A Closer Look at PTSD (Referenced)

Introduction

I have written this document from my reading of the literature alongside my experience as a GP with an interest in mental health. Trauma is an area that affects many areas of mental health across multiple diagnostic categories. I have focused on the diagnosis of PTSD here and aimed to outline evidence on what is happening in the brain when somebody experiences a traumatic event as well as PTSD itself as a diagnosis. Where the evidence is uncertain or contested I have tried to make this clear. The guide covers PTSD across the lifespan - where children and adults differ I've stated the differences.

This guide is the referenced companion to the PTSD patient leaflet. It is for interested patients and clinicians to read more and have a look at original sources. It is not intended to be exhaustive or any kind of definitive guide. It is based on my own experience and interpretation of the evidence and is what I would share if I had unlimited consultation time.

Part One: Understanding PTSD

What kind of diagnosis PTSD is

PTSD is a widely recognised diagnosis, defined in both DSM-5-TR and ICD-11 by a characteristic symptom pattern — re-experiencing, avoidance, hyperarousal and negative alterations in mood and cognition — following exposure to a traumatic event.[1,2,3] When trained clinicians use agreed criteria and structured measures such as the CAPS-5 they tend to agree reasonably well, and the diagnosis picks out a group with substantial needs and raised risk.

What makes PTSD unusual within psychiatry is that the definition names an external cause — the trauma — rather than resting on symptoms alone. That gives it a clearer rationale than many diagnoses, but it remains a description of a pattern rather than a disease with a single physical fault: there is no scan or blood test that confirms or excludes it in an individual.[2] It is a helpful diagnostic category that can lead to validation, treatment and support of various kinds, and remains descriptive rather than explanatory in nature. I have looked at some of the objective scientific findings relating to the effects of trauma later in the document.

Complex PTSD

ICD-11 now formally distinguishes complex PTSD (CPTSD) from PTSD.[3] CPTSD requires the core PTSD features plus persistent 'disturbances in self-organisation': difficulties with affect regulation, a negative self-concept (pervasive shame, guilt or worthlessness), and disturbances in relationships. It typically follows prolonged or repeated trauma from which escape was difficult — childhood abuse and neglect,

domestic violence, captivity — though the diagnosis is made on the symptom profile, not the trauma history alone. This is a meaningful development because NICE NG116 (2018) predates the formal category, and C-PTSD generally calls for a longer, phased approach (stabilisation and affect regulation before, and alongside, memory-focused work) rather than brief intervention.[2]

Who develops it

Most people exposed to trauma do not develop PTSD; recovery without treatment is the more common course. Lifetime prevalence in the general population is in the region of 4–8%, higher in groups with greater trauma exposure such as refugees and veterans. Risk is shaped by the nature and severity of the event, prior trauma, peri-traumatic response, and the social and material support available afterwards.[2]

What is happening in the brain and body

During a traumatic event the brain's threat circuitry takes over. The amygdala, which flags danger and drives the fight-or-flight response, becomes more reactive, while the medial prefrontal cortex — needed to put a frightening moment into context and regulate the alarm — and the hippocampus — needed to tie an experience to its time and place — are relatively suppressed by the surge of stress hormones. This pattern of amygdala hyperactivity alongside prefrontal and hippocampal hypoactivity is one of the more consistently replicated findings in PTSD neuroimaging, seen across multiple meta-analyses.[26,27]

The hippocampus is what normally files an experience away with its context intact — where it happened, when, and that it is now over. Under the surge of arousal and stress hormones that accompanies a traumatic event, hippocampal function is impaired at exactly the point the memory is being laid down, while the amygdala's role in emotional encoding remains intact or enhanced. The result is a memory that is vivid and easily triggered but poorly contextualised — well remembered as sensation, but not accurately filed as historical. This is the neurobiological counterpart to Ehlers and Clark's and Brewin's accounts below, and the likely reason trauma memories intrude as sensory fragments rather than as a story with a beginning, middle and end.[28,29]

