

Types of psychological trauma by Ego disintegration mechanism and their neurobiological profiles: a causal model

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Abstract

This article presents a typology of psychological trauma developed within the framework of Integrative Causal Psychotherapy** and based on the primary mechanism of Ego disintegration. Four etiopathogenetic trauma types are distinguished: vital, invasive, egotomic, and constitutive trauma***. For each type, the following are consistently described: (a) definition and essence of the trauma, (b) mechanism of Ego damage, (c) clinical phenotype and its neurobiological correlates, (d) clinical implications, and (e) illustrative clinical case. The proposed typology forms a causal algorithm for choosing a therapeutic strategy according to the leading mechanism of traumatization. The compatibility and integrability of the presented typology with ICD-11 and DSM-5 are shown, allowing the translation of a nosological diagnosis into an etiotropic and personalized treatment plan. The article presents a theoretical and clinical model offering a causally oriented framework for conceptualizing psychological trauma. The presented material is intended for mental health professionals and aims to initiate discussion and empirical validation of the proposed typology.

Keywords: psychological trauma, Ego disintegration, causal typology of psychological traumas, vital trauma, invasive trauma, egotomic trauma, constitutive trauma, PTSD, Integrative Causal Psychotherapy.

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Introduction

The problem of effective therapy for psychological trauma remains one of the central challenges in modern clinical psychology and psychotherapy. Despite a large number of methods, their effectiveness varies significantly, especially when working with chronic, complex, and hidden forms of traumatization: approximately two-thirds of patients may continue to experience clinically significant symptoms even after the application of first-choice protocols [1, 2]. This underscores the need for causally oriented models that allow not only the reduction of symptoms but also the diagnosis and impact on the very mechanism of their occurrence.

Existing approaches mainly rely on phenomenological or syndromal classifications

(PTSD, CPTSD, acute stress disorder, grief reaction, dissociative disorders) or on reductive dichotomies (PTSD and CPTSD, single and recurring trauma, etc.) [3]. However, ICD/DSM classifications remain syndromal and do not reflect neurobiological and structural-dynamic differences between forms of traumatization [4, 5]. Even the distinction between “simple” and “complex” PTSD, clinically useful, does not explain the mechanism of the problem’s emergence and does not allow the selection of an etiotropic therapeutic target. Similarly, classifications based on the type of traumatic event (sexual, military, childhood trauma, etc.) ignore what constitutes the decisive mediator in the etiology of post-

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** Integrative Causal Psychotherapy (ICP; Serbian: *Integralna kauzalna psihoterapija*, IKP; Russian: *Integral'naya kauzal'naya psikhoterapiya*, IKP) is a psychodynamically oriented psychotherapeutic approach developed by Yu. N. Tor within a Serbian-Russian psychotherapy school. ICP's therapeutic protocols focus on identifying and eliminating the causes of mental disorders and integrate neuromodeling methods, elements of Katathymic-Imaginative Psychotherapy, and psychoanalytic techniques.

*** Hereinafter, the word “trauma” is used to mean “psychological trauma”.

traumatic disorders: the individual mode of mental functioning and the specific mechanism of Ego impairment.

Despite advances in the study of amygdala functions, self-referential networks (default mode network (DMN), salience network (SN), central executive network (CEN)), dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, neuroinflammation, and epigenetics, their integration into clinical models remains fragmented. Neurobiological research describes final phenotypes but does not link them to disturbances in Ego structures [6, 7]. Consequently, a gap remains between neuroscience, describing dysfunctions of individual systems, and clinical practice, operating with concepts such as symptom and syndrome.

Traditional approaches explained trauma through threat to life, stress response, memory processing disorders, or attachment. However, no existing classification answers the key question of etiologic trauma therapy: which specific basic mechanism of Ego integrity and self-regulation was disrupted? Without understanding this mechanism, the therapist is forced to select interventions empirically, and the strategy either becomes universal (“one protocol for all”) or excessively individualized, not taking systemic guidelines into account.

Clinical experience indicates that psychological trauma is not homogeneous: it disrupts different Ego functions and forms various clinical phenotypes. Therefore, without a typology of psychological traumas based on the mechanism of Ego damage, it is impossible to accurately determine therapeutic targets, predict therapeutic response, and build a causally oriented treatment strategy. The proposed causal model attempts to fill this gap by revealing, for each trauma type, a specific mechanism of Ego damage and its unique neurobiological picture. The integration of neurobiological data provides not only the possibility of a more accurate understanding of the clinical phenotype but also a rationale for the use of pharmacotherapy in cases of its pathogenetic necessity.

Thus, the purpose of this article is to present and substantiate a new clinical hypothesis — the causal typology of psychological traumas based on the mechanism of Ego disintegration. This article does not aim to provide exhaustive empirical proof of the model, but propose it as a systematizing framework and practical tool for clinical reasoning, which requires further operationalization and rigorous empirical validation. The subsequent presentation should be regarded as a detailed substantiation of this hypothesis, supported by clinical illustrations.

Methods

This study employs theoretical modeling supported by clinical observations. The development of the typology proceeded in three stages:

1. Theoretical synthesis of key concepts from psychoanalysis (structural theory and attachment theory), contemporary psychotraumatology, and the neurobiology of affect regulation.
2. Inductive generalization of patterns observed by the authors in many years of clinical practice working with cases of psychological trauma.
3. For the purpose of demonstrating the internal logic of each type, four key cases were deliberately selected for the article, as they most clearly and representatively illustrate the proposed typology. These cases are drawn from the authors’ extensive clinical experience but do not constitute a systematic sample. They are presented for illustrative purposes to demonstrate the internal coherence and practical applicability of the model.

In the presented clinical cases, outcome evaluation is based on the criterion of stable clinical remission, defined as the absence of the core maladaptive symptoms that prompted treatment and the restoration of premorbid levels of functioning in key life domains (social, occupational, and personal). Given the retrospective nature of the analysis and its focus on demonstrating pathogenetic logic, standardized psychodiagnostic scales were not used in these cases, which constitutes a limitation for the generalizability of the findings but does not diminish their value for illustrating the proposed model.

Causal model of psychological trauma by Ego disintegration mechanism

The typology of psychological traumas by the mechanism of Ego disintegration, developed in Integrative Causal Psychotherapy, includes four types: vital, invasive, egotomic, and constitutive traumas. The model relies on the structural theory of the Ego [8], the personality theory of Integrative Causal Psychotherapy [9], modern data on trauma neurobiology [10–12], polyvagal theory [13], as well as the theory of allostatic load and epigenetic changes [14, 15].

The novelty of the proposed typology lies in its systemic integration: it consistently links the primary mechanism of Ego damage, the clinical phenotype, and the specific neurobiological profile for each type of trauma. Unlike classifications based on the external event or syndrome, this model makes it possible to:

- determine hypothetically the leading mechanism of damage (“point of failure”) at the stage of diagnostic conceptualization;

To learn more about Integrative Causal Psychotherapy, follow the link: <https://icpmodality.org>

- build therapy based on the causes of the disorder, not just its symptoms;
- predict therapeutic dynamics and avoid errors arising from incorrect understanding of the trauma type;
- integrate methods from various psychotherapeutic schools around restoring a specific damaged Ego function;
- propose hypothetically justified indications for pharmacotherapy in accordance with the presumed pathogenesis of a specific trauma type.

The proposed typology provides the clinician with a map that allows them to identify the damaged part of the personality architecture behind the symptoms and determine how it can be restored.

Type 1. Vital trauma

Vital trauma (from Latin *vita* — “life”) arises from a collision with a force that, perceived as life-threatening and overwhelming, destroys the basic sense of safety and predictability of the world. This is a type of psychological trauma in which the core mechanism involves a severe overload of the psychological safety system as a result of direct confrontation with a real or perceived threat to physical existence or bodily integrity. The emotional core of this trauma is intense fear. The mechanism leads to a global collapse of the psychological safety system, resulting in a pervasive reorganization of the personality around the traumatic experience. This represents a general failure of the basic Ego structures responsible for regulation, integration, and maintenance of personality integrity. It disrupts the hierarchical organization of the psyche and forms a new, pathological architecture where the dominant organizing principle becomes constant readiness for threat [16].

