

How a Failure to Symbolise Trauma Produces Dissociation and Repetition Compulsion: Toward an Integrated Psychoanalytic-Neurobiological Model of Psychic Trauma

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ABSTRACT

Psychic trauma has long been understood, within the Janetian and Freudian traditions, as an experience that overwhelms the mind's capacity to symbolise: to transform raw sensory and affective excitation into image and word. Contemporary neuroscience has independently documented the biological mechanisms of an overwhelmed threat-response system, yet the psychoanalytic and neurobiological literatures on trauma have rarely been brought into sustained theoretical dialogue, and the psychoanalytic distinction between a primary register of symbolisation, the transformation of sensation into a first mental image, and a secondary register, the binding of image to word and narrative, has seldom been mapped onto distinct symptom domains. This paper undertakes that synthesis, treating the correspondence between the two literatures' constructs as theoretically motivated rather than empirically established. Drawing on Janet, Freud, Roussillon's distinction between primary and secondary symbolisation, Bion's container-contained model, and structural dissociation theory, alongside the neurobiological literatures on fear circuitry, memory systems, hemispheric lateralisation, interoception, and polyvagal defence, the paper proposes a double-dissociation model in which a failure of primary symbolisation is theorised to produce somatoform dissociation and dissociative amnesia, while a failure of secondary symbolisation is theorised to produce the compulsion to repeat. The model's sensory-somatic limb already has initial empirical support from a companion study; its narrative-linguistic limb remains an untested hypothesis. The paper further distinguishes somatoform dissociation from the neighbouring but conceptually distinct categories of psychosomatic disorder and somatoform disorder, addresses whether symbolisation failure causes dissociation or dissociation forecloses symbolisation, and cautiously situates the model within the somatic idioms of distress documented in conflict-affected sub-Saharan African populations, including survivors of the ongoing Anglophone crisis in Cameroon. The model is offered as a testable theoretical framework, with specific, falsifiable predictions for future empirical research in trauma-exposed clinical populations.

Keywords: Psychic Trauma; Symbolisation; Dissociation; Neurobiology; Psychoanalysis

INTRODUCTION

Clinicians working with survivors of protracted armed conflict repeatedly encounter a puzzle that symptom checklists alone cannot resolve. Two civilians exposed to comparably overwhelming violence, a raid, an ambush, an arbitrary detention, may present in starkly different ways: one intrudes upon the present with

vivid, unbidden re-enactments of the event, while the other reports a numbness, an absence, or a bodily complaint for which no organic cause can be found and for which the patient can offer no coherent account. Among conflict-affected civilians of Cameroon's North-West and South-West Regions, where an internal armed conflict has produced mass displacement and repeated, cumulative civilian trauma exposure since 2016, this divergence is a routine clinical observation rather than an exceptional one (Amin, 2025). The present paper argues that this divergence is not incidental but theoretically patterned, and that the pattern becomes visible once trauma is understood through the lens of a single, underlying construct: psychic symbolisation.

Symbolisation, in the sense this paper adopts, is the psychic work by which raw, unformulated sensory and affective experience is transformed into representable mental content, first into image, then into word and narrative, so that it can be felt, thought about, and eventually spoken. The proposition that trauma which cannot be symbolised is instead evacuated through the body or split off from ordinary consciousness is traceable to Janet's (1889, 1907) foundational clinical studies and was given a parallel formulation by Freud (1920/1955) in his account of a breach of the psyche's protective barrier against excessive excitation.

Since Janet and Freud, two literatures have developed this claim in directions that rarely meet. Psychoanalytic metapsychology, and in particular the contemporary French school associated with Roussillon (1999), has refined the concept of symbolisation into two distinguishable registers, a primary register that transforms sensation into a first mental image, and a secondary register that binds that image to word and narrative, without, however, systematically mapping this two-register distinction onto distinct clinical outcomes. Cognitive and affective neuroscience, for its part, has independently documented in precise physiological detail how an overwhelmed threat-response system disrupts memory encoding, suppresses the brain's language apparatus, and produces the two broad poles of post-traumatic presentation, hyperaroused intrusion and hypoaroused dissociative shutdown, largely without engaging the psychoanalytic vocabulary of symbolisation at all (van der Kolk, 2003, 2014). This paper argues that the two literatures describe the same phenomenon at complementary levels, and that their integration yields a more differentiated and testable account than either supplies alone.