The stress-hormone picture is less clear. The popular account has trauma leaving someone permanently flooded with cortisol, but the evidence for the hypothalamic-pituitary-adrenal (HPA) axis in PTSD is mixed: one large meta-analysis found no overall difference in basal cortisol between people with PTSD and controls, though a subgroup — in this case women, and those with an abuse history — actually showed lower levels.[30,31]

The autonomic nervous system shows a more consistent signature: reduced heart-rate variability (HRV) and reduced vagal tone at rest, reflecting a system tilted toward sympathetic dominance and away from the parasympathetic 'rest and digest' state. Meta-analyses confirm this reliably across studies, and it maps directly onto

hyperarousal— the jumpiness, poor sleep and constant sense of threat that patients describe. It is also the rationale for the arousal-regulation work described below: HRV biofeedback and paced breathing act directly on this branch of the physiology, even though, as noted, they do not process the memory itself.[32]

This circuitry describes a robust average pattern across group studies — there is the same heterogeneity here as everywhere else in PTSD research, and not every patient will show every feature.

What happens — several overlapping models

There is good evidence that many treatments help, and several plausible, overlapping psychological accounts of why, but no single agreed mechanism. The neurobiology set out above is one layer; alongside it are four psychological accounts that can be useful in understanding trauma:

Ehlers and Clark's cognitive model. PTSD persists when the trauma is processed in a way that produces a sense of current threat — through excessively negative appraisals and a poorly elaborated, disjointed memory that is easily triggered and feels 'present'. Therapy targets the appraisals and the memory.[4]

Brewin's dual representation theory. Trauma memories are held in two systems — a verbally accessible, contextualised representation and a sensory, image-based one. Flashbacks reflect sensory memory that has not been bound to its context in time and place; treatment helps integrate the two. This is a more formal version of the clinical intuition that the memory needs 'filing' as past.[5]

Extinction and inhibitory learning. The learning-theory basis for exposure: the trauma memory is not erased but new, safety-based learning is built that competes with and inhibits the old fear response. Craske's work reframes exposure as maximising this inhibitory learning rather than simply habituating arousal.[6]

Memory reconsolidation. When a memory is retrieved it can briefly become labile and open to updating before being stored again; new information introduced in that window may change the memory itself.[7,8] This is one of the more modern accounts and the one several brief techniques invoke. It is genuinely promising, but the research is still in an early stage[9]

These accounts overlap quite a lot — all describe a memory being brought to mind under conditions of safety and then changing — and the practical implication is the same: controlled re-engagement with the memory, with arousal kept manageable, is what helps. It has been argued that multiple approaches such as the rewind technique, EMDR and tapping share a mechanism: lowering arousal and splitting attention (between the memory and the eye movement, tapping or effort of watching the memory backwards for example) lets the memory be re-experienced in a context the brain registers as present and safe, so it is re-coded and updated as having happened in the past.[10] The core of that idea — arousal reduction plus dual attention

enabling recontextualisation — fits alongside the models above, and is very close to the leading laboratory account of EMDR's eye movements (described later).

A note on 'the body keeps the score' and polyvagal theory

Popular trauma writing, including van der Kolk's influential book, has done real good in conveying that trauma affects the body and the autonomic nervous system, not only the mind or conscious memory, and that point is well supported.[24] Some of the framework it popularised — in particular polyvagal theory — is more contested, and parts of the original theory have been challenged.

Part Two: What helps

Psychological therapy is first-line

NICE NG116 is clear that psychological treatment comes before medication. For adults presenting more than a month after the trauma, it recommends offering an individual trauma-focused CBT intervention (for example cognitive therapy for PTSD, cognitive processing therapy, or prolonged exposure), and considering EMDR, particularly where the person presents more than three months after a non-combat trauma or prefers it.[2,25] A typical course is 8–12 sessions, more for multiple or complex trauma. In the first month NICE advises active monitoring rather than routine early intervention, and psychological debriefing should not be offered — it does not prevent PTSD and may do harm.[2]

Across meta-analyses the trauma-focused therapies show medium-to-large effect sizes and are the best-evidenced treatments available, though they are therapist-time intensive and drop-out is not trivial.[13] It can be helpful to explain to patients that the a temporary increase in distress during the work is expected and time-limited – see the patient leaflet for more information.

