded and empirically-grounded spirit that has characterised the DSM-5 consideration of PTSD will also inform the development of DSM-6 in due course.
INTRODUCTION

The diagnosis of PTSD has been an important topic of research by our group [8–10] and we are pleased that our recommendations might be finding echoes in the final formulation of DSM-5. This commentary addresses the ‘clinical’ (rather than research) aspects of PTSD. We will present here our reflections in two parts: the first draws on our practice of clinical psychiatry and psychology, and the second on our forensic experience.

ARE WE MISSING PTSD IN THE CLINICAL SETTING?

While the diagnosis of PTSD is quite straightforward for all members of our department (because of our ongoing field research), we do not make such a diagnosis as often as might be expected. This is also the case in many treatment settings around the world. We have pondered on the reasons for the relative rarity of this diagnosis.

The possible explanations that occurred to us are: patients with PTSD consult our centre less or consult less in general, or else we are not looking hard enough for PTSD. We intuitively dismiss the first two reasons. Our practice is vibrant and draws on all of Lebanon and the entire Middle East region and we are well
known for our work on trauma and post-traumatic experiences, offer our services for free at times of big disasters, and have publicised it on radio, TV and so on for the past 30 years. Furthermore, even if patients with PTSD avoid coming to mental health specialists for fear of reliving their trauma, we would at least expect those with severe impairment in their daily lives to be brought by relatives and family, as occurs very frequently for other disorders in this part of the world.

It is possible, then, that we are not looking ‘aggressively’ enough for PTSD. We have had similar internal discussions on why adult ADHD was not as commonly diagnosed by our group (despite the fact that again we had published on that condition) [11, 12]; we decided we had not been used to looking for it, and the more we did, the more we found it, without falling into the trap of over-diagnosis. Reflecting on this for the purpose of this commentary, it seems to be true too for PTSD. Under-diagnosis at our centre appears to be more common during the first visit, especially when the examining clinician is a psychiatrist. Diagnosis is higher when patients are referred (independently or by the psychiatrists themselves) to our psychologists, or when a second opinion is requested, or during bedside rounds with interns and residents where a mandatory full history is taken.

Systematically, there are five situations which lead us to consider this diagnosis:

1. If the setting (geographically or temporally) favours the focus on the stressor criterion: war, earthquake, plane crash and so on.
2. If the presenting complaint by the patient or the informant is clearly and spontaneously announced to be linked to a trauma.
3. When the patient complains spontaneously of reexperiencing and avoidance symptoms.
4. If the presenting clinical picture is overwhelmingly that of severe anxiety with possible dissociative states and fear.
5. If the treatment provider is interested (for reasons other than the above) in the diagnosis of PTSD.

As stated above, we think this state of affairs is common in most treatment settings across the globe. With more clinical awareness, with more public knowledge, we could gather more data on the wide panoply of PTSD ‘presentations’ that are probably missed today.

Why is it that some patients have symptoms after trauma and others do not? Looking at risk factors, specifically in the case of PTSD, is not entirely satisfactory to clinicians. Risk factors are not automatically thought of as explaining the emergence of any disorder when the disorder has a readily identifiable ‘cause’. If a person is hit by a car, and has a brain haemorrhage, we are not likely to consider risk factors; he or she has simply had a car accident. We think about risk factors only when an uncommon clinical picture is produced by the accident, such as generalised bleeding (was the patient already on anticoagulants before the accident?). The recent tendency to be more cautious about the word ‘cause’
in PTSD will have an impact on research (treatment and prevention) and move the debate in novel directions.

The possible mechanisms involved in the emergence of PTSD and its treatment carry a huge potential not only for improving understanding of why we react the way we do but also for future possibilities in recoding our memories. In the future we might be able not only to modify the emotional tone surrounding the trauma but also to change specifics of it [13, 14]; these are only glimpses of how far research in PTSD could propel us. Ultimately we could build resilience and modify memories: limits would be pushed further in the evolution of Homo sapiens. Undoubtedly, as with many scientific breakthroughs, this would raise a plethora of ethical issues.

Additionally, PTSD is a relative newcomer and consequently does not have the benefit of well-established (and trimmed) entities such as depression and mania; more so, it must earn its reputation before joining the club, especially since embedded in it is the bold contention of linking trauma to a disorder, something that psychiatric nosology has veered away from in the past 25 years, during the relative decline of psychoanalysis. This brings us to the second part of this commentary: the forensic experience.

DOES PTSD ADEQUATELY COVER TRAUMA-RELATED DISORDERS IN THE FORENSIC SETTING?

Our group has recently been called to assess dozens of prisoners of war from another country who claimed to have been tortured or abused. We had never before seen such a large number of patients with PTSD back to back. We worked as a group and naturally exchanged our impressions as we went along. We will not dwell here on the general misery that this group of subjects shared with us: listening to the atrocities they endured is akin to the experience reported by body handlers in situations of mass disasters [15]. The impairment these ex-prisoners claimed varied and was pervasive: professional, marital, social and so on. Most frequently the trauma had occurred months or years earlier; they did not look scared or horrified and did not volunteer complaints of nightmares or flashes.

