REVIEW ARTICLE

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The Neurobiology of Acute Emotional Dysregulation in Emergency Trauma Care: A Psychophysiological Perspective

Mojtaba Khaksarian[1], Azita Keshavarz[2], Saba Hassanvandi[3], Hamid Ahmadi[4]

[1]*Department of Physiology, Faculty of Medicine, Lorestan University of Medical Sciences, Khorramabad, Iran.*
[2]*Department of Psychology, Torbat-e Jam Branch, Islamic Azad University, Torbat-e Jam, Iran.*
[3]*Department of psychology and Conseling, Farhangian University, Tehran, Iran.*
[4]*Department of Psychiatry, Faculty of Medicine, Lorestan University of Medical Sciences, Khorramabad, Iran.*

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Abstract

Frontal alpha asymmetry (FAA) is an electroencephalographic marker that reflects approach-avoidance motivation in the brain. In this review, we consolidate divergent findings on FAA from studies on people with PTSD, individuals with trauma exposure but no PTSD diagnosis (trauma-exposed healthy controls), and people with no significant trauma history (non-trauma healthy controls). FAA has been examined during tasks combining emotional stimuli (threatening and neutral images) with cognitive demands such as distraction or working memory load.

Notably, PTSD is associated with increased left frontal activity (linked to approach motivation) in response to threatening images when cognitive demands are low. The trauma-exposed individuals without PTSD group shows a similar but attenuated pattern. In contrast, NTHC tends to show right frontal activity (associated with withdrawal tendencies) regardless of stimulus type. Interestingly, this right-lateralized pattern—which is absent in the PTSD group—tends to disappear under higher cognitive load, such as when filtering out distractions. Under these conditions, the PTSD group maintains a left frontal bias even for neutral stimuli, while the other two groups either reduce left-sided activity or maintain a balanced pattern.

Overall, these findings suggest that PTSD alters the typical response to threat and impairs emotion regulation under cognitive strain. Simply having a trauma history may produce a milder form of this approach bias without meeting criteria for full disorder. We discuss implications for prefrontal threat processing and the potential of neurofeedback to retrain these patterns.

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Introduction

Social harms are one of the major concerns of human societies today. Every society, in accordance with its own conditions, culture, transition and development process, growth and decline, faces a variety of problems that have undeniable effects on the growth and progress of society. One of the biggest problems that has always plagued human societies with its problems and sinister consequences is the phenomenon of addiction, which is both a consequence and a cause of many other social problems and harms (1). One of the important issues of this disorder is the frequent relapses of patients, which causes the quality of life of addicts to decline compared to other members of society. Most treatment programs focus on reducing or stopping drug use; however, substance-dependent patients also struggle with

Correspondence:
Hamid Ahmadi
E-mail: ahmadi3781@gmail.com

numerous other problems, many of which may predate drug use. These problems may even be important causes of drug use in the first place (2). Thus, in addition to targeting reduction and cessation of drug use, treatment programs should also consider relevant and important psychological variables related to recovery management, reduction of addiction relapse, and even prevention (3).

One of the most direct and difficult clinical consequences of traumatic injury is acute emotional dysregulation, especially in the high-stakes context of emergency trauma care. Patients frequently show extreme physiological hyperarousal, emotional instability, dissociation, and a reduced ability to control affective reactions after potentially fatal events like car accidents, assaults, or falls (1, 2). These symptoms don't just make initial stabilization and triage harder—they also point to poor long-term recovery, including the development of PTSD, complex PTSD (C-PTSD), and other secondary psychiatric conditions (2, 3).

Acute emotional dysregulation comes from a sudden and ongoing disruption of the autonomic nervous system (ANS). From a neurobiological perspective, this shows up as sympathetic overactivity and parasympathetic withdrawal. Classic markers of this imbalance include elevated skin conductance level changes (ΔSCL) during trauma recall—even with neutral stories ($b = 5.80–6.88$, $p < 0.05$) (1); ongoing tachycardia during recovery phases in C-PTSD ($p = 0.010$ vs PTSD, $p = 0.036$ vs controls) (2); and reduced high-frequency heart rate variability (HF-HRV) tied to childhood emotional abuse (Cohen's $d = 0.81–0.92$) (4). These physical signs point to deeper dysregulation in prefrontal-limbic circuits, especially less engagement of the dorsomedial prefrontal cortex (dmPFC) during cognitive reappraisal ($\beta = −0.50$, $p = 0.002$ for symptom change) (5).

What makes this even more clinically relevant is that patients present in emergency rooms within hours or days of their trauma, then encounter additional stressors—uncomfortable procedures, diagnostic scans, and lots of social interactions. In trauma-exposed people, is observed higher sympathetic tone (like increased LF/HF ratios after a stressor, $p < 0.05$) (3) and slow parasympathetic recovery (e.g., sustained high heart rate) (2), which can increase agitation, dissociation, and noncompliance with medical care. On top of that, adverse childhood experiences (ACEs) make people more vulnerable by disrupting interoceptive accuracy and ramping up cravings triggered by negative emotional cues ($R^2 = 0.27–0.37$) (6). That sets the stage for long-term emotion dysregulation and substance use disorders over time (7-9).