This paper pursues that integration with five specific aims. First, it traces the concept of symbolisation failure from its Janetian and Freudian origins through Roussillon's distinction between primary and secondary symbolisation and Bion's container-contained model of how the capacity to symbolise is first acquired. Second, it reviews the neurobiological literature on the fear circuitry, memory systems, hemispheric lateralisation, and interoception, and shows that these mechanisms supply a physiological correlate for each of the two symbolisation registers. Third, it proposes a double-dissociation model in which a failure of primary symbolisation is theorised to produce somatoform dissociation and dissociative amnesia, while a failure of secondary symbolisation is theorised to produce the compulsion to repeat, and it distinguishes the construct of somatoform dissociation from the neighbouring categories of psychosomatic and somatoform disorder with which it is frequently, and wrongly, conflated. Fourth, it addresses the unresolved question of whether symbolisation failure causes dissociation or dissociation itself forecloses symbolisation. Fifth, it situates the model within the somatic idioms of distress documented in conflict-affected sub-Saharan African populations, and proposes directions for the empirical research the model requires.

Psychic Trauma as A Failure to Symbolise: From Janet to Contemporary Theory

Janet (1889) was the first clinician to systematically link traumatic pathology to a specific failure of psychic functioning. Working with hysterical patients whose symptoms could not be explained organically, he proposed that traumatic disturbance reflects a lowering of the mind's integrative capacity under the impact of vehement emotion, so that an overwhelming experience remains outside the personality's ordinary synthesis rather than being assimilated into it. Janet (1907) drew a careful distinction, since obscured by a century of broadening definitions, between dissociation proper, a division of the personality into insufficiently integrated parts, and the milder alterations of attention or consciousness, such as absorption, with which dissociation is now routinely conflated. That original, narrow conception, in which dissociation is

specifically a failure of integration rather than any alteration of awareness whatsoever, is the conception this paper adopts, and it is the conception on which the model proposed below depends.

Freud (1920/1955) arrived at a structurally similar formulation by a different route. In his account, trauma constitutes a breach of the stimulus barrier, an influx of excitation so large that the psychic apparatus cannot bind it economically, leaving an unbound quantity of excitation that presses for discharge and finds it, in the absence of representation, in repetition rather than in memory. Where Janet emphasised a failure of integrative synthesis, Freud emphasised a failure of economic binding; both, however, describe an experience that has escaped the ordinary work of representation and that consequently returns, in one form or another, as symptom rather than as recollection. This convergence, reached independently and from different starting premises, is what justifies treating symbolisation failure as the organising construct beneath both the dissociative and the intrusive-repetitive faces of post-traumatic pathology.

The modern heir to Janet's formulation is the theory of structural dissociation of the personality developed by Van der Hart et al. (2006), which reconceptualises dissociation as a division between an apparently normal part of the personality, oriented to the tasks of daily life, and one or more emotional parts, fixed in the sensory, affective, and defensive elements of the traumatic experience that could not be integrated. On this account, dissociation is not a vague blurring of consciousness but the structural signature of a failure to symbolise, the same failure Janet described a century earlier, now given a differentiated clinical architecture that distinguishes primary, secondary, and tertiary degrees of structural division according to the number of apparently normal and emotional parts involved.