EMDR and the eye-movement question

EMDR is effective for PTSD; what remains unsettled is whether the eye movements themselves add anything. The leading laboratory explanation is working-memory taxation: holding a vivid memory in mind while performing a demanding dual task (eye movements, but also tapping or counting) leaves fewer resources for the memory, which becomes less vivid and less emotional and is re-stored that way.[11] A clinical trial found fixating on a therapist's moving or non-moving hand produced equivalent symptom reduction, so it is not clear whether the clinical benefit comes from the eye movements specifically.[12]

The rewind technique

The rewind technique (Muss' development of a visual–kinaesthetic dissociation protocol, refined within the Human Givens approach) is used across the NHS, the third sector and private practice in the UK. Its evidence base has advanced:

alongside the Adams and Allan multi-site pilot and the Rwanda group-session work,[14,16] there is now an exploratory randomised controlled trial reporting a large effect size (Cohen's d around 1.05) at eight weeks, maintained at sixteen.[15] A separate but related protocol, reconsolidation of traumatic memories (RTM), has also been tested in a randomised trial in veterans.[17]

The RCT is small (around 40 participants), exploratory, compared against a waitlist (which tends to inflate effect sizes), and conducted by investigators close to the method, including its originator; there is as yet no head-to-head comparison with trauma-focused CBT or EMDR, and rewind is not recommended in NICE.[2,15] So the fair summary is that it has moved from anecdote to a preliminary efficacy signal — promising, plausibly working through the same arousal-reduction and reconsolidation routes as the established therapies, but not yet established to the same standard. Its attractions are that it is brief, non-intrusive, and does not require detailed verbal disclosure.

EFT / 'tapping'

Emotional freedom techniques have some trial-level support and enthusiastic advocates,[18] but the literature carries real methodological weaknesses and conflicts of interest, and the consensus reading is that any benefit derives from the exposure and non-specific therapeutic factors rather than from the meridian-tapping rationale, which has no credible basis. It is not recommended in guidelines and I would not offer it ahead of the evidence-based therapies.

Settling the nervous system: HRV biofeedback and HeartMath

Arousal-regulation work is a reasonable adjunct, distinct from memory processing. Its evidence-based core is slow, paced breathing at around the 0.1 Hz resonance frequency and heart-rate-variability (HRV) biofeedback, which raise vagal tone and have decent support as adjuncts for stress, anxiety and low mood.[19] For PTSD specifically the evidence is thinner and mostly small-scale — for example a randomised trial of HRV biofeedback for post-deployment PTSD prevention, and earlier coherence-training work in veterans.[20,21]

HeartMath is the best-known branded version of this, and is what I trained in (including its trauma-focused training). Two caveats keep it honest. First, much of the strongest-sounding evidence comes from researchers affiliated with the organisation, and the broader brand claims — 'heart coherence,' the heart's electromagnetic field, 'global coherence' — run well beyond what mainstream physiology supports; the defensible mechanism is ordinary vagal regulation through paced breathing, which is available without proprietary kit. Second, this is arousal regulation, not trauma processing: it lowers baseline arousal and can make the rest of the work more tolerable, but it does not update the memory and should not be offered as a substitute for trauma-focused therapy.

As an aside on feasibility rather than efficacy: the psychotherapist and HeartMath trainer Majd Kamalmaz built brief self-regulation programmes for Syrian refugees in Lebanon, conducting tens of thousands of needs assessments across several centres before he was detained in Syria in 2017; his death was announced in 2024. The widely repeated figure of a near-90% symptom-reduction rate is an uncontrolled, self-reported programme statistic, not efficacy data — but the work is a striking demonstration that brief, teachable regulation tools can be delivered acceptably in extreme, low-resource settings, which is a genuine point in their favour as an adjunct.