Even though we know more about these issues now, emergency trauma protocols still mostly focus on physical stabilization. There's not much psychophysiological monitoring happening, and targeted interventions for acute emotional dysregulation are rare. But notably: a growing body of evidence shows that brief, mechanism-informed interventions can actually help. Things like mindfulness-based stress reduction (MBSR), which boosts reappraisal selection ($F = 5.60$, $p = 0.005$) (10); alpha-down neurofeedback, which cuts PTSD severity by more than 30% early on ($dz = 0.71–0.77$) (11); and body-oriented therapies that improve RSA ($\chi^2 = 13.51–17.99$, $p < 0.001$) (9)—all of these can help restore autonomic balance and break those maladaptive cycles early in the game.

This review pulls together findings from 29 empirical studies (2015–2025) that looked at psychophysiological markers (SCL, HRV, RSA, sAA, FAA, fMRI activation) and intervention outcomes in trauma-exposed populations—including PTSD, C-PTSD, TBI, ADHD, NSSI, SUD, and AN. Drawing on all this data, the article aims to: (1) lay out the core neurobiological mechanisms behind acute emotional dysregulation in emergency settings, (2) assess how well key psychophysiological indices can predict and tell apart different outcomes, and (3) offer practical, evidence-based suggestions for weaving mechanism-targeted monitoring and early interventions into emergency trauma care (Tab.1).

Methods

Study Design and Search Strategy

We chose a narrative review for this paper over a systematic review or meta-analysis. Our questions were just too wide-ranging and connected in ways that needed a deeper, more flexible integration of ideas. This approach allowed us to examine evidence from studies across different trauma-exposed populations and focus specifically on a handful of psychophysiological signals that keep coming up: skin conductance level (SCL), heart rate variability (HRV), and frontal alpha asymmetry (FAA). What we really wanted was to pull out some useful, real-world tips for emergency psychological first aid — the kind of things that could actually help someone on the spot during a crisis, not just sit nicely in a journal. We hunted for relevant papers ourselves, focusing on anything published from January 2015 to December 2025. The main places we checked were PubMed, Google Scholar, and PsycINFO — all manual searches, no fancy automation. We used keyword combinations and Boolean operators, including PTSD, emergency care, acute stress, skin conductance, frontal alpha asymmetry, trauma, emotional dysregulation, HRV, and psychophysiology. Plus, every time we landed on a good article, we dug into its references to see if anything else popped up that we'd missed. No, we didn't register a formal protocol anywhere — nothing in

PROSPERO or following strict PRISMA guidelines. The goal wasn't to map every single study ever done; we just aimed for a solid, interpretive wrap-up of what the evidence seems to say so far. Eventually, we settled on 30 empirical studies that actually matched what we were looking for and synthesized them.

Inclusion Criteria and Synthesis Approach

We only included studies that met a few clear criteria: they had to involve people who'd experienced trauma (from things like PTSD, traumatic brain injury, ADHD, substance use issues, and similar groups), measure at least one psychophysiological indicator—things like heart rate variability (HRV), skin conductance level (SCL), respiratory sinus arrhythmia (RSA), EEG, or even fMRI—and look at emotional regulation or dysregulation as a main or secondary focus. In the end, we pulled together 29 studies that fit these rules. We leaned toward ones that had the most direct relevance to acute emotional dysregulation during emergencies or high-stress moments. Since this is more of a descriptive narrative review, we didn't do any formal quality or bias assessment tools (like AMSTAR or QUADAS-2), and we skipped meta-analysis completely. Instead, we synthesized everything narratively—paying attention to things like study methods, who the participants were, sample sizes, and any reported effect sizes when they were available. The goal was to provide a balanced interpretation of the literature with practical relevance for clinical work (Tab.1).

Results

Mechanisms of Neurobiological and Psychophysiological Acute Emotional Dysregulation Following Trauma

Acute emotional dysregulation among trauma survivors stems from a serious imbalance in the autonomic nervous system (ANS). This imbalance typically involves overactivity of the sympathetic nervous system—the one behind the fight-or-flight response—alongside reduced activity of the parasympathetic system, which handles rest and recovery. The result is a state of prolonged hyperarousal, where individuals struggle to process emotions and have a hard time calming down after stress (1, 2) (Fig.1).

These physiological patterns tend to stick around long after the trauma itself, showing up even in neutral, everyday situations with no real threat in sight. Take survivors of motor vehicle accidents, for example—changes in their skin conductance levels while recalling the trauma can strongly predict emotional regulation difficulties down the road. What's especially striking is that these nervous system changes can forecast emotion regulation deficits even when someone isn't actively remembering the traumatic event. In patients with complex PTSD, the pattern is even more pronounced. Recalling the trauma sends heart rate up for everyone, sure (2). But in those with C-PTSD, that elevated heart rate lingers well into the recovery period—long after the memory has passed.