Primary and Secondary Symbolisation: Roussillon's Paradigm and the Container-Contained Model

Freud's topographical model supplies the vocabulary from which the primary-secondary distinction developed. In the unconscious, psychic energy is unbound and organised around thing-presentations, archaic, sensory, and affective memory traces that cannot themselves be spoken. In the preconscious and conscious systems, this raw material is bound into word-presentations, so that an unconscious experience becomes available to reflective thought and, crucially, to communication with another person (Rabeyron & Massicotte, 2020). The transition from thing-presentation to word-presentation is, in the classical Freudian sense, the essence of symbolisation, and free association in psychoanalytic treatment is designed to facilitate this transition.

Roussillon (1999) refined this bipartite model into a paradigm this paper adopts throughout: a distinction between primary symbolisation, the preliminary transformation of raw sensory and affective experience into a first mental image, a somatic schema, or an affect-representation, and secondary symbolisation, the subsequent binding of that image into verbal, narrative, and reflective form. Before an experience can be spoken, in other words, it must first be pictured, felt as belonging to the self, and organised in space and time; a patient's incapacity may therefore lie not in finding words for pain already mentally represented, a failure of secondary symbolisation, but in the pain never having been formulated into a coherent internal experience at all, a failure of primary symbolisation. This distinction is the conceptual hinge of the model developed in this paper, because it is precisely what allows two clinically distinct symptom pictures, somatic and amnesic dissociation on one hand, intrusive repetition on the other, to be derived from a single underlying construct rather than treated as an undifferentiated global deficit.

Because primary and secondary symbolisation are, at present, theoretical constructs rather than constructs with dedicated, purpose-built measures, their empirical study depends on identifying indicators that plausibly index each register even though none was designed explicitly for it. A primary-symbolisation deficit is operationalised here, inversely, through alexithymia, using the Toronto Alexithymia Scale as a proxy on the reasoning that a difficulty identifying and describing one's own emotional states reflects a failure of the image-level binding that primary symbolisation performs; this proxy was used in the companion empirical study summarised below. Candidate indicators for secondary-symbolisation capacity, not yet tested in this research programme, include measures of narrative coherence in trauma-memory narratives, reflective-functioning or mentalisation scales, and structured coding of autobiographical-memory specificity, each of which indexes, in a different way, whether an experience has been bound into organised,

narratable form. Candidate indicators for the compulsion to repeat include intrusion-symptom severity subscales, structured logs of enactment or re-victimisation behaviour, and psychophysiological reactivity to trauma cues. Naming these candidate operationalisations explicitly is intended to make the model's constructs tractable for future researchers, not to claim that any one of them has already been validated as a measure of symbolisation specifically.

Neither register of symbolisation develops autonomously. Bion's (1962) model of the container-contained holds that the infant is initially overwhelmed by raw, unformulated sensory and emotional turbulence, termed beta-elements, which can only be suffered or evacuated, never thought. The infant projects these intolerable elements into the primary caregiver, whose task is to receive them without being destroyed by them and to process them through a state of calm, receptive mental digestion, which Bion called reverie. Through this alpha-function, beta-elements are metabolised into alpha-elements, the building blocks of dream-thought and, eventually, of primary symbolisation, and are returned to the infant in tolerable form. Repeated many thousands of times, this exchange is gradually internalised, so that the child comes to possess not merely the caregiver's detoxified content but the alpha-function itself, the capacity to symbolise autonomously. Magnenat (2016) extended this model with the image of the psychosomatic breast, a caregiver capable of reverie whose regulated presence lays the structural scaffolding for symbolisation, contrasted with the alexithymic breast, a caregiver whose own unintegrated anxiety leaves the infant's projections uncontained, either ignored or returned amplified, and who thereby transmits, developmentally, a vulnerability to symbolisation failure that later trauma may then exploit.

Primary symbolisation is observed clinically, and can be facilitated therapeutically, through the modality of play. In the child's repeated throwing and retrieval of an object, Freud's Fort/Da game, an initially passive and overwhelming experience, the mother's absence, is subjected to an active, repeatable, and pleasurable transformation: the germinal act of primary symbolisation (Freud, 1920/1955; Rabeyron & Loose, 2015). The same logic underlies the therapeutic use of art, psychodrama, and sandplay with adults for whom verbal, secondary symbolisation is not yet accessible: each creates a transitional space in which unformulated experience can first take shape and texture before it needs words.