Medication

Drug treatment is second-line. Where a patient declines or cannot access psychological therapy, or has prominent comorbid depression or anxiety, NICE supports venlafaxine or an SSRI (sertraline or paroxetine), used alongside rather than instead of psychological treatment; in UK practice these are off-label for PTSD.[2] The average effect is modest, and medication does not process the trauma memory. For severe or treatment-resistant presentations a specialist may add an antipsychotic, with the usual caution about benefit versus harm.

For trauma-related nightmares the picture is genuinely mixed. The largest randomised trial (PACT) was negative,[22] which prompted a downgrading of prazosin; yet subsequent meta-analyses still find it improves nightmares and insomnia, though not overall PTSD symptoms.[23] A reasonable position is that prazosin is worth considering for distressing trauma nightmares specifically, with realistic expectations, and that imagery rehearsal therapy is a comparably effective non-drug option for the same problem.

In children and young people, NICE is explicit that drug treatments should not be offered for the prevention or treatment of PTSD in under-18s.[2]

Children and young people

Presentation varies with development. Young children may show trauma through repetitive play and re-enactment, regression (for example bed-wetting), separation anxiety, sleep disturbance, irritability or withdrawal rather than clearly articulated re-experiencing; adolescents may present more like adults but also with anger, risk-taking or self-blame. NICE recommends individual trauma-focused CBT adapted to the child's age and development as first-line, and considers EMDR only where the child has not responded to or engaged with trauma-focused CBT — a position EMDR advocates contest, but it is the current guidance.[2]

Caregiver involvement and a stable, safe environment are central, and parents often need support and reassurance that the child's reactions are understandable and treatable.

The service context

Access is the practical bottleneck. Trauma-focused therapy is effective but therapist-time intensive, and waits for treatment can be long, with NHS Talking Therapies handling milder presentations and specialist services needed for complex PTSD and high-risk cases. It helps to frame these as pressures on the system, and to begin support, stabilisation and arousal-regulation work according to availability of local resources.

References

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed, text revision (DSM-5-TR). Washington, DC: American Psychiatric Association Publishing; 2022.
2. National Institute for Health and Care Excellence. Post-traumatic stress disorder. NICE guideline NG116. London: NICE; 2018.
3. World Health Organization. International Classification of Diseases, 11th Revision (ICD-11): 6B40 Post-traumatic stress disorder; 6B41 Complex post-traumatic stress disorder. Geneva: WHO; 2019/2022.
4. Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. Behav Res Ther. 2000;38(4):319–345.
5. Brewin CR, Gregory JD, Lipton M, Burgess N. Intrusive images in psychological disorders: characteristics, neural mechanisms, and treatment implications. Psychol Rev. 2010;117(1):210–232.
6. Craske MG, Treanor M, Conway CC, Zbozinek T, Vervliet B. Maximizing exposure therapy: an inhibitory learning approach. Behav Res Ther. 2014;58:10–23.
7. Nader K, Schafe GE, LeDoux JE. Fear memories require protein synthesis in the amygdala for reconsolidation after retrieval. Nature. 2000;406(6797):722–726.
8. Schiller D, Monfils MH, Raio CM, Johnson DC, LeDoux JE, Phelps EA. Preventing the return of fear in humans using reconsolidation update mechanisms. Nature. 2010;463(7277):49–53.
9. Lane RD, Ryan L, Nadel L, Greenberg L. Memory reconsolidation, emotional arousal, and the process of change in psychotherapy. Behav Brain Sci. 2015;38:e1.
10. Griffin J. PTSD: why some techniques for treating it can work so fast. Human Givens Journal. 2005;12(3) (updated 2025); see also Griffin J, Tyrrell I. Human Givens. Chalvington: HG Publishing; 2003.
11. van den Hout MA, Engelhard IM. How does EMDR work? J Exp Psychopathol. 2012;3(5):724–738.
12. van Veen SC, Kang S, van Schie K, et al. On EMDR: measuring the working memory taxation of various types of eye (non-)movement conditions. J Behav Ther Exp Psychiatry. 2019;65:101494.
13. Lewis C, Roberts NP, Andrew M, Starling E, Bisson JI. Psychological therapies for post-traumatic stress disorder in adults: systematic review and meta-analysis. Eur J Psychotraumatol. 2020;11(1):1729633.
14. Adams S, Allan S. Muss' rewind treatment for trauma: description and multi-site pilot study. J Ment Health. 2018;27(5):468–474.