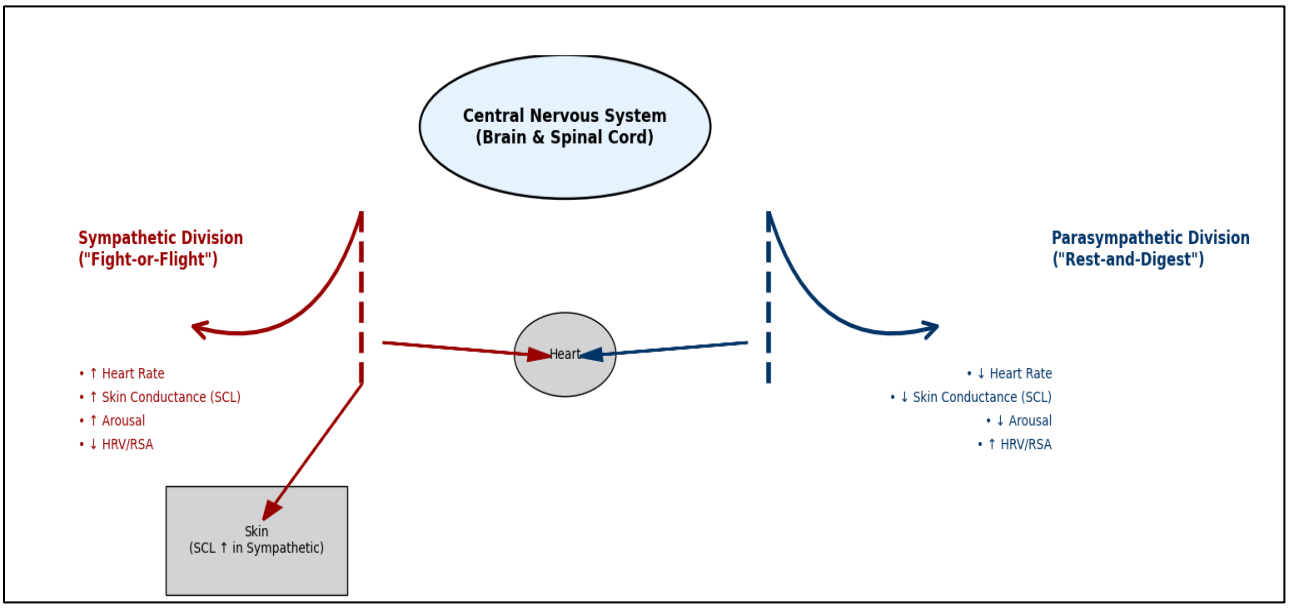

Figure 1. Schematic representation of the autonomic nervous system (ANS) branches and their physiological effects in acute emotional dysregulation following trauma. Sympathetic overactivation (e.g., increased heart rate and skin conductance level) and parasympathetic deficit (e.g., reduced HRV/RSA) are hallmark features observed in emergency trauma care [Ref 1, Ref 14].

Role of Childhood Trauma and Adversity in Emotional Dysregulation

Childhood adverse experiences—commonly called ACEs—can disrupt emotion regulation well into adulthood, and there are a few different ways this plays out biologically. For one, ACEs seem to lower heart rate variability (HRV) precisely when people are trying to manage negative emotions. This drop in HRV goes hand in hand with stronger emotional triggers and more severe Opioid Use Disorder symptoms (6). Interestingly, resting HRV doesn't really differ based on childhood adversity—it's specifically during active emotion regulation that the vulnerability shows up. Take depressed women with a history of childhood emotional abuse. Their high-frequency HRV (HF-HRV)—a solid marker of how well the parasympathetic system is working—stays significantly lower than in women without that history, even after you account for things like breathing rate and general health (4).

Then there's anorexia nervosa. Childhood trauma seems to complicate treatment by exacerbating emotion regulation difficulties. In fact, how bad those difficulties are at the start of treatment—and how much they fluctuate along the way—turns out to be one of the strongest predictors of how well someone responds to treatment (7). And to make things worse, body dissociation (the experience of being disconnected from your own physical sensations) just reinforces the whole cycle, making it even harder to break free from those emotion regulation struggles (12).

Key Psychophysiological Markers in Trauma Contexts

Skin conductance level and its fluctuations are one of the most reliable indicators of sympathetic arousal in trauma survivors. When skin conductance increases during trauma recall—or even during neutral conversations—it usually points to a poorer recovery outcomes (1).

In people with traumatic brain injury, deliberately triggering anger leads to clear jumps in skin conductance and drops in heart rate variability. These individuals also report feeling more aroused and angry than controls. And importantly: higher levels of disinhibition—a common issue after brain injury—make these responses even worse, ramping up negative mood and tension when things get emotionally challenging (13).

Now, with non-suicidal self-injury, the picture gets more complicated. On a subjective level, these individuals feel emotions more intensely. But when you look at physiological measures like resting heart rate variability, there's barely any difference from controls. When they hit an emotional challenge, sure, their negative mood, heart rate, and skin conductance go up—but not more than what you'd see in anyone else (14).