Vital trauma typically arises in the case of repeated life-threatening situations or a single high-intensity life-threatening event (torture, participation in combat, terrorist attack, armed assault, etc.). It leads to complex and persistent changes in the functioning of the central nervous system. Its neurobiological profile represents not merely a set of isolated dysfunctions, but rather a self-sustaining pathological system. In this system, chronic hyperactivation of threat detection nodes, exacerbated by insufficient descending control and supported by fundamental pathophysiological processes, forms a stable state of maladaptation underlying the following clinical symptomatology [17]:

- *affective sphere*: background fear, generalized anxiety, hyperarousal, self-aggression, suppression of feelings up to alexithymia;
- *cognitive sphere*: intrusive memories and flashbacks, rumination, hypervigilance to potential threats;

- *somatic level*: high startle response, chronic muscle tension, increased fatigue, high sympathetic tone, possible sleep disturbances (nightmares, insomnia);
- *behavioral manifestations*: avoidance of triggers and situations reminiscent of the trauma, aggressive or impulsive reactions during activation.

The neurobiological picture of vital trauma is characterized by the following features.

1. *Chronic hyperactivation of threat detection and response systems* leads to a state of constant hypervigilance and readiness for a stress response. Excessive activation of the amygdala is associated with reactions to neutral or conditionally safe stimuli, interpreting them as potentially dangerous. High HPA axis activity, manifested in persistently elevated basal cortisol levels or its atypical diurnal rhythm, maintains a state of metabolic and immune readiness for stress. Hyperactivity of the anterior insula is manifested in somatosensory symptoms, such as increased pain sensitivity, nausea, and general physical discomfort in response to psychological stress [18].
2. *Dysfunction of regulatory and modulating systems* causes insufficient descending inhibitory control from higher cortical structures. Reduced activity of the prefrontal cortex, particularly in dorsolateral and ventromedial areas, leads to deficits in executive functions (planning, decision-making, working memory), impaired conscious cognitive control over emotional reactions, and weakened ability to reflect on them. Hippocampal hypofunction under the influence of excess glucocorticoids impairs the ability to differentiate past threatening situations from current safe reality and contributes to declarative memory deficits.
3. *Dysregulation of neurochemical systems* regulating mood, motivation, and general psychophysiological state is described by the following changes. Reduced dopaminergic activity in the mesolimbic pathway leads to anhedonia, apathy, and reduced motivation for goal-directed behavior. Increased activation of raphe nuclei and locus coeruleus leads to the development of depression, anxiety, and sleep/appetite regulation disorders. A shift in autonomic balance towards sympathetic dominance with simultaneous reduction in vagal control efficiency causes difficulties with relaxation and impaired restorative functions.

4. *Chronic pathophysiological processes* maintain the above functional changes and include the following mechanisms:

- General pro-inflammatory status of the body: elevated levels of pro-inflammatory cytokines (e.g., IL-1 β , IL-6, TNF- α) negatively impact neuroplasticity, exacerbate HPA axis dysfunction, and contribute to “brain fog” and asthenia;
- Oxidative stress and mitochondrial dysfunction: chronic hyperactivation of stress axes and inflammation leads to excessive generation of reactive oxygen species (ROS), damage to neuronal lipids, proteins, and DNA, reduced cellular energy supply, and accelerated apoptotic death of neurons and glia;
- Reduced neuroplasticity: decreased expression of brain-derived neurotrophic factor (BDNF) leads to impaired learning and adaptation ability, as well as hinders recovery processes and the formation of new, more adaptive neural networks;
- Epigenetic modifications: persistent changes in gene expression through DNA methylation and histone modifications can affect genes encoding glucocorticoid receptors, neurotrophins, and components of neurotransmitter systems, cementing stress

vulnerability and potentially transmitting it intergenerationally.

This diagram illustrates that the core pathological circuit is formed by the threat system and the dysfunction of regulatory structures. A hyperactive amygdala and HPA axis suppress the activity of the hippocampus and prefrontal cortex. In turn, the weakened prefrontal cortex and hippocampus cannot provide top-down inhibitory control over the amygdala and HPA axis. This vicious cycle serves as the primary driver of the pathological state [19, 20]. Chronic processes act as a reinforcing mechanism triggered by constant stress. They sustain and exacerbate the dysfunction of both the threat system and the regulatory systems [21]. Sympathetic dominance and autonomic nervous system rigidity are presented as both a consequence and an amplifier of the imbalance between the core systems. The clinical manifestations represent the final outcome of the interplay among all these disrupted neurobiological systems [22, 23]. The model presented in Figure 1 helps to understand why vital trauma is such a persistent state: the system closes in on itself, and the mentioned chronic processes prevent escape from this dysfunctional cycle. Therefore, it requires comprehensive psychotherapy at different levels of the personality, which includes the following targets aimed at the stepwise reorganization of the

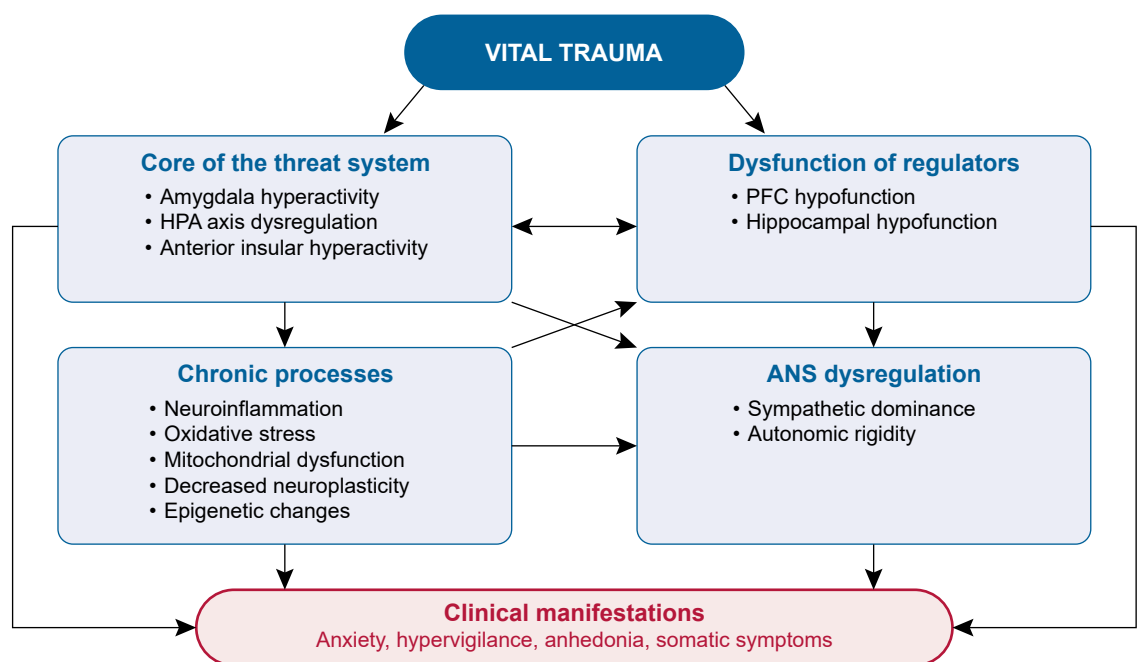


Figure 1. Pathogenetic model of vital trauma

maladaptive system of “traumatic survival” and the creation of a new, safe psychic architecture:

- restoration of basic trust through the therapeutic relationship;
- reprocessing and integration of traumatic experience — deactivation of pathological engrams*;
- formation of a new identity;
- creation of adaptive coping strategies.

In working with vital trauma, Katathym-Imaginative Psychotherapy (Symbol drama)** has shown clinical utility. We recommend it as the main method for treating this type of trauma in combination with neuromodeling techniques*** of Integrative Causal Psychotherapy. Clinical experience with this approach indicates that vital

trauma, in the absence of comorbid disorders, demonstrates stable positive dynamics, which, within our model, creates the prerequisites for achieving remission within medium-term therapy.

For the synergistic enhancement of psychotherapy for vital psychotrauma, pharmacotherapy may be recommended, specifically the use of selective serotonin reuptake inhibitors (SSRIs). Their application aims to create neurochemical stability, which contributes to reducing the overall reactivity of the amygdala and may indirectly influence the normalization of hypothalamic-pituitary-adrenal (HPA) axis dysregulation. This somatotrophic effect can help establish a more stable neurobiological foundation, thereby increasing the effectiveness of psychotherapeutic work.