15. Astill Wright L, Barawi K, Kitchiner NJ, et al. Rewind for posttraumatic stress disorder: a randomised controlled trial. Depress Anxiety. 2023;2023:6279649.
16. Utuza AJ, Joseph S, Muss D. Treating traumatic memories in Rwanda with the rewind technique: two-week follow-up after a single group session. Traumatology. 2012;18(1):75–78.
17. Gray R, Budden-Potts D, Bourke F. Reconsolidation of traumatic memories for PTSD: a randomized controlled trial of 74 male veterans. Psychother Res. 2019;29(5):621–639.
18. Sebastian B, Nelms J. The effectiveness of emotional freedom techniques in the treatment of posttraumatic stress disorder: a meta-analysis. Explore (NY). 2017;13(1):16–25.
19. Lehrer PM, Gevirtz R. Heart rate variability biofeedback: how and why does it work? Front Psychol. 2014;5:756.
20. Pyne JM, Constans JI, Nanney JT, et al. Heart rate variability and cognitive bias feedback interventions to prevent post-deployment PTSD: results from a randomized controlled trial. Mil Med. 2019;184(1–2):e124–e132.
21. Ginsberg JP, Berry ME, Powell DA. Cardiac coherence and posttraumatic stress disorder in combat veterans. Altern Ther Health Med. 2010;16(4):52–60.
22. Raskind MA, Peskind ER, Chow B, et al. Trial of prazosin for post-traumatic stress disorder in military veterans. N Engl J Med. 2018;378(6):507–517.
23. Zhang Y, Ren R, Sanford LD, et al. The effects of prazosin on sleep disturbances in post-traumatic stress disorder: a systematic review and meta-analysis. Sleep Med. 2020;67:225–231.
24. van der Kolk BA. The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma. New York: Viking; 2014.
25. Watkins LE, Sprang KR, Rothbaum BO. Treating PTSD: a review of evidence-based psychotherapy interventions. Front Behav Neurosci. 2018;12:258.
26. Shin LM, Rauch SL, Pitman RK. Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. Ann N Y Acad Sci. 2006;1071:67–79.
27. Etkin A, Wager TD. Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. Am J Psychiatry. 2007;164(10):1476–1488.
28. Elzinga BM, Bremner JD. Are the neural substrates of memory the final common pathway in posttraumatic stress disorder (PTSD)? J Affect Disord. 2002;70(1):1–17.
29. Whalley MG, Kroes MC, Huntley Z, Rugg MD, Davis SW, Brewin CR. An fMRI investigation of posttraumatic flashbacks. Brain Cogn. 2013;81(1):151–159.
30. Meewisse ML, Reitsma JB, de Vries GJ, Gersons BP, Olff M. Cortisol and post-traumatic stress disorder in adults: systematic review and meta-analysis. Br J Psychiatry. 2007;191:387–392.
31. Pan X, Wang Z, Wu X, Wen SW, Liu A. Salivary cortisol in post-traumatic stress disorder: a systematic review and meta-analysis. BMC Psychiatry. 2018;18:324.

32. Ge F, Yuan M, Li Y, Zhang W. Posttraumatic stress disorder and alterations in resting heart rate variability: a systematic review and meta-analysis. Psychiatry Investig. 2020;17(1):9–20.