Avoidance, anger, shame and irritability were the cardinal symptoms. Many of them heard a voice calling them or felt someone was following them. A large number attempted suicide. While they admitted having the other symptoms of PTSD, their complaints were tilted in favour of these cardinal symptoms. Should emotional dysregulation/regulation, as anger, still be thought of only as an associated feature?

Many of the victims were still ‘intra-traumatic’: they were confronted not by reminders, but by the actual alleged perpetrators (or their representatives); thus not only did they react to reminders but also to their ‘persecutors’. How does PTSD symptomatology differ in such cases?

We wondered what to make of the very frequent and more pervasive major depression that followed the trauma. Was this part of the PTSD? PTSD was not
constructed so as to give particular weight to depressive symptoms. Yet in this large group of ex-prisoners, depression symptoms looked at least as important as the classical PTSD symptoms. Are we talking here of multiple comorbid pathologies? Is it a matter of severity or of perspective?

In short, the present DSM-IV criteria of PTSD did not adequately mirror the complaints of this subset of the population. We think PTSD should be part of a larger category, namely trauma-related disorders. As a side note: we wondered at times if these ex-prisoners were fabricating the stories for compensation; but they had not been questioned previously, to our knowledge, on the DSM symptoms of PTSD; an indirect proof being that they overwhelmingly reported irritability, shame and anger as major emotional states, which are not cardinal in the present DSM nosology. We expect this issue (moving from PTSD to trauma-related disorders) to be of importance not only in future developments of psychiatric nosologies but also in giving a new impetus to environmentally induced mental health pathologies. On the other hand, verifying the symptomatology from neurobiological perspectives will be of paramount importance in the continuing debate over the validity of trauma-related disorders in legal circles [16].

CONCLUSION

We think some of the problems with PTSD lie in its bold revival of linking a mental disorder to an event; this means that clinicians might expect that all symptoms secondary to trauma (albeit ‘severe’) can be encompassed under the umbrella of PTSD. They cannot. There are several subsets of symptoms that arise following trauma. It is true that many individuals present more with the classical symptoms of PTSD while many others, though fulfilling these criteria, still present a plethora of other symptoms (including major depression and psychotic symptoms); still others relate the onset of their symptomatology to a variety of trauma that cooccurred or followed each other, leading to a complex picture not readily identifiable under the present concept of PTSD. Others still are constantly exposed to the ‘traumatic’ experience. It would be simplistic to think that we could find a criteria-based category that will address all these issues; however, and by analogy, it would be as simplistic to find an entity that would encapsulate all symptoms secondary to direct ‘physical’ trauma; these range from bruises to wounds, fracture of the spleen, subdural haematoma, chronic pain, broken bones, blindness and so on. To think that trauma will affect only one area of the brain with a highly specific set of symptoms is not realistic. On the other hand, since physiology is essential in developing novel treatments, an understanding of how the brain responds to psychological trauma would undoubtedly contribute to better treatment; furthermore, the future might witness a revolution not only in the way we handle memories but also in how to code them or even change them, a path that is all too clear to those who observes the evolution of this field.
There are few opportunities that will provide us such a substantial opportunity for shaping the future of the study of traumatic stress as that created by the publication of DSM-5. The challenge involved is how to best distill the epidemiological and longitudinal research, published since the formulation the DSM-IV PTSD diagnostic criteria in 1994, without causing any major unintended consequences. In his discussion of the proposed criteria, Friedman states how at its core, PTSD is a condition manifest during an individual’s interaction with interpersonal and physical environments; in essence, an information-processing disorder. A test of diagnostic criteria is the need for a high degree of inter-rater reliability and diagnostic specificity and sensitivity, different properties than describing the core characteristics of a disorder. These requirements represent a difficult goal.

Considerable thought has gone into how to reformulate the stressor criterion because it is the critical entry to the diagnosis. In particular, the dropping of Criterion A2 is welcomed because of the complexity of the affective response at the times of trauma exposure and particularly amongst groups such as police and combat troops whose training is likely to suppress the nature of their immediate awareness of affect [17]. Furthermore, the importance of the range of emotions related to traumatic events, such as guilt, shame, disgust and horror, has been underexplored. In this vein, Panksepp [18] has stressed the need for a clearer elucidation of the precise categorisation of the nature of emotion because it logically, from an evolutionary perspective, underpins cognition.

A caution needs to be made about one of the proposed modifications that emphasise the temporal rather than the aetiological relationship between the A1
and B–F PTSD symptoms. A recent meta-analysis of delayed-onset PTSD [19] reviewed longitudinal studies and in the combined population found that 24.8% had delayed-onset PTSD. These data showed that the proportion of individuals with delayed-onset PTSD was larger when the duration of follow-up was longer, particularly amongst military populations. Hence, the wording of the temporal relationship between traumatic exposure and symptom development needs to be carefully sculptured to address the importance of delayed-onset PTSD, and minimising this delayed temporal relationship and symptom development would be a mistake.