Clinical case of vital trauma in childhood

Anamnesis

The patient, a 40-year-old female, experienced severe mistreatment over several years during her preschool age by her governess: she was locked in a dark room, subjected to painful pinches, episodically choked, regularly punished for imaginary transgressions, and kept in a state of constant fear and tension.

Diagnostic conceptualization

The clinical picture was identified as vital trauma. The key diagnostic markers were:

- introjected sadistic Superego: systematic self-restriction and self-punishment, active blocking of access to positive experiences, joy, and satisfaction;
- high degree of self-aggression and deep distortions in self-esteem and self-image;
- high-intensity trigger-conditioned panic states upon encountering potentially pleasant opportunities (rest, travel);
- activation of pronounced psychosomatic reactions upon attempts to overcome fear, serving the function of secondary defense against realizing desired goals.

Therapeutic process

Treatment using the Integrative Causal Psychotherapy approach with elements of Symbol drama lasted two years, consisting of once-weekly 50-minute sessions. It was aimed at restoring the basic system of psychological safety, processing affects related to traumatic situations, forming a stable positive self-relationship, and developing adaptive coping strategies.

Outcome and Follow-up

Sustained remission of trauma-related symptoms was observed. The patient formed a more stable and positive self-relationship and regained the ability to experience pleasure and positive emotions. Her life became more productive, and her emotional state became consistently favorable.

Conclusion

This clinical case suggests that an etiotropic strategy targeted at the identified mechanism of Ego disintegration may allow for achieving not only symptomatic improvement but also structural restoration of the impaired function, which clinically manifests as stable remission.

* An engram is a sensorimotor-affective trace of a lived experience, consolidated within the nervous system and psyche, which can be automatically reactivated and influence perception, emotions, and behavior.

** Katathym-imaginative psychotherapy is a depth-oriented psychotherapeutic approach developed by the German psychotherapist Hanscarl Leuner in the 1950s, based on the ideas of psychoanalysis and Jungian psychology. In this method, unconscious material emerges and is processed through guided work with images and symbols in a state of katathymic imagination.

*** Neuromodeling is a set of targeted therapeutic techniques developed within Integrative Causal Psychotherapy. These techniques enable the correction of pathological neural circuits associated with traumatic experiences, negative affects, repressed emotions, and dysfunctional behavioral patterns, thereby facilitating the restoration of a healthy Ego and authentic Self.

Local vital trauma

There is a subtype of vital trauma, represented by local (focal) trauma. In this subtype, the mechanism of Ego damage involves the formation of a stable but isolated pathological engram without generalization of the impairment to the entire personality structure and without a radical restructuring of the overall psychic architecture. Its distinguishing feature is the formation of a highly specific focus of hyperactivity in the threat system while overall Ego functioning remains intact.

In local vital trauma, a clearly delineated system of neurons is formed, including sensory triggers, an emotional component, and corresponding autonomic patterns. Outside the activation of this specific network, the brain and psyche function within normal limits. This trauma represents an example of a unique adaptation of the psyche to an overwhelming impact. The threat is processed and “packaged” into an isolated focus [24]. Examples of typical situations that can lead to local vital trauma include car accidents with physical injuries or severe

fright, industrial accidents, street muggings, animal attacks, witnessing a violent scene, etc.

As a result of local vital trauma, sensory-autonomic flashbacks are formed — involuntary, instantaneous, and intense reproductions of the sensory and autonomic components of the traumatic experience in response to a trigger, sometimes without full-scale conscious memory of the event. The formation of selective phobias with clearly delineated trigger reactions is also possible.

Integrative Causal Psychotherapy offers a model for working with trauma, focused on a profound process of transformation. We assume that traumatic experience is consolidated in the form of dysfunctional engrams — persistent memory traces that retain pain and fear. Our key technique, “Memory Catharsis,” is designed to initiate the process of complete affective and cognitive reprocessing of these engrams. Clinical observations and our theoretical model suggest that such reprocessing leads to their integration into personal history and the elimination of their pathogenic influence on the present.

Clinical case of hydrophobia due to local vital trauma

Anamnesis

The patient, a 45-year-old female, complained of a persistent fear of bodies of water, present throughout her subjective life experience. Symptomatology manifested during annual family trips to the sea: the possibility of entering the water was limited to ankle depth, accompanied by pronounced autonomic reactions (tachycardia, tremor, hyperventilation) and a subjective experience of uncontrollable fear. The patient had no access to an autobiographical memory explaining the genesis of the phobia.

Diagnostic conceptualization

The key diagnostic markers of local vital trauma were:

- clear trigger specificity (sight and feel of a body of water);
- intense autonomic reaction to the trigger;
- preservation of general functioning and absence of generalized anxiety outside the trigger context.

Therapeutic process

During therapy, the patient regained access to a dissociated childhood episode of a dangerous drowning incident. Subsequently, a cathartic technique aimed at processing its affective components was applied. After targeted intervention aimed at resolving the isolated traumatic memory, sustained clinical remission of phobic symptomatology was achieved, which, within our model, is interpreted as deactivation of the corresponding pathological engram.

Outcome and Follow-up

Sustained clinical remission of symptoms was noted upon completion of therapy. A behavioral marker of success was the patient’s independent purchase of a pool membership and starting swimming lessons without any trace of the former fear. Information from an external observer (the coach), who expressed disbelief regarding the 40-year history of the phobia, confirmed the clinically significant and stable changes.

Conclusion

The presented case demonstrates the high efficacy of targeted processing methods for isolated traumatic engrams. The localization of the damage, absence of structural personality changes, and preservation of general Ego regulatory functions made rapid and sustained clinical remission possible through the restoration of integration between sensory-affective and narrative memory components.

Type 2. Invasive Trauma

Invasive trauma (from Latin *invadere* — “to invade,” “to penetrate”) is a type of psychological trauma where the mechanism of damage to the Ego consists of overwhelming and uncontrolled penetration of external stimulation. This stimulation severely violates personal boundaries, exceeds the Ego’s metabolic capacities, and leads to “affective flooding.”

The essence of the trauma lies in a catastrophic failure of the function of psychological boundaries — the Ego’s fundamental ability to filter, dose, and assimilate incoming stimulation. The psychic container is flooded with contents that, being incapable of being processed and integrated, persist in the psyche as foreign and harmful. This leads to a collapse of subjective autonomy and the development of a clinical phenotype characterized by a dominance of ontological shame and disgust toward one’s own body and Self, chronic dissociation, gaps in memory related to traumatic episodes, a sense of permeable boundaries and contamination of the personality [25, 26]. Patients with this type of trauma exhibit profound disturbances in self-relationship, including self-aggression and destructive beliefs about themselves. They have difficulties distinguishing their own mental contents (thoughts, feelings, impulses) from those of others, simultaneous rigidity and permeability of psychological boundaries, fear of intimacy, and a tendency to recreate traumatic patterns in relationships. Somatoform disorders are often present in the form of conversion symptoms, psychosomatic illnesses, and hypochondriacal symptoms [27, 28].

A secondary formation in this type of trauma is suppressed aggression. Normally, aggression is energy for protecting boundaries, asserting oneself, and separation. In invasive trauma, the very function of boundary protection is broken. Aggression, having no avenue for healthy external expression, turns against the Ego itself and becomes the pathodynamic core of the trauma, causing chronic self-destruction, dissociation, and somatization.

Examples of situations leading to invasive trauma: repeated sexual, physical, or psychological abuse and other forms of massive, uncontrolled invasion of a person’s bodily and mental boundaries.

The clinical phenotype of invasive trauma may include the following disturbances:

- *Affective:* Anxiety, chronic feelings of shame as an ontological quality (“there is something wrong with me at the level of my essence”), disgust toward one’s own body, emotional instability with sharp fluctuations between numbness and affective outbursts, intolerance

of arousal (bodily and emotional) leading to automatic “shut-down” (dissociation).

