

Acute Stress Disorder

DSM-5-TR 308.3 | ICD-10-CM F43.0

Marked stress symptoms lasting 3 days to 1 month after trauma, spanning intrusion, dissociation, avoidance, negative mood, and arousal.

PREVALENCE AFTER TRAUMA

~6-33% by event type

DIAGNOSTIC WINDOW

3 days to 1 month post-event

SEX RATIO

Higher in females

PROGRESSION

~50% go on to develop PTSD

CLINICAL PICTURE

- The presentation blends intrusion, negative mood, dissociation, avoidance, and arousal without requiring symptoms from every category.
- Dissociative features are prominent early: emotional numbing, reduced awareness of surroundings, derealization, and dissociative amnesia.
- Sleep disturbance, hypervigilance, exaggerated startle, and irritable or aggressive behavior appear within days of the index event.
- Patients most often surface in emergency departments, trauma centers, and primary care rather than in mental health settings.
- Functional collapse can be abrupt, with inability to work, drive, sleep alone, or return to the location where the trauma occurred.
- Many people recover spontaneously, so the diagnosis flags acute distress needing support rather than an inevitable path to PTSD.

DIAGNOSTIC CRITERIA AT A GLANCE

- Exposure to actual or threatened death, serious injury, or sexual violence, directly, as a witness, by learning of it, or through repeated occupational exposure.
- Requires at least nine symptoms drawn from five categories: intrusion, negative mood, dissociation, avoidance, and arousal.
- Duration is 3 days to 1 month after the trauma; beyond one month the presentation must be reassessed for posttraumatic stress disorder.
- Symptoms cause clinically significant distress or impairment and are not attributable to substances, medication, or traumatic brain injury.
- Unlike PTSD, no minimum count is required within any single cluster, which permits dissociation-heavy or arousal-heavy symptom profiles.

NEUROBIOLOGY & PHYSIOLOGY

- An acute noradrenergic and catecholamine surge at the time of trauma over-consolidates emotional memory and predicts later PTSD severity.
- Elevated heart rate in the first 24-48 hours after trauma is among the more replicated biological predictors of subsequent PTSD.
- A blunted cortisol response at the time of trauma is associated with failure to contain the sympathetic stress response afterward.
- Amygdala hyperreactivity with weak ventromedial prefrontal regulation is already measurable acutely, mirroring the chronic PTSD pattern.
- Peritraumatic dissociation appears to reflect prefrontal overmodulation of limbic arousal rather than the more typical underregulation.
- Prior trauma, female sex, low cortisol reactivity, and comorbid traumatic brain injury all raise the probability of progression to PTSD.

PSYCHOLOGICAL MECHANISMS

- Acute fear conditioning to trauma cues is established within days, and avoidance that begins early blocks the natural course of extinction.
- Catastrophic appraisals of early symptoms as evidence of permanent damage strongly predict symptom persistence beyond one month.
- Peritraumatic dissociation impairs encoding and integration of the trauma memory, producing the fragmented recall seen later in PTSD.
- Perceived social support and validation during the first weeks are among the strongest modifiable protective factors against chronicity.
- Rumination about the event and about one's own conduct during it converts a time-limited stress reaction into a chronic disorder.

DIFFERENTIAL & COMORBIDITY

- Beyond one month the diagnosis becomes PTSD, while adjustment disorder applies when the stressor or the response falls short of criteria.
- Rule out concussion and traumatic brain injury, substance intoxication or withdrawal, and delirium in medically injured patients.
- Brief psychotic disorder with marked stressors, and normal acute stress reactions without impairment, are the key boundary conditions.
- Comorbid depression, panic attacks, and escalating alcohol use develop quickly after trauma and warrant direct, repeated screening.
- Assess suicide risk, means access, and ongoing interpersonal safety, especially after assault or intimate partner violence.

Treatment EVIDENCE-BASED MANAGEMENT

PHARMACOLOGIC

- No medication is FDA-approved for acute stress disorder; pharmacotherapy targets specific symptoms rather than the syndrome itself.
- Avoid **benzodiazepines** in the acute period, since early use is associated with higher rather than lower rates of later PTSD.
- Short-term sleep support with **trazodone 25-100 mg** or **prazosin 1-5 mg at bedtime** for nightmares is a reasonable bridge.
- Do not use routine **propranolol** or hydrocortisone prophylaxis; trial evidence is inconsistent and neither is standard of care.
- Start an **SSRI** only when depression or panic is prominent, or when symptoms persist past one month and meet PTSD criteria.

PSYCHOTHERAPY

- Trauma-focused CBT** delivered over 5-6 sessions beginning about two weeks post-trauma reduces progression to PTSD.
- Do not use single-session psychological debriefing; **critical incident stress debriefing** shows no benefit and may increase symptoms.
- Psychological First Aid** is the appropriate immediate intervention: safety, calming, connectedness, self-efficacy, and hope.
- Watchful waiting with structured follow-up at two and four weeks is appropriate for mild presentations that are already improving.
- Brief **prolonged exposure** started in the emergency department within hours of trauma reduced later PTSD in controlled trials.

ADJUNCT & SUPPORTIVE

- Screen with the **Acute Stress Disorder Scale** or **PCL-5** and repeat at one month to determine whether PTSD has emerged.
- Restore sleep, nutrition, and daily routine early, since sleep disruption in the first week predicts chronic symptom trajectories.
- Mobilize practical support including housing, finances, legal advocacy, and reconnection with family and community networks.
- Limit repeated media exposure to the event, which sustains physiologic arousal and reinforces intrusive imagery.
- Arrange definite scheduled follow-up rather than as-needed return, because most patients will not self-refer despite ongoing distress.

- ☆ **CLINICAL PEARLS**
- Three days to one month is the window; past that, reassess for PTSD.
 - Skip debriefing and skip benzodiazepines; both can worsen outcomes.
 - Nine of fourteen symptoms, with no per-cluster minimum, unlike PTSD.

References

American Psychiatric Association. (2022). *Diagnostic and statistical manual of mental disorders* (5th ed., text rev.).

<https://doi.org/10.1176/appi.books.9780890425787>

Bryant, R. A. (2011). Acute stress disorder as a predictor of posttraumatic stress disorder: A systematic review. *The Journal of Clinical Psychiatry*, 72(2), 233-239.

National Institute for Health and Care Excellence. (2018). *Post-traumatic stress disorder* (NICE guideline NG116).

<https://www.nice.org.uk/guidance/ng116>

National Institute of Mental Health. (n.d.). *Post-traumatic stress disorder*. U.S. Department of Health and Human Services.

<https://www.nimh.nih.gov/health/topics/post-traumatic-stress-disorder-ptsd>

Sadock, B. J., Sadock, V. A., & Ruiz, P. (2021). *Kaplan & Sadock's synopsis of psychiatry* (12th ed.). Wolters Kluwer.

Stahl, S. M. (2021). *Stahl's essential psychopharmacology* (5th ed.). Cambridge University Press.

U.S. Department of Veterans Affairs & U.S. Department of Defense. (2023). *VA/DoD clinical practice guideline for the management of posttraumatic stress disorder and acute stress disorder* (Version 4.0). <https://www.healthquality.va.gov/guidelines/MH/ptsd/>

NOTE

References are formatted in APA 7th edition style with hanging indents. Diagnostic terminology, criteria structure, and coding follow the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed., text rev.; APA, 2022); criteria are paraphrased for instructional use and are not reproduced verbatim. Verify all citations against the original sources before use in academic work, and confirm medication dosing against current prescribing information.