A related issue that appears to have had little attention in the deliberations about the stressor criterion is the role of multiple trauma exposures as a cause of PTSD. Evidence demonstrates that individuals who have repeated exposure to traumatic stresses have an increased probability of suffering from PTSD through further exposure [20–22]. The evidence supports the role of an underlying sensitisation with subsequent trauma exposures [23, 24]. Hence the definition of the stressor criterion needs to consider the role of sensitisation in those groups that have multiple trauma exposures, such as emergency service workers and military personnel exposed to combat.

The proposal to include the AD/PTSD subtype in order to address the issue of subsyndromal PTSD is critically dependent on the definition of ‘disproportional distress’ following exposure to a traumatic event. Such a definition demands consensus about what is typical in the aftermath of traumatic events. The benchmark presented by Friedman is that ‘most people exhibit distress during the acute aftermath of traumatic exposure’, a position partly and erroneously based on longitudinal studies [25] of a treatment-seeking population. Such a patient population is not a valid group for drawing conclusions about normal patterns of distress after events as these are the individuals who are predictably going to have high levels of acute symptoms: the motivation to seek assistance. Further research has examined symptom progression from the acute post-trauma phase and shows clearly that the majority of injury survivors have low symptom levels in the acute setting and that this pattern persists over time [26]. Further evidence from aggregated data sets of disasters [27] shows that a stable mild reaction is the most common symptomatic pattern, being present in 34.5% of the combined samples, and that symptoms often escalate in those with higher levels of symptoms [26–28]. If an AD subtype is to be included, a more considered definition of the expected ‘normal’ response is required.

The gatekeeper to PTSD DSM-IV was the avoidance criterion, as these symptoms had the lowest rates of endorsement, an issue that created substantial differences in prevalence estimates from ICD-10 [29]. Therefore, reformulation of the avoidance and estrangement symptoms will be a benchmark against which the new criteria are likely to be judged, given that will be compared with the competing ICD-10 diagnostic criteria. An important departure from DSM-IV is the decision to separate the avoidance symptoms (C1 and C2) from those indicative
of catastrophic or maladaptive appraisals. Friedman refers to the role of cognitive behaviour theory in informing this decision. However, the introduction of theoretic models takes the field back to the era of DSM-I and II, which were dominated by the psychoanalytical concept of neurosis, a relationship actively broken in DSM-III. This decision may also have unintended consequences that perhaps require more consideration from a theoretical and clinical perspective. First, True et al. [30] showed that individuals who develop PTSD in the military and emergency services temperamentally tend to place themselves in harm’s way. Such individuals do not characteristically use avoidance as a coping mechanism. Hence, requiring such symptoms in the diagnostic criteria may lead to an underestimation of PTSD in these groups, a dilemma addressed in ICD-10 by introducing the concept of ‘preferred’ avoidance.

Second, identifying avoidance linked to a trauma can lead to problems in inter-rater reliability. The ability of individuals to accurately report avoidance directly linked to the trauma in question presumes explicit knowledge of the association. For example, in a 21-year longitudinal follow-up study of children exposed to a bushfire disaster [31], many participants did not understand the link between their weather phobia and the exposure to the storm that caused the bushfires, the origin of these symptoms. Hence, avoidance phenomena have the substantial potential to have high rates of false negatives because individuals often do not recognise the traumatic origins of their avoidance behaviour.

A further phenomenological issue is that avoidance phenomena are only one of the secondary responses to the distress arising from traumatic memories and hyperarousal [32]. Other phenomena that mitigate the arousal response include dissociation and numbing. Creating avoidance as a necessary symptom cluster has the potential to exclude individuals from the PTSD diagnosis (e.g. police) who temperamentally do not avoid highly distressing circumstances, even though they may have substantial levels of other symptoms. Designating the primacy of avoidance behaviour as a separate set of criteria is also partially driven by the conceptual importance placed on addressing avoidance in behaviour therapy, potentially opening it up to the criticism that it is not atheoretical.

A related issue is the welcomed introduction of post-traumatic reckless behaviour and post-traumatic aggressive behaviour into the hyperarousal criterion. The question arises as to whether, alternatively, these should be included as part of the avoidance criterion. From a psychodynamic perspective, aggression is an emotional state that tends to suppress fear. Similarly, reckless behaviour is a counterphobic response, demonstrating a disregard for consequences and seeking increased arousal, replacing avoidance with risk-taking. As these behaviours may potentially represent the opposite polarity of avoidance, it would be clinically more logical to include them in the new specific avoidance criterion.

Perhaps, before avoidance behaviour is separated into a specific subcategory, this decision should be assessed in epidemiological samples. Introducing the
proposed avoidance criterion may have the unintended consequence of increasing false-negative rates for PTSD, particularly in emergency service and military populations. Friedman’s chapter is the welcomed entrance into a brave new age with all its unanticipated risks and possibilities.

REFERENCES