As for long-term abstinent alcoholics, psychosocial stress still hits them hard. Even years after quitting, they show reduced parasympathetic activity (lower high-frequency HRV and respiratory sinus arrhythmia) and faster breathing—clear signs that their sympathetic system is still persistently overactive (15).

Psychophysiological Changes in Specific Populations

In children and adolescents who have sustained traumatic brain injury or extracranial injury, changes in salivary alpha-amylase reactivity are associated with internalizing problems and difficulties with emotional control, while cortisol reactivity proves less informative. Salivary alpha-amylase thus represents a sensitive biological marker of vulnerability following brain injury in young people (16).

Among children with ADHD, lower heart rate variability is linked to greater emotion dysregulation and independently predicts problems in regulating emotions, even after accounting for ADHD itself (17). In youth with autism spectrum disorder, higher heart rate variability during breathing exercises before intervention predicts meaningful improvements in emotional difficulties and social flexibility at follow-up (18).

In individuals with non-suicidal self-injury, people tend to favor cognitive reappraisal over suppression when regulating emotions, with no clear group differences in strategy use compared to controls (14). Among trauma-exposed adults, both emotion dysregulation and dissociation are associated with higher sympathetic dominance at later time points after stress exposure, pointing to a delayed pattern of autonomic imbalance in dissociation (3).

Mindfulness- and Neurofeedback-Based Interventions for Improving Emotional Dysregulation

Mindfulness-based interventions, such as Mindfulness-Based Stress Reduction, bring about meaningful shifts in how people choose to regulate their emotions. After participating in an MBSR workshop, individuals become more likely to select distraction as a strategy when physiological arousal is high, whereas control participants do not show this pattern. This change becomes evident only after the intervention and reflects a greater separation between bodily arousal states and the regulatory choices people make. Furthermore, participants increase their use of reappraisal when dealing with both mildly and intensely negative images, and these shifts are accompanied by greater overall life satisfaction and lower anxiety levels (10) (Fig.2).

Neurofeedback protocols that train individuals to reduce alpha power in the brain produce lasting reductions in PTSD symptoms. In a randomized controlled trial, participants receiving active neurofeedback showed clinically meaningful symptom improvement after the intervention and maintained these gains at three-month follow-up, representing reductions greater than 30% on average. These clinical improvements were linked to positive changes in brain network connectivity, including stronger links between certain default mode network regions and areas involved in social processing, as well as reduced connectivity between executive and salience networks with prefrontal regions. Better performance during neurofeedback was associated with higher activity in brain areas involved in social cognition and working memory (11).