- *Identity:* A sense that thoughts, feelings, or impulses are “not mine,” difficulty separating one’s own Self from the internalized image of the boundary violator, chronic vulnerability to external evaluation and control.
- *Cognitive:* Memory fragmentation — gaps in memories of traumatic episodes, distorted perception of one’s own bodily sensations, intrusive thoughts of contamination, depravity, or defectiveness, difficulties with mentalization;
- *Interpersonal disturbances:* Fear of intimacy and trust due to anticipation of invasion, control, or violence, inability to adequately regulate the degree of emotional or physical distance, tendency to recreate traumatic relationships, tendency to lose autonomy.
- *Behavioral:* Avoidance of situations requiring bodily closeness or trust; counter-phobic behavior; actions aimed at reducing the feeling of contamination; in severe cases, self-harming behavior as an attempt to restore the boundaries of one’s own body or reduce dissociative tension.

Neurobiological picture of invasive trauma

Invasive trauma overloads sensory, affective, and cognitive systems with excessive stimulation and leads to a state of neurobiological openness, where the brain loses its ability to modulate incoming information and protect the mental core.

1. *Increased overall activation of the thalamus* disrupts the function of input sensory control and leads to “sensory flooding” — significantly increasing the load on neural networks processing pain, tactile, and interoceptive signals. Additionally, excessive activation of the amygdala significantly heightens the response to any signals of penetration or threat of invasion, leading to a constant feeling of being flooded from inside and outside, an experience of boundary rupture.
2. *Significant reduction in dorsolateral prefrontal cortex activity* leads to a cessation of protective inhibition of the amygdala, causing a loss of the ability to maintain a cognitive frame and tolerate stimuli. Disintegration of executive control networks hinders making sense of, evaluating, or restraining what is happening. The volume and intensity of incoming stimulation significantly exceed the bandwidth of the Ego’s regulatory and integrative mechanisms, manifesting as a feeling of flooding, loss of control, and breach of personal boundaries.

3. *Dysfunction of the anterior cingulate cortex* reduces the ability for cognitive distancing from emotionally significant stimuli and voluntary switching of attention to other activities. Significant impairments in the Salience Network (SN) disrupt the function of the insular cortex and distort the perception of one's own bodily boundaries, while additional high activity of the amygdala within this network causes and sustains an "emotional storm" with the subjective experience of exhausting the psyche's regulatory resources and its "surrender."
4. *Activation of the periaqueductal gray*, occurring at the peak of stimulation, is accompanied by compensatory activation of endogenous opioid and cannabinoid systems, causing marked emotional numbness, a state of affective "power loss," and immobilization. The effectiveness of communication between the hippocampus and prefrontal cortex decreases, leading to context collapse and memory fragmentation. As a result, when the Ego's assimilative capacity is exceeded, a dissociative shut-down occurs, preserving the trauma as an internal invasion.
5. *Persistent changes in neural circuits* are characterized by the following processes. Hypersensitivity of the amygdala to signals of penetration and chronic hyperactivation of the insular cortex lead to painful interoception. Reduced control from the prefrontal cortex results in poor boundary restoration. Sensory hyperreactivity leads to persistent hypersensitivity to any forms of approach, resulting in a "syndrome of lost boundaries."

The pathogenetic model (Figure 2) shows how invasive trauma triggers persistent brain maladaptation through the formation of a pathological dominance of the Salience Network, which is chronically hyperactivated along with the amygdala complex and the HPA axis. Simultaneously, regulatory systems are suppressed — prefrontal cortex activity and hippocampal functionality are reduced, creating a pronounced imbalance between key neural circuits of regulation. Clinically, this manifests as a complex of hypervigilance, intrusions, emotional numbness, and somatization, reflecting a profound disturbance in self-regulation and experience integration processes. These changes are consolidated at a fundamental level through neuroinflammation, oxidative stress, and epigenetic mechanisms, forming a stable pathological state with a loss of mental boundaries [29].

Differential diagnosis from vital trauma

- Shame, guilt, and self-aggression dominate vs. fear for life, hypervigilance.
- Triggers are interpersonal relationships vs. external reminders of the traumatic event.
- Basic trust is globally impaired: fear of intimacy as a repetition of invasion vs. preserved capacity for selective trust.
- Dissociative symptoms are chronic or become personality traits vs. episodic dissociations only in response to triggers.

The described dynamics of invasive trauma explain the stability of symptoms and the necessity for long-term therapy aimed at restoring boundaries and processing accumulated suppressed aggression. Key therapeutic targets are:

- deactivation of pathological engrams — central experiences of traumatic boundary violation;
- destigmatizing prohibitions on awareness and expression of aggression and processing suppressed aggression;
- forming positive self-relationship based on unconditional love and self-acceptance;
- developing new adaptive coping strategies in situations of personal boundary violation.

For healing invasive trauma, a method for working with suppressed aggression developed in Integrative Causal Psychotherapy has proven effective [30]. According to our therapeutic model, focusing on the deactivation of boundary violation engrams and the processing of suppressed aggression create conditions for achieving stable remission within the framework of medium-term psychotherapy. Pharmacotherapy for invasive trauma has limited application and is not the first-line treatment. In cases of acute states, its prescription for symptom relief requires particular caution and is recommended only as part of a comprehensive approach, where it serves as an adjunctive means for stabilization alongside psychotherapy. This is necessary to prevent decompensation and secondary traumatization (re-victimization), the risk of which is associated with a number of psychological mechanisms. These include:

- an unconscious perception of the medication as a repetition of the invasion, which can reactivate the traumatic experience and increase anxiety;
- pathological shame, in which symptom relief may evoke feelings of guilt ("I do not deserve relief") and paradoxically intensify suffering;
- the psychological function of numbness. Emotional blunting often serves as a defense against unbearable affect. Its abrupt pharmacological removal without psychotherapeutic support can lead to an affective breakthrough and re-traumatization.

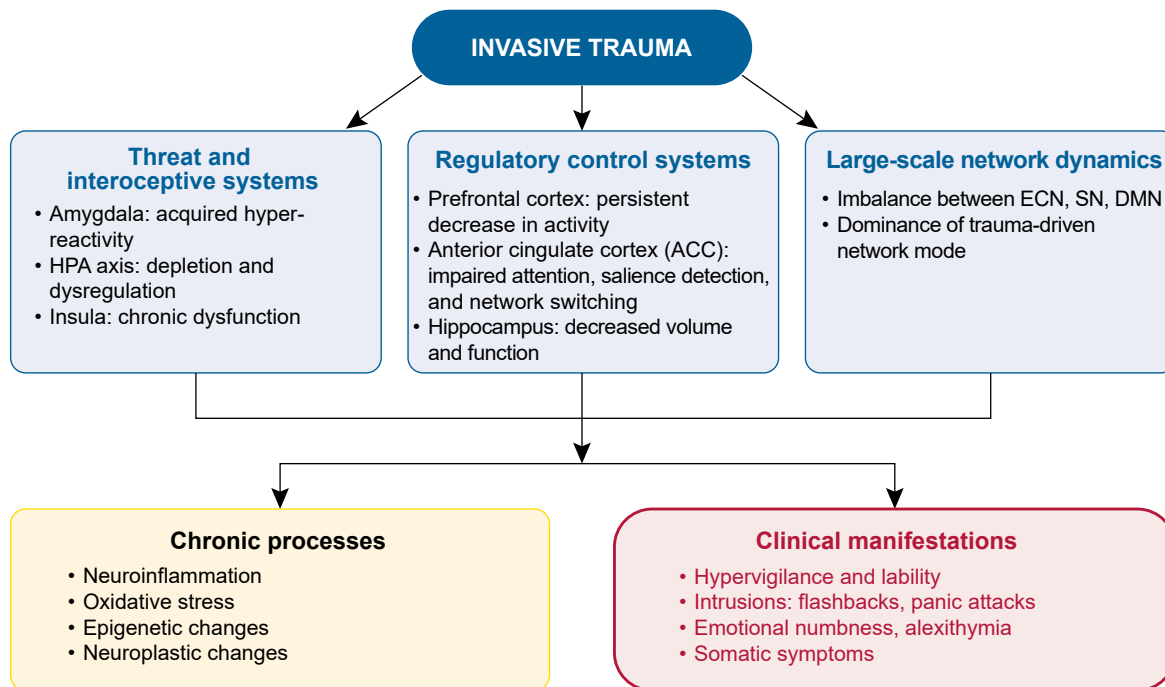


Figure 2. Pathogenetic model of invasive trauma

Clinical case of invasive trauma resulting from emotional incest

Anamnesis

The patient, a 24-year-old male, was an only child in the family and grew up in conditions of parentification*, fulfilling the role of a mother for his own mother: containing her affects, giving advice, mediating her conflicts with her spouse (his father), and being responsible for her emotional state. The situation was exacerbated by the fact that the patient's mother was aggressively biased against men, devalued them, and under the guise of "pacifist upbringing" gave her son messages "don't you dare become a man." Thus, the mother intruded into her son's psyche as an object of total dependence while simultaneously blocking the development of his gender and sexual subjectivity.