Clinical Consequences of Symbolisation Failure: Psychosomatics, Alexithymia, and the Compulsion To Repeat

When primary symbolisation fails, overwhelming affect cannot be bound even into mental imagery, and the resulting tension is characteristically expelled through the body rather than represented in the mind. Severe psychosomatic presentations, in which the body is made to speak a distress the mind lacks the vocabulary to stage, are one clinical sequela of this collapse (Rabeyron & Loose, 2015). Alexithymia, the inability to identify and verbally describe one's own emotional states, is another, and is best understood not as a willed withholding of feeling but as the structural hallmark of a psyche in which the transition from primary to secondary symbolisation has not taken place.

Symbolisation, on this account, is theorised to be the mechanism that converts the compulsion to repeat into the capacity to remember, with its failure at the secondary register proposed as what keeps that conversion from occurring; unlike the primary-symbolisation limb of the model, this specific causal claim has not yet been tested directly and should be read as a hypothesis rather than an established mechanism.

These two failure pathways, one bypassing representation altogether and discharging through the body, the other failing only at the final, verbal step, are not merely a conceptual nicety. They predict, as developed further below, two clinically distinguishable symptom pictures, and the present paper's double-dissociation model formalises this differentiation, rarely made explicit in either the psychoanalytic or the neurobiological literature.

The Neurobiology of Symbolisation Failure

The neurobiological literature on trauma, developed largely independently of the psychoanalytic tradition reviewed above, converges with it when read through the lens of symbolisation. Trauma engages

phylogenetically old, subcortical systems that operate faster than, and largely outside, conscious verbal thought, so defensive responses to threat are often initiated before the cortex completes its appraisal (LeDoux, 1996). The amygdala assigns emotional significance to incoming stimuli and drives autonomic and hormonal defence; the medial prefrontal cortex ordinarily exerts top-down regulatory control over this alarm system; and in post-traumatic states this regulatory balance is characteristically disturbed, an overactive amygdala insufficiently restrained by an underactive prefrontal cortex, a pattern replicated across two decades of symptom-provocation neuroimaging (Bremner, 2006; Rauch et al., 1996). The hippocampus, whose volume is reduced in a substantial body of imaging studies of chronic post-traumatic stress disorder (Bremner et al., 1995), ordinarily situates experience in time and place; its impaired function under traumatic overwhelm undermines exactly the contextualisation that would allow an event to be remembered as past rather than re-lived as present.

What French psychotraumatology describes as a failure to inscribe the traumatic real within the symbolic order therefore has an indirect but suggestive neurobiological counterpart, consistent with recent meta-analytic evidence that trauma memories in post-traumatic stress disorder are, on average, less coherent and less well contextualised than memories of comparably significant non-traumatic events, a pattern compatible with impaired hippocampal contextualisation at encoding (Brewin & Field, 2024).

The insula integrates interoceptive signals from the viscera and the autonomic nervous system into the felt sense of the body's condition; its disrupted functioning under trauma produces either an alarming amplification of bodily sensation or a numbing disconnection from it, and supplies a mechanism by which unsymbolised experience is expressed somatically rather than disappearing (Lanius et al., 2010). A recent scoping review confirms that disrupted interoceptive awareness is consistently associated with post-traumatic and dissociative symptomatology across the contemporary literature, lending further support to this mechanism (Leech et al., 2024). Porges's (2011) polyvagal theory, distinguishing a ventral vagal circuit that supports calm social engagement from a phylogenetically older dorsal vagal circuit theorised to mediate a shutdown response to inescapable threat, together with Fanselow's (1994) and Bracha's (2004) account of a graded defensive cascade running from active fight-or-flight to passive freeze and collapse, offers one account of why civilians exposed to inescapable-threat scenarios, ambush, detention, the destruction of a home, are disproportionately likely to have recruited the immobilisation defences associated with dissociative rather than purely hyperaroused presentations (Schauer & Elbert, 2010). The polyvagal theory's specific anatomical and evolutionary claims, in particular the phylogenetic ranking of the two vagal branches and the precision with which distinct autonomic states are said to map onto distinct behavioural presentations, remain a matter of active scientific dispute (Grossman, 2023); the theory is drawn on here only for its broader, less contested behavioural observation, that defensive responses to inescapable threat range across a hyperarousal-to-shutdown continuum, and not for its more specific and more contested physiological architecture.