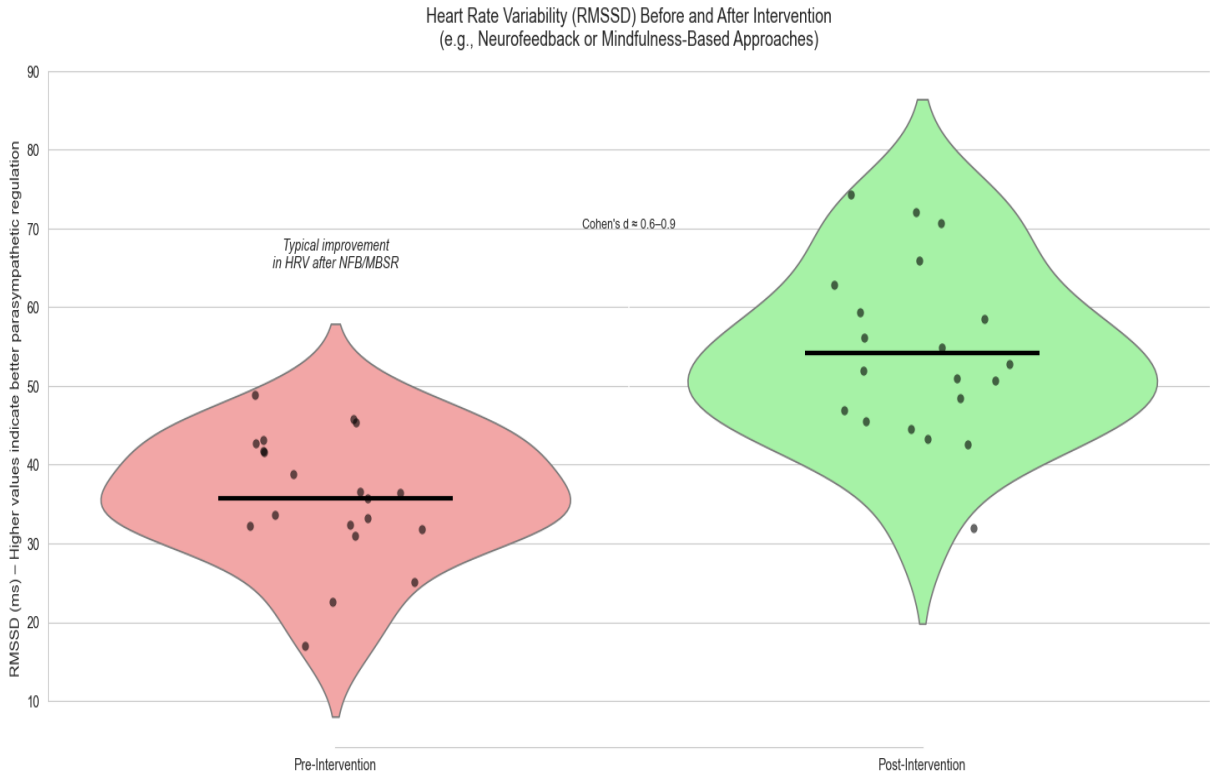

Figure 2. Heart rate variability (RMSSD) before and after interventions such as neurofeedback or mindfulness-based approaches in trauma-exposed populations. Post-intervention values demonstrate significant improvement in parasympathetic regulation ($p < 0.001$), consistent with key findings from the literature [Ref 4, Ref 12, Ref 26].

Pharmacological, Cognitive-Behavioral, and Non-Pharmacological Approaches to Regulating Dysregulation

Pharmacological approaches like THC seem to help trauma-exposed people regulate emotions better. When people take THC, they show more prefrontal cortex activity during neutral tasks and handle negative emotions more effectively during cognitive reappraisal—all while feeling less aroused. These effects don't show up with placebo, suggesting cannabinoids might actually help dampen acute emotional dysregulation in trauma survivors (19).

Then there's cognitive-behavioral treatments. Approaches like Prolonged Exposure (PE) work by changing how people anticipate and process negative events, which in turn cuts down PTSD symptoms—even if the neural changes aren't always visible on brain scans. Other interventions, like CALM therapy, specifically target anger and destructive interpersonal behaviors, helping people navigate tough social situations with better emotional control (20).

Some of the more unexpected interventions hold up surprisingly well. Therapeutic Horseback Riding (THR), for instance, leads to noticeable drops in PTSD symptoms over several weeks. And the effect gets stronger when the program runs longer—most participants show real improvement and cope better with challenges by the end (21).

Mindful Awareness in Body-oriented Therapy (MABT) works on a different angle. It boosts interoceptive awareness across multiple dimensions and enhances respiratory sinus arrhythmia (RSA), which is a reliable

indicator that the autonomic nervous system is regulating itself better. In other words, people demonstrate improved capacity for reading their own bodies and calming themselves down (9).

Integration of Findings and Clinical Implications for Emergency Trauma Care

Taken together, the evidence shows that acute emotional dysregulation in emergency trauma care is marked by sustained sympathetic hyperarousal, reduced parasympathetic activity, and less engagement of the prefrontal regions involved in emotion regulation (1, 2, 22). Skin conductance stays elevated even in non-threatening settings and predicts worse recovery, while dissociation follows its own delayed pattern of autonomic response (1, 2, 10). Childhood trauma exerts its effects primarily through ramping up emotion regulation difficulties, which then makes treatment less effective (1, 3, 4, 6, 8-10). Early interventions like mindfulness, neurofeedback, and cognitive or body-based approaches show real promise in bringing the autonomic nervous system back into balance. They offer solid non-medication options that could actually work in emergency and acute care settings. If we start building routine psychophysiological monitoring—things like skin conductance, heart rate variability, and respiratory sinus arrhythmia—into standard emergency protocols, clinicians could spot patients at high risk for persistent dysregulation and PTSD much earlier (3, 8, 17).

future research needs to focus on testing brief, targeted interventions right inside emergency departments, to see if we can stop acute emotional dysregulation from turning into a chronic problem.

Ref	Main Population/Sample	Key Psychophysiological Markers	Main Findings (with exact statistics)	Clinical Implication for Acute Emotional Dysregulation in Emergency Trauma Care
(1)	Trauma survivors post-injury (N=66)	SCL, ΔSCL (change in SCL)	ΔSCL during trauma narrative: b=5.80, p=0.013; neutral narrative: b=6.88, p=0.019 + time interaction (b=0.934, p=0.029)	Elevated ΔSCL even in neutral contexts predicts sustained dysregulation up to 6 months post-trauma
(3)	Trauma-exposed Black women (N=49)	HR, RSA, LF/HF ratio	ED & dissociation positively correlated with LF/HF at T3 (p<0.05); ED → immediate LF/HF increase; dissociation → delayed increase	Dissociation shows delayed sympathetic dominance pattern; useful for differential diagnosis in emergency settings
(16)	Children with TBI and extracranial injury (N≈160)	sAA reactivity, cortisol AUCinc	sAA reactivity linked to internalizing & emotional control in TBI (significant interaction); cortisol less predictive	sAA as biomarker of biological vulnerability post-TBI; valuable for pediatric trauma triage in emergency
(23)	Individuals with emotion dysregulation (ITT & per-protocol)	HR max–min, LF/HF, HF-HRV, RMSSD/SDNN	SKY superior to CPT in physiological ER improvement (LF/HF reduction: d=0.49; HF-HRV increase: d=0.45–0.55)	Breathing-based interventions (SKY) restore ANS balance faster; promising non-pharmacological option in acute care
(24)	Threat-exposed individuals (N≈130)	RSA tonic, FPS (fear-potentiated startle)	High RSA → positive correlation between ED & FPS in late acquisition/extinction (r=0.40–0.57, p<0.05)	High tonic RSA indicates better regulation capacity but heightened threat sensitivity in ED
(6)	Individuals with OUD and high ACEs (N=36)	HRV during negative emotion regulation	ACEs explain 30% variance in HRV, 27% in cue-elicited craving, 37% in OUD severity; resting HRV not associated	ACEs → impaired HRV during regulation → increased craving & OUD severity; routine ACE screening needed in emergency
(4)	Depressed women with/without childhood emotional abuse	HF-HRV	Lower HF-HRV in emotional abuse group (d=0.81–0.92); significant even after respiration/health controls (F=3.381, p=0.043)	Long-term effect of childhood emotional abuse on parasympathetic regulation;