Diagnostic conceptualization

To survive in conditions of pathological symbiosis, the patient formed a conformist-martyred personality, fully compliant with maternal expectations. At the same time, male gender identity and sexual initiative became unconsciously equated with destructive aggression, inevitably followed by condemnation and punishment. The following markers indicated invasive trauma:

- prohibition on one's own subjectivity and corporeality;
- fear of criticism, especially from women;
- chronic shame and guilt in situations related to gender roles;
- repression of erotic fantasies and avoidance of penetrative forms of sexuality;
- pathological pattern of relationships with women, where intimacy was equated with servitude to an idealized maternal figure;
- psychological defenses guarding against gender-colored content and situations: using the gender-neutral "person" instead of "girl"; feminized intonation and speech in a higher pitch than his natural timbre; controlling his facial expressions and gestures to eliminate the slightest hint of "crude masculinity" and sexual interest.

* Parentification is the inversion of roles in the family, where a child is forced to perform adult functions: emotionally or practically caring for a parent, taking responsibility for the family's well-being.

Therapeutic process

Duration — 1.5 years in a setting of once-weekly, 50-minute sessions. Approach — Integrative Causal Psychotherapy, using motifs from Symbiodrama. Psychoanalytic interventions for interpreting parentification played a significant role in working with this case. The main directions of work were lifting prohibitions on aggression and separation, processing traumatic affects and suppressed aggression, appropriating and integrating male gender identity, and forming adaptive coping strategies in communication and intimate relationships with women.

Outcome and Follow-up

Sustained clinical remission of symptoms was achieved. The patient began forming love relationships with women, entered into harmonious sexual relationships, which later led to a successful marriage. Boundaries and healthy distance appeared in the relationship with his mother. Masculinity and healthy aggression became integrated parts of his personality.

Conclusion

The presented case illustrates that in invasive traumas, for restoring boundaries and the Ego's metabolic function, awakening healthy aggression plays a paramount role. This is achieved by lifting prohibitions on its awareness and expression, processing suppressed aggression, and forming effective coping strategies for protecting personal boundaries.

Type 3. Egotomic Trauma

Egotomic trauma (from Latin *ego* — “I” and Greek *tomē* — “cutting, sectioning”) is a type of psychological trauma associated with the irreversible loss of a highly significant element of the mental structure that served as a support for the Ego through attachment or identification. Such a loss disrupts the inner integrity of the personality, leading to a persistent Ego-dissociative disintegration, and evokes profound mental pain whose emotional core is existential emptiness and anguish, accompanied by a sensation of a “torn-out part of oneself.”

The essence of this trauma lies in the loss of an internal object that performed a stabilizing and structuring function for the Ego. This loss leaves behind a structural deficit in the internal representational system, leading to a collapse of internal support, disturbance of narcissistic balance, and continuity of existence. A future devoid of the object feels impossible. Affective dissociation arises — a split between intellectual understanding of the loss and its emotional rejection, forming “frozen grief” and splitting off parts of the Ego associated with the lost object.

Examples of possible situations of such loss: loss of a close person (death, breakup of a relationship), loss of health and functionality (a disabling illness, age-related loss of function), loss of a role, status, identity, symbolic losses (systems of values, habitual way of life, idealized beliefs).

The clinical phenotype is characterized by the following disturbances:

- *Affective*: Chronic yearning and mental pain, anhedonia and emotional numbness, attacks of acute grief with somatic manifestations, feelings of emptiness and meaninglessness of life.

- *Related to identity*: Sensation of losing a part of oneself, blurring of the boundaries of one's own Self, difficulties with self-identification outside the context of the loss, feeling of “being stuck” in the past.
- *Cognitive*: Intrusive thoughts about the lost object, idealization of the past and devaluation of the present, difficulties with concentration and decision-making, fragmentation of autobiographical memory.
- *Existential*: Loss of life direction and prospects, crisis of meaning and values, feeling of a broken connection with the world, a depressive position with a sense of hopelessness.
- *Somatic*: Sensation of physical emptiness/coldness in the chest or abdomen, psychogenic pain in the heart area, sleep disturbances, loss of energy tone.
- *Interpersonal*: Inability for emotional investment in new relationships, idealization of the lost object, tendency to recreate situations of loss.
- *Behavioral*: Dysfunctional preservation of habits and rituals associated with the lost object, social isolation and refusal of new activities, neurotic search for a real or symbolic substitute for the lost object.

Egotomic trauma forms a specific neuropsychological profile related to the loss of the Ego's structural support and the subsequent collapse of the brain's integrative systems [31–34]. This profile includes several interrelated levels.

1. Primary impairment of the brain's integrative mechanisms

Egotomic trauma strikes at the fundamental systems that ensure a holistic experience of self and the

world. Structures performing integrative functions are the most vulnerable:

- the hippocampus loses its ability to bind context, time, and affect into a unified episodic field, leading to fragmentation of autobiographical memory;
- the anterior cingulate cortex (ACC) loses the ability to flexibly switch between conflicting signals and ensure coherent decision-making;
- the insular cortex stops synchronizing interoception, affect, and the sense of bodily Self, leading to feelings of internal emptiness and alienation from the body;
- the corpus callosum demonstrates reduced efficiency in interhemispheric interaction, weakening the integration of sensory and cognitive streams.

As a result, implicit memory (bodily, sensorimotor, emotional traces) becomes hyperdominant, while explicit/narrative memory becomes fragmented or inaccessible. The person experiences not a story, but a set of disjointed sensory-affective fragments not integrated into a coherent autobiography [35].

2. Dissociative disintegration of brain macrosystems

The inability to maintain integration shifts the brain into an “emergency mode” of functioning. Characteristic changes occur in the operation of large functional networks:

- the Default Mode Network (DMN) becomes either hyperactive (rumination, fixation on the loss) or hypoactive (emptiness, anhedonia, breaks in the continuity of Self-perception);
- systems for automatic threat processing begin to dominate, leading to a crude dichotomization of stimuli: “threat / not a threat”;
- subcortical survival systems (amygdala, periaqueductal gray, bed nucleus of the stria terminalis) are activated by the slightest signal, forming automatic reactions bypassing conscious control;
- the Central Executive Network (CEN) is weakened: the dorsolateral prefrontal cortex loses its capacity for top-down regulation, planning, and cognitive flexibility.

This forms a dissociative mode in which the brain loses the ability to maintain the coherence of experience [36].

3. Neurochemical and pathophysiological circuit sustaining pathology

The neurobiological architecture of egotomic trauma is consolidated by a stable complex of changes:

- neuroendocrine dysregulation: dysregulation of the HPA axis with a shift to an exhaustion phase (reduced baseline cortisol contributes to the predominance of freeze and dissociation

responses) and dysfunction of the oxytocin system (loss of attachment, trust, ability to experience support);

- chronic neuroinflammation exacerbates damage to integrative structures (especially the hippocampus), reduces neuroplasticity, and hinders the restoration of experiential coherence;
- epigenetic changes consolidate increased stress sensitivity, alter the expression of genes related to GABA, glutamate, and glucocorticoid receptors, creating persistent vulnerability and potentially transmitting it to subsequent generations.

4. Formation of autonomous pathological Ego-states (hierarchical collapse)

In egotomic trauma, the brain shifts to a fragmentary organization: rigidly segregated emotional and defensive configurations, anchored in separate activation patterns of the amygdala, insula, ACC, and medial prefrontal areas, begin to activate rigidly, autonomously, and beyond voluntary control. Within our model, we assume that this is not a dissociative splitting of the personality, but rather rigidly segregated, insufficiently integrated Ego-states that can activate alternately, creating the phenomenology of internal fragmentation while preserving an overall unified sense of Self.

The Figure 3 shows how egotomic trauma causes dissociative disintegration of large-scale networks and hyperactivation of subcortical survival systems. This destroys integrative mechanisms, while dysregulation of the HPA axis, oxytocin, neuroinflammation, and epigenetic changes consolidate the process. As a result, a hierarchical collapse of self-regulation occurs, with the formation of autonomous pathological Ego-states and a clinical picture of fragmented identity, dissociation, and loss of a continuous self.

Differential diagnosis of egotomic trauma

From vital trauma:

- longing, psychic pain, passionate desire to regain the lost vs. fear of possible threat repetition, panic before potential annihilation;
- emptiness as a direct consequence of losing a part of the Ego vs. exhaustion from constant hypervigilance;
- sensitivity to triggers related to reminders of the lost object vs. hypervigilance to external triggers associated with threat;
- stress response: withdrawal, collapse, immobility vs. mobilization, hyperarousal;
- attachment impairment: Unavailability vs. Wariness;
- future is meaningless vs. future is dangerous;
- avoidance of the present moment: immersion in the past vs. scanning the future.

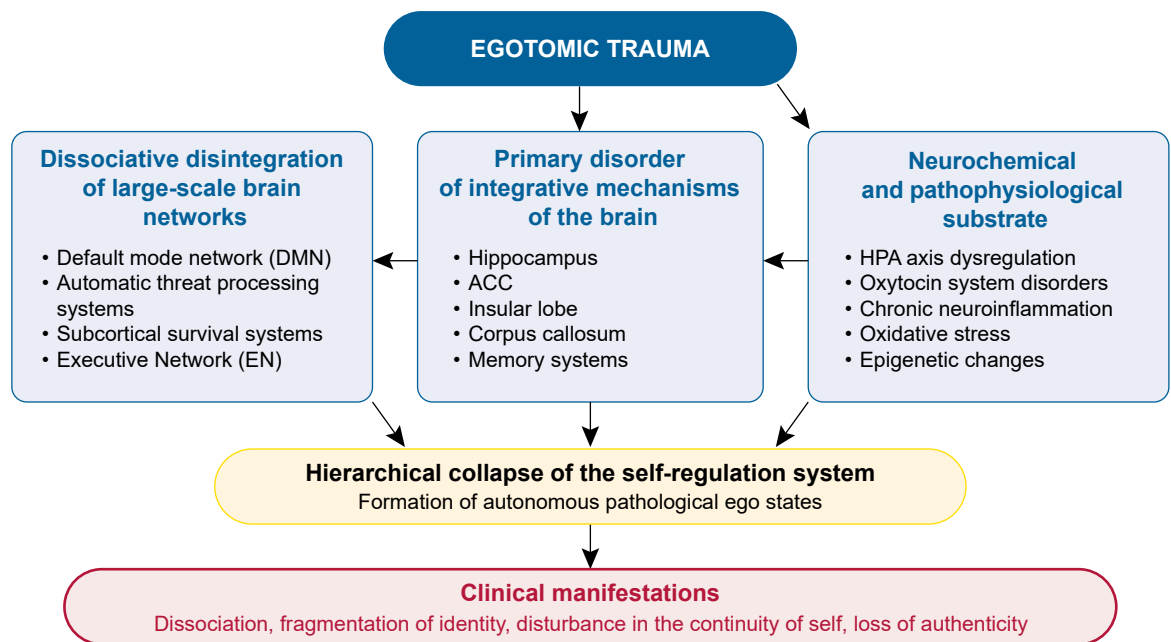


Figure 3. Pathogenetic model of egotomic trauma

From invasive trauma:

- emptiness vs. affective flooding due to boundary violation;
- feeling of internal deficiency vs. feeling of “internal contamination”;
- longing, despair vs. shame, self-disgust;
- body as a lifeless shell vs. body as a betrayer and source of shame;
- triggers: Anything reminding of the loss vs. anything symbolizing boundary violation;
- ego boundaries are preserved, but support is lost vs. boundaries are permeable;
- internal object: Idealized vs. devouring/contaminating;
- inability for closeness vs. fear of closeness.

Therapy should be aimed at restoring the integration of brain structures and include the following key targets:

- processing the emotions of grief;
- legitimizing and channeling suppressed aggression;
- reintegrating the split-off part of the Ego;
- restoring identity;
- repairing the future after collapse;
- psychic investment in new projects and plans.

Egotomic trauma, in the absence of comorbid disorders, may show positive progress within a relatively short period, provided that work focuses on key targets, such as suppressed aggression.

In cases of egotomic trauma, pharmacotherapy, according to our theoretical model, appears to be a method with limited applicability. Its primary mechanism of action — neurochemical modulation — fundamentally differs from the goal of in-depth psychotherapy, which in this context is aimed at restoring psychic integrity through the experiencing and integration of the loss. We hypothesize that this divergence in approaches not only limits the etiotropic potential of pharmacotherapy but also creates a risk of pathogenetic contradiction. Clinical observations within our model point to a number of specific risks. Serotonergic antidepressants, targeting the affective sphere, may alleviate symptomatic anguish but, according to our conceptualization, do not address the presumed primary deficit in integrative functions (of the hippocampus, cingulate, and insular cortices). Anxiolytics (benzodiazepines), in turn, may intensify dissociation and subjective alienation, pharmacologically reinforcing a state of mental distancing.

A key potential iatrogenic effect in this context is the risk of substituting and interrupting the natural process of psychic grief work, which theoretically could contribute to the preservation of a “frozen” state — the central phenomenon of this trauma. Finally, the pharmacological suppression of affect, particularly aggression — which in our model is considered a key resource for separation — may

deprive psychotherapy of an important catalyst for change, complicating the transformation of the pathological bond with the lost object. This criticism does not constitute an absolute contraindication. In cases of severe comorbid symptomatology (e.g., depression with suicidal risk), short-term pharmacotherapy may be considered as an adjunctive measure to stabilize the patient's condition with the aim of making them accessible for subsequent psychotherapeutic work.

Type 4. Constitutive trauma

Constitutive trauma (from Latin *constitutio* — “to establish,” “to form a structure”) is a type of psychological trauma in which the primary formation

of basic psychic structures is systemically impaired due to a chronic deficiency of the environmental conditions necessary for healthy development during the critical period of ontogenesis (up to around 6 years of age). The core affect of this trauma is existential fear and a deep experience of inherent defectiveness.

Constitutive trauma is caused not by a single event but by the overall quality of the developmental environment, which is unable to provide conditions sufficient for the sequential maturation of personality structures in early childhood: unpredictability and unreliability of attachment figures, chronic emotional deprivation, a chaotic environment.

Clinical case: prolonged grief disorder

Anamnesis

The patient, an 18-year-old female, with prolonged grief disorder that developed after the suicide of a close friend. For over 6 months, there was persistent pathological fixation on the loss: intrusive thoughts about the deceased, regular visits to the place of death, social self-isolation with broken family contacts. Academic maladaptation was noted with declining performance due to concentration difficulties, as well as irritability and emotional lability. She sought help after unsuccessful prior psychotherapy at a European clinic, where she was ultimately recommended to switch to pharmacotherapy.

Diagnostic conceptualization

The clinical picture was identified as egotomic trauma leading to prolonged grief disorder. Key diagnostic markers were:

- pathological fixation on the image of the lost object with compulsive reproduction of the circumstances of death;
- social autistic withdrawal and emotional alienation from the supportive environment;
- cognitive deficit in the form of concentration impairments and academic maladaptation;
- affective lability with predominance of irritability and psychomotor agitation.

This symptomatology reflects a structural impairment of the grieving process with the formation of a pathological symbiosis with the lost object, characteristic of egotomic trauma resulting from the death of a significant other.

Therapeutic process

Psychotherapy lasting 15 sessions focused on transforming the intrapsychic dynamics of grief. The key therapeutic mechanism was work with suppressed aggression, which with this patient was primarily conducted using the “Turning Aggression Towards the Therapist” technique. This allowed for legitimizing and channeling repressed aggressive affects and ensured positive dynamics already after the fourth session, with fairly rapid subsequent restoration of affective balance and social functioning.

Outcome and Follow-up

Sustained clinical remission of symptoms with restoration of the premorbid level of functioning was noted. No relapses were observed during a 4-year follow-up period. The patient fully readapted in academic and social spheres.