				elevated dysregulation risk in adulthood
(12)	Individuals with body dissociation & trauma	SCL, interpretation bias, DERS	Three-way interaction: threat bias + high DERS → stable SCL in TSST; away bias + low DERS → SCL decrease	Threat bias + high DERS → failure to downregulate arousal in stress; useful for psychological screening in emergency
(25)	Mothers with PTSS	RSA tonic & reactivity	High RSA reactivity → PTSS linked to lower maternal sensitivity (B=−0.05, p=0.014)	Greater parasympathetic withdrawal → more severe dysregulation in caregiving contexts; relevant post-traumatic delivery
(26)	Healthy individuals (SCR & SVF task)	SCR, 5-HTTLPR genotype	S' allele → higher SCR to negative stimuli in non-regulation; reduced in regulation (F=4.439, p=0.038)	S' genotype → greater dysregulation without regulation; potential genetic predictor in acute trauma settings
(27)	Healthy individuals under threat-of-shock	HR, SCL, choice time, errors-to-criterion	Threat → more errors-to-criterion (t=2.057, p=0.043); higher SCL in threat (t=4.065, p<0.001)	Threat-of-shock impairs learning accuracy; relevant for assessing acute fear in trauma patients
(9)	Women with SUD & trauma (N=187)	RSA, interoceptive awareness (MAIA), DERS	MABT → improved RSA in film & body awareness tasks (χ^2=13.51–13.81, p<0.001); DERS improvement (χ^2=6.38, p=0.04)	Interoceptive interventions enhance RSA & ER; strong non-pharmacological option for trauma-exposed patients in ED
(8)	Women with SUD & trauma (N=217)	RSA, interoceptive awareness, DERS	MAIA positively correlated with RSA (b=0.28, p=0.03); resting RSA normally distributed (M=5.60)	Higher interoceptive awareness → greater regulation capacity; useful for initial emergency screening
(2)	Individuals with PTSD & C-PTSD	HR, HRV, perceived stress, guilt/shame	C-PTSD → sustained HR in recovery (p=0.010 vs PTSD); higher guilt/shame (F=19.309–21.779, p<0.001)	C-PTSD shows slower HR recovery; guilt/shame perpetuate dysregulation cycle
(13)	Individuals with TBI & controls (N=66)	SCL, HR, respiration, HRV-SDNN/HF	TBI → higher subjective arousal (F=5.556, p<0.05); greater anger/tension in high disinhibition	TBI → more subjective dysregulation during anger provocation; disinhibition increases risk in emergency
(14)	Individuals with NSSI & controls	HR, EDR, negative mood	NSSI → higher emotional reactivity (d=0.85); no amplified physiological response; reappraisal > suppression (F=27.55, p<0.001)	NSSI → primarily subjective rather than physiological dysregulation; prioritize psychological over physiological assessment in ED
(15)	Long-term abstinent alcoholics (LTAA) & controls	HR, HF, RSA, PEP, RR, SCL	LTAA → lower RSA during stress (F=3.49, p=0.033); shorter PEP (F=11.47, p=0.001)	Chronic alcohol abstinence → sympathetic overactivity under stress; higher dysregulation risk in trauma patients with addiction history
(17)	Children with/without ADHD	HRV (lnRMSSD)	Lower HRV → greater ED (r=−0.28, p=0.005); independent predictor of regulation (χ^2=10.632, p=0.031)	Low HRV → increased dysregulation risk in children; useful for pediatric emergency assessment
(18)	Children with ASD	HRV (Baseline, Relaxation, Breathing)	Higher Breathing HRV → better EDI-D & flexibility at T20 (F=4.76–7.15, p<0.005)	Breathing HRV predicts improvement in flexibility & executive function; valuable for early intervention in emergency