Conclusion

Successful therapy became possible due to the placement of aggressive affects within the therapeutic space, which allowed for interrupting the pathological symbiosis with the lost object and restoring the process of adaptive grieving. This clinical case demonstrates the importance and efficacy of aggression processing in egotomic traumas.

Mechanism of Ego formation impairment and clinical manifestation

In constitutive trauma, the Ego is not damaged; rather, it never forms adequately in the first place due to a systematic deficit of adequate mirroring, containment, and support during critical developmental periods. This leads to a cascade of impairments manifesting as follows:

- *Lack of stable mirroring* makes it impossible to establish a boundary between Self and Not-Self and to form a coherent self-image. The result is diffuse identity: the person doesn't know "who they really are," constantly "re-assembles" themselves from reflections in others, suffers from unstable self-esteem, and experiences identity crises with the slightest change.
- *The maternal figure's inability to perform the containment function* leaves affects unnamed and unbearable. Severe affective dysregulation forms: periodic "flooding" with affects or, conversely, emotional numbness and alexithymia; derealization and depersonalization become a baseline state of existence; body schema disturbances appear; chronic muscle tension or a feeling of "dissolving" in space; and self-regulation is only possible through external objects or primitive means (self-harm, overeating/starvation, etc.).
- *Breakdown in separation-individuation processes* reinforces symbiotic patterns and leads to the formation of a False Self* as a primary survival strategy and often forms disorganized attachment, leading to intense, unstable relationships and the recreation of traumatic scenarios.
- *Deficit in basic trust and primary narcissistic support* destroys basic self-worth and the sense of one's own right to exist, leading to chronic feelings of emptiness, worthlessness or, conversely, compensatory grandiosity.
- *Impairment in the formation of internal objects* leaves representations of Self and Other fragmented, dichotomous, and unstable. The patient struggles to tolerate ambivalence, has poor mentalization, fragmented autobiographical memory, and dysfunctional core beliefs.

All of this together creates a systemic disintegration of the personality dominated by the False Self, deficits in volitional regulation, maladaptive coping strategies, and self-regulation methods.

Neurobiological picture of constitutive trauma

Constitutive trauma disrupts the sequential maturation of the brain, creating a deficient foundation upon which higher functions cannot adequately form [37–39].

- *Primary deficit: disruption of basic neurosomatic homeostasis.* Early chronic stress and caregiving deficits lead to: 1) hyperactivation of the insular cortex — resulting in impaired interoception and emotional awareness; 2) cerebellar system dysfunction — disrupting the coordination of emotion, attention, and reaction time; 3) persistent activation of the hypothalamic-pituitary-adrenal (HPA) axis (chronic stress mode) — characterized by elevated corticotropin-releasing hormone (CRH), dysregulation of the diurnal cortisol rhythm, and downregulation of glucocorticoid (GR) and mineralocorticoid (MR) receptors; 4) autonomic rigidity — manifesting as low heart rate variability (HRV), impaired ventral vagal complex function (compromising self-regulation via social engagement), and hyperactive dorsal vagal complex (predisposing to freeze and dissociation responses).
- *Secondary deficit: breakdown of attachment and motivation systems.* Decreased density of dopamine D2 receptors in the nucleus accumbens (NAc) leads to anhedonia, apathy, or conversely, a drive for extreme stimulation. Decreased oxytocin receptors in the amygdala and NAc hinders the formation of secure attachment.
- *Tertiary deficit: underdevelopment of higher integrative functions.* As a result of the preceding breakdowns, there is a reduction in volume of the hippocampus and prefrontal cortex, anomalies of the corpus callosum, and fragmentation of large-scale neural networks: Default Mode Network (DMN) — no stable sense of self over time; Central Executive Network (CEN) — deficits in planning, impulse control, working memory; Salience Network (SN) — neutral stimuli are perceived as threatening.

At the molecular level, the described pathology is reinforced by: 1) hypermethylation of NR3C1, OXTR, and GABA-system genes, causing a persistent reorganization of the system into a stress mode; 2) chronically low levels of Brain-Derived Neurotrophic Factor (BDNF), reducing neuroplasticity and the brain's ability to learn and recover; 3) dominance of "survival" neural circuits over social and integrative ones.

The Figure 4 shows how constitutive trauma disrupts the sequential maturation of the brain. Beginning with a breakdown in basic neuro-corporeal homeostasis, it makes normal formation of attachment and motivation systems impossible, which in turn catastrophically impacts the development of higher integrative functions.

* False Self — conformist, hyper-adaptive personality organization.

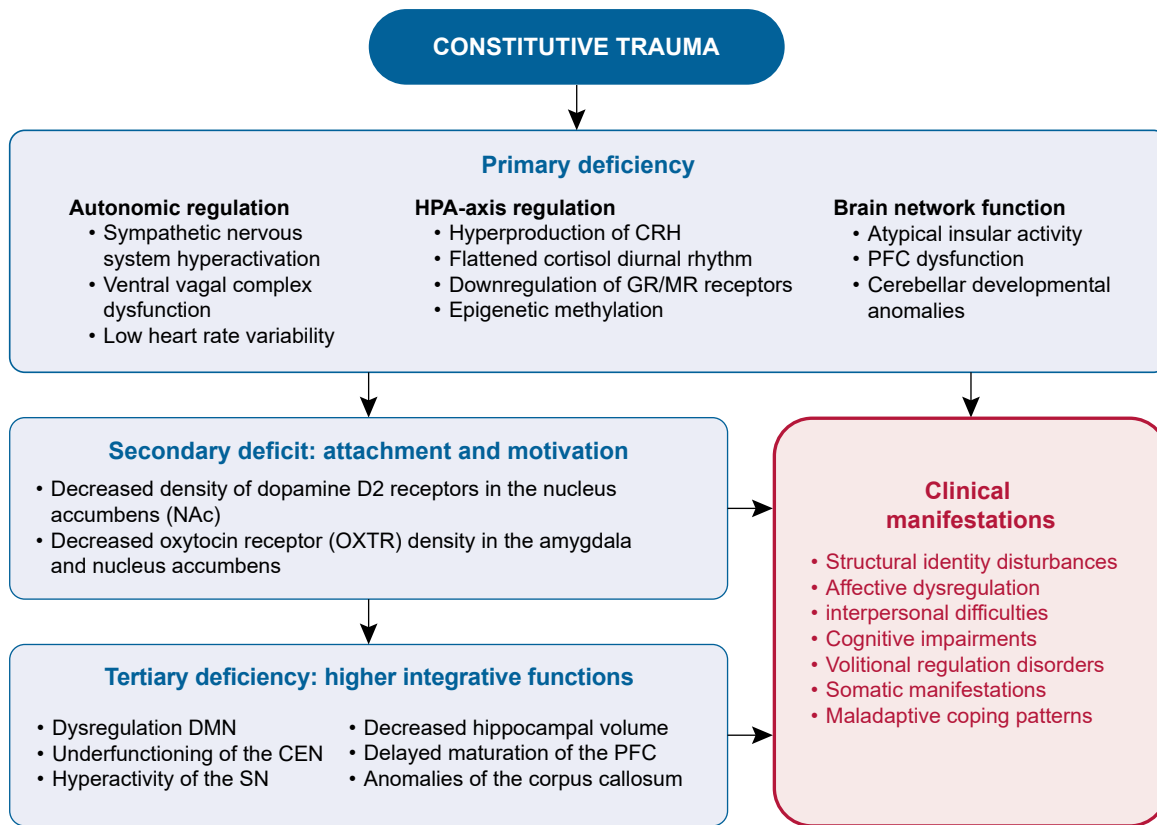


Figure 4. Pathogenetic model of constitutive trauma

Epigenetics and impaired neuroplasticity act as a transversal mechanism, “cementing” impairments at all previous stages and making the brain’s pathological organization stable. The final result of these processes is not just a set of symptoms, but a systemically disintegrated personality, manifested as severe impairments in self-regulation, identity, and interpersonal functioning.

Differential diagnosis from other trauma types

Unlike other trauma types, in constitutive trauma the personality structure itself is traumatic. Symptoms are not a consequence of damage but a manifestation of an inherently deficient personality organization. This is not a “healthy person who experienced trauma,” but a “personality formed by trauma.” Constitutive trauma is distinguished by:

- an inherently deficient, not damaged, Ego structure;
- absence of premorbid normal functioning;
- structural dissociation of personality as the basic architecture;
- absence of continuous autobiographical memory from early childhood.