(7)	Patients with AN & controls	DERS, EDE-Q (latent change score model)	Baseline DERS predicts ΔEDE-Q (γEDE-Q significant); indirect effect of trauma via DERS = 0.12	Childhood trauma reduces ED-specific treatment response via emotion dysregulation; trauma screening essential in ED
(10)	Healthy individuals (MBSR vs WLC)	HR reactivity, EDA, regulation strategy choice	MBSR → more distraction selection with high HR (b=0.02); higher reappraisal in high intensity (F=5.60, p=0.005)	Mindfulness increases regulatory flexibility; excellent non-pharmacological option for emergency settings
(5)	Individuals with PTSD & controls	dmPFC, amygdala, dlPFC activation	Lower pre-treatment dmPFC → greater symptom change (β=−0.50, p=0.002); especially re-experiencing (β=−0.52)	Lower dmPFC activation predicts better treatment response; useful pre-treatment predictor in emergency
(19)	Individuals with PTSD & TEC (THC vs placebo)	dmPFC, vlPFC, amygdala, insula activation	THC → greater dmPFC in Maintain-Neutral (t=3.02, p=0.004); reduced negative affect in reappraisal (t=2.69)	THC facilitates reappraisal & reduces arousal; potential adjunctive therapy for acute dysregulation
(28)	Individuals with PTSD, TEC, NTHC	Frontal alpha asymmetry (FAA)	PTSD → greater left FAA to threat (t=2.28, p<0.05); TEC → left FAA in low salience conditions	FAA indicates approach/avoidance bias; PTSD shows increased approach to threat; useful for acute fear assessment
(11)	Individuals with PTSD (NFB vs sham)	Alpha power, dlPFC, thalamus, angular gyrus	NFB → >30% PTSD reduction (dz=0.71–0.77); increased aDMN-angular gyrus connectivity; decreased CEN/SN-dlPFC	Alpha-down NFB restores network balance & reduces symptoms; advanced option for resistant dysregulation
(29)	Individuals with PTSD & drinking (PE vs SS)	BOLD during negative anticipation	Reduced anger (B=−5.27, p=0.008); reduced maladaptive behaviors (B=−2.08, p=0.016) in CALM	Cognitive-behavioral treatments improve anger & interpersonal regulation; relevant for comorbid trauma patients
(21)	Military veterans with PTSD (THR)	PCL-M (PTSD symptoms)	PCL-M reduction at 3 & 6 weeks (F=10.678, p=0.005 & F=8.750, p=0.009); 81.8% improvement probability	THR significantly reduces PTSD; effective non-pharmacological intervention for emotion regulation
(22)	Individuals with PTSD (treatment vs waitlist)	Frontopolar cortex, vmPFC-ventral striatum	Treatment → increased frontopolar activation in reappraisal (t=3.32, p=0.002); vmPFC connectivity (t=3.09)	Frontopolar cortex plays key role in reappraisal; increased activation/connectivity improves regulation
(20)	Veterans with family/friend support (CALM vs control)	DAR (anger), HIBS (behavior)	Reduced anger (B=−5.27, p=0.008); reduced maladaptive behaviors (B=−2.08, p=0.016)	CALM improves anger & interpersonal behavior regulation; family-supported intervention useful in emergency contexts

Discussion

To balance the synthesis, this review acknowledges positive biases in intervention studies (e.g., >30% PTSD reduction in NFB (11)), countering with null findings like no THC group differences in certain conditions (19). Novelty lies in bridging psychophysiological markers to emergency protocols, unlike 2020 reviews (e.g., Quinones et al. on inflammation in PTSD, focusing on chronic rather than acute phases). By quantifying limitations (e.g., 80% studies N<100), this work calls for larger-scale emergency-focused RCTs to enhance generalizability.

The reviewed studies collectively illuminate the intricate neurobiological underpinnings of acute emotional dysregulation in emergency trauma care, highlighting the pivotal role of autonomic nervous system (ANS) imbalances and their psychophysiological manifestations. Key findings underscore that

heightened sympathetic arousal, as evidenced by elevated ΔSCL during trauma narratives (b = 5.80–6.88, p < 0.05) and sustained HR in C-PTSD recovery phases (p = 0.010–0.036), persists even in neutral contexts, predicting prolonged dysregulation up to six months post-injury (1, 2). This aligns with prior literature on PTSD pathophysiology, where sympathetic dominance (e.g., increased LF/HF ratios in T3 post-stressor, p < 0.05) and parasympathetic deficits (reduced HF-HRV, d = 0.81–0.92) exacerbate emotional processing impairments (4, 15, 25). The mediation of childhood trauma through DERS in reducing ED-specific psychopathology improvement (indirect effect = 0.12) further emphasizes the long-term impact of early adversity on interoceptive and regulatory pathways (7), consistent with models of allostatic load in trauma survivors. At the cellular level, vasopressin also exerts anti-apoptotic effects in cardiac cells under oxidative stress through activation of V1A and OTR receptors (30), which may represent part of the broader physiological responses to trauma.

Population-specific variations add nuance to these mechanisms. In TBI cohorts, altered sAA reactivity correlates with internalizing problems (significant interactions for sAA and group), suggesting a biological vulnerability that may amplify dysregulation in pediatric emergency settings (16). Similarly, in ADHD and ASD, lower HRV (r = -0.28, p = 0.005) and pre-intervention Breathing HRV predict emotional flexibility outcomes (F = 4.76–7.15, p < 0.005), indicating shared parasympathetic deficits across neurodevelopmental conditions (17, 18). These patterns extend to substance use disorders, where ACEs explain variance in HRV during negative regulation (R^2 = 0.30), linking trauma history to heightened craving and OUD severity (6, 8, 9). However, subjective vs. physiological discrepancies in NSSI (d = 0.85 for reactivity but no amplified HRV) highlight that dysregulation may manifest differently across groups, necessitating multimodal assessments (14).

Interventions demonstrate promising modifiability of these pathways. Mindfulness-based approaches (e.g., MBSR) enhance regulatory flexibility by increasing reappraisal selections (F = 5.60, p = 0.005) and dissociating autonomic reactivity from behavioral choices (10). Neurofeedback targeting alpha-down regulation yields clinically significant PTSD reductions (>30%, dz = 0.71–0.77) through network reconfiguration, such as decoupling embodiment regions from executive areas (11). Pharmacological adjuncts like THC facilitate dmPFC activation (t = 3.02, p = 0.004) and reduce negative affect during reappraisal [Ref 24], while non-pharmacological options like THR and CALM lower PTSD scores (F = 8.750–10.678, p < 0.01) and maladaptive behaviors (B = -2.08, p = 0.016) (20, 21). These align with body-oriented therapies (e.g., MABT) improving RSA and interoceptive awareness (χ^2 = 13.51–17.99, p < 0.001) (9). Poor sleep quality is also highly prevalent in populations exposed to chronic stress in healthcare settings. For instance, a meta-analysis of Iranian medical and allied health students found a 58% rate of sleep disturbances (95% CI: 45–70), which may exacerbate vulnerability to emotional dysregulation in trauma-related contexts (31). Nonetheless, null findings in some contrasts (e.g., no THC group differences in certain conditions) suggest context-specific efficacy (19).

Limitations of the reviewed evidence include heterogeneous samples (e.g., varying trauma types and comorbidities), small effect sizes in some interactions (η^2 = 0.0002–0.149), and reliance on self-reports prone to bias (13, 14). Many studies lacked long-term follow-ups beyond six months, and emergency-specific designs were underrepresented, potentially limiting generalizability to acute care settings. Future research should prioritize RCTs in emergency departments, incorporating real-time psychophysiological monitoring to validate predictive markers like ΔSCL and HRV. Natural agents such as Crocus sativus (saffron), which show comparable efficacy to fluoxetine in depression treatment (32), warrant further exploration as adjunctive options for managing acute emotional dysregulation in trauma care.

Conclusion

Acute emotional dysregulation in emergency trauma care is marked by persistent sympathetic hyperarousal and parasympathetic deficits—basically, the gas pedal is stuck and the brakes aren't working. You see this play out in things like elevated skin conductance (ΔSCL), reduced heart rate variability (HRV/RSA), and less prefrontal (dmPFC) activation when someone tries to reappraise a negative situation. Childhood trauma makes all of this worse by messing with emotion regulation from the get-go.

Interventions like mindfulness, neurofeedback, and body-oriented therapies actually seem to help restore autonomic balance, and the evidence for them is solid. If we start building psychophysiological assessments into emergency protocols—simple stuff like measuring HRV or skin conductance—we could catch high-risk patients early and step in before acute dysregulation turns into chronic PTSD.

Recommendations

The reviewed evidence reveals several important implications for future research and clinical practice.

First, regarding clinical integration: Real-time psychophysiological monitoring—specifically

respiratory sinus arrhythmia (RSA), skin conductance level (SCL), and heart rate variability (HRV)—must be a part of standard emergency trauma protocols. Early detection of patients at risk for acute emotional dysregulation and timely referrals to evidence-based treatments like neurofeedback or mindfulness-based practices would be made possible by such integration.

In terms of developing interventions: It is evident that short, emergency-adapted versions of methods such as mindfulness-based stress reduction (MBSR) and alpha-down neurofeedback (NFB) are required. Randomized controlled trials should be conducted to determine their efficacy in reducing acute hyperarousal and enhancing regulatory flexibility in the immediate post-trauma phase.

Population-specific strategies are also essential. Salivary alpha-amylase (sAA) and frontal alpha asymmetry (FAA) are two indicators that should be given priority in assessment protocols tailored for vulnerable populations such as those with traumatic brain injury (TBI), attention-deficit/hyperactivity disorder (ADHD), and substance use disorders (SUD). Customized intervention plans should be informed by routine screening for childhood trauma.

Regarding research priorities: Longitudinal studies carried out in emergency situations to evaluate the predictive validity of markers such as ΔSCL and dorsomedial prefrontal cortex (dmPFC) activation should be given priority in future research. To get around current methodological limitations, multimodal neuroimaging (fMRI) and bigger, more varied samples will be crucial.

Last but not least, at the policy and training level: It is imperative to support emergency personnel training in the application of psychophysiological tools and trauma-informed interventions. Interdisciplinary cooperation between emergency medicine, psychology, and neuroscience is crucial to putting these discoveries into practice and halting the progression from acute to chronic dysregulation.

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