Thus, constitutive trauma is a complex disorder of neuro-ontogenesis, requiring a multi-level therapeutic approach aimed at sequentially compensating for structural deficits through long-term “reconstruction” of the brain from the bottom up. The psychotherapy plan includes the following targets:

- stabilization and formation of states of basic safety;
- formation of reliable and stable internal objects;
- development of mentalization and reflective function;
- development of the capacity for affective self-regulation and autonomous maintenance of internal stability;
- formation of object constancy and emotional predictability in relationships;
- formation of a coherent self-image.

Constitutive trauma is the primary risk factor for the development of Borderline Personality Structure and represents the greatest challenge in trauma therapy. Unlike other types, constitutive trauma cannot be fully cured because specific structures did not mature during critical periods, and the brain can no longer return to that “window of plasticity.”

Clinical case: constitutive trauma

Anamnesis

Patient: 35-year-old female, married. Presenting issues: generalized anxiety, apathy, volitional deficit, difficulties with separation from parental figures and professional self-actualization.

Diagnostic conceptualization

Clinical picture identified as constitutive trauma. Key diagnostic markers:

- diffuse identity;
- developed False Self;
- narcissistic issues;
- chronic anxiety;
- deficits in volitional regulation;
- unstable self-esteem.

Therapeutic Process

Treatment lasting 90 sessions (once weekly, 50 min.) included the following target:

- formation of an experience of non-judgmental acceptance within the therapeutic relationship;
- weakening of manic defenses and establishing contact with suppressed negative emotions;
- legitimizing the right to boundaries and self-expression;
- restoring contact with aggression as a marker of the Self's existence and a resource for completing separation;
- separation from parental figures;
- work on narcissistic issues;
- development of the True Self;
- formation of professional identity.

Results

- The patient stopped presenting a false "ideal" Self and denying psychological problems;
- Manic defenses weakened, providing access to previously repressed negative emotions;
- Anxiety significantly decreased;
- Self-esteem stabilized.

At the time of writing, psychotherapy continues, focusing on therapeutic goals 4–8.

Conclusion

This case demonstrates that in constitutive trauma, therapy focused on gradually weakening the False Self and restoring contact with the True Self leads to sustained reduction in anxiety, stabilization of self-esteem, and emergence of volitional regulation, even while moderate narcissistic issues persist. The key therapeutic factor was the sequential creation of a new experience of non-judgmental acceptance, along with interpretations of psychological defenses and False Self scripts. This allowed the patient to begin the process of real separation and formation of an autonomous identity. Thus, the case demonstrates the possibility of satisfactory compensation for constitutive trauma and underscores the necessity of long-term therapy (two years or more) to achieve structural personality changes.

However, this trauma can be significantly compensated through long-term psychotherapy, making the patient relatively functional, stable, and emotionally mature. For work with this trauma type, we recommend psychoanalytic psychotherapy combined with Katathymic-Imaginative Psychotherapy.

In working with constitutive traumas, where the pathology is entrenched at the level of reorganized basic neurosomatic networks, pharmacotherapy proves to be of limited efficacy because it cannot

address the root cause—dysfunctional patterns of memory and affective regulation. Its role is confined to the palliative reduction of symptoms, without affecting the etiological core of the disorder.

Discussion

The proposed causal model of psychological traumas deepens and differentiates existing diagnostic models, allowing a shift from a syndromal diagnosis to a pathogenetically substantiated classification.

Integration with ICD-11

PTSD

The picture of Post-Traumatic Stress Disorder corresponds to the phenotype of vital trauma: re-experiencing, avoidance, and hyperarousal reflect a failure of the safety system and fixation on threat. Neurobiological data (amygdala hyperactivation, HPA axis dysregulation) confirm this mechanism. The typology allows differentiation between local and generalized forms of PTSD, which is crucial for prognosis and therapy selection.

Complex PTSD (CPTSD)

CPTSD encompasses the consequences of constitutive and invasive traumas, often in combination. The central CPTSD criterion of “Disturbances in Self-Organization” is the clinical manifestation of the deficit in basic Ego structures characteristic of constitutive trauma. Its components— affective dysregulation, negative self-concept, and relational difficulties—directly derive from the damage mechanisms of this trauma type. Simultaneously, dissociative symptomatology often accompanying CPTSD reflects the mechanism of invasive trauma— boundary violation and subsequent fragmentation.

Attachment disorders

In childhood, attachment disorders represent a direct consequence of constitutive trauma, as their etiological core is a deficit or distortion of early relationships with the caregiving figure.

Integration with DSM-5

PTSD: DSM-5 interprets PTSD more broadly than ICD-11, encompassing situations typical of invasive trauma (e.g., sexual assault). However, the basic clinical structure of PTSD remains closest to vital trauma.

Borderline Personality Disorder (BPD): BPD can be conceptualized as a severe outcome of constitutive trauma, often complicated by features of invasive trauma. Affective instability, identity disturbances, chaotic relationships, and dissociation are clinical manifestations of Ego structure deficits and boundary violations described in our typology.

Clinical Value of Integration

Integrating the causal typology with ICD-11 and DSM-5 creates a bridge between a standard diagnosis and pathogenetically grounded therapy. A standard diagnosis (e.g., 6B41 “Complex PTSD”) answers the question “what?“, while typological analysis (e.g., “constitutive trauma with elements of invasive“) answers the questions “why?“ and “how?“. This enables a shift from symptom-centric to etiotropic and personalized treatment.

Clinical Advantages of Integration

- Focus on mechanisms — from symptoms to understanding their formative mechanisms.
- Individualization of therapy — method selection according to trauma type and severity.
- Predictive value — prognosis determined by trauma type, not nosological category.
- Understanding comorbidity — comorbidity viewed as a logical combination of trauma types.
- Theoretical continuity — different models unified into a single pathogenetic system.
- The proposed typology retains the advantages of standardized diagnostic systems while supplementing them with a deep explanation of psychological trauma mechanisms, creating a foundation for genuinely personalized psychotherapy.

Limitations

The presented model and conclusions have several limitations. First, they are based on a retrospective analysis of clinical cases and theoretical synthesis, which does not allow for drawing conclusions about causal relationships to the same extent as in randomized controlled trials. Second, the assessment of effectiveness in the described cases was conducted clinically but without the use of a double-blind method. Third, the model requires further operationalization and validation on larger and more diverse clinical samples.

Despite these limitations, the proposed typology represents a heuristic system for clinical reasoning and requires further empirical verification. The aforementioned limitations do not negate the main advantage of the model — its ability to shift clinical thinking from a syndromal to a causal plane. The proposed framework opens new perspectives for personalized trauma therapy and serves as a foundation for the development of targeted therapeutic protocols.

Future Research Directions and Hypothesis Testing

The proposed causal typology represents a heuristic clinical hypothesis. Its validity and practical utility must be established through further research. We propose the following stepwise validation strategy:

1. Operationalization and development of a diagnostic instrument: the creation of a structured clinical interview or a screening questionnaire to identify the dominant trauma type according to the mechanism of Ego disintegration. This will allow for the assessment of inter-rater reliability of the diagnosis.

2. Empirical validation on representative samples: the conduct of cross-sectional studies to test hypotheses regarding specific neurobiological profiles (using EEG, fMRI, and hormonal assays) and psychometric patterns (using test batteries assessing affect regulation, dissociation, and personality pathology) for each type.
3. Testing prognostic and therapeutic validity: the implementation of randomized controlled trials comparing the effectiveness of trauma-type-targeted therapy (according to our model) with that of standard unified treatment protocols for PTSD and complex PTSD.

Conclusion

This article presents and substantiates a new clinical hypothesis — a causal typology of psychological trauma based on the mechanism of Ego disintegration. We propose that it may serve as an effective framework for case conceptualization, shifting the clinician's perspective from a syndrome-focused to a pathogenetic approach, while providing a coherent scheme for selecting therapeutic targets. If the hypothesis is confirmed in future research, it could pave the way for a fundamentally new level of personalization in trauma therapy. The empirical validation plan proposed in the "Discussion" section provides concrete guidelines for realizing this potential.

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