Table 1: Neurobiological Systems Implicated in Trauma and Their Relevance to Symbolisation Failure

System / process	Key structures or mechanisms	Effect of traumatic overwhelm	Relevance to symbolisation failure
Fear circuitry	Amygdala, hippocampus, medial prefrontal cortex	Amygdala hyperreactivity with reduced prefrontal and hippocampal modulation	Explains the failure of top-down regulation that would ordinarily bind raw affective excitation to language and context.
Memory systems	Hippocampal contextual encoding versus amygdala-mediated	Impaired hippocampal contextualisation;	Neurobiological correlate of primary-symbolisation failure: sensation is stored without being bound into a

	implicit encoding	encoding shifts toward implicit, fragmentary form	first, contextualised image.
Hemispheric lateralisation	Right-hemisphere affective dominance; relative left-hemisphere (Broca's area) deactivation	Reduced activation of language-dependent regions during traumatic recall	Neurobiological correlate of secondary-symbolisation failure: an image may form but cannot be bound to word and narrative ("speechless terror").
Interoception	Insula and related interoceptive pathways	Disrupted mapping of the felt sense of the body onto representable, ownable states	Supplies the mechanism by which unsymbolised experience is expressed as somatoform dissociation rather than disappearing.
Polyvagal / defensive cascade	Ventral and dorsal vagal complexes; graded fight-flight-freeze repertoire	Oscillation between sympathetic hyperarousal and dorsal-vagal hypoarousal/shutdown	Explains why inescapable-threat exposure recruits immobilisation defences associated with dissociative rather than purely intrusive presentations.
HPA axis / allostatic load	Hypothalamus, pituitary, adrenal cortex; cortisol	Dysregulated cortisol response under chronic or repeated threat	Supplies the physiological substrate of chronic hyperarousal that keeps the subject outside the window within which symbolising work can occur.
Neuroplasticity	Activity-dependent synaptic and structural change across the lifespan	Mechanism by which traumatic patterns become entrenched, and by which they may be reversed	Grounds the possibility that symbolisation-focused intervention can restore an integrative capacity that trauma has disrupted.

Read together, these findings do not compete with the psychoanalytic account of symbolisation; at minimum, they offer a plausible physiological context for it, though the correspondences proposed below are conceptual alignments rather than empirically demonstrated identities, and each should be read as a hypothesis the double-dissociation model generates rather than a finding it rests on. What Freud (1920/1955) described as a breach of the stimulus barrier is proposed to correspond to the overwhelming of the organism's regulatory systems; what Roussillon (1999) described as a failure of primary symbolisation is proposed to correspond to implicit, sensory encoding under impaired hippocampal contextualisation; and what this paper terms a failure of secondary symbolisation is proposed to correspond to the deactivation of the brain's own language apparatus during traumatic recall, a striking but as yet indirect parallel, since no study has measured symbolisation failure and Broca's-area deactivation in the same individuals. Siegel's (2003) framework of interpersonal neurobiology, in which psychopathology is understood as a failure of neural integration across differentiated systems, offers a plausible organising principle for this convergence: what neuroscience calls integration and what psychoanalysis calls symbolisation may be two descriptions, at two levels of explanation developed within different epistemological traditions, of a related phenomenon, though treating them as strictly equivalent would overstate what the evidence, reviewed construct by construct above, currently supports.

Reconceiving Dissociation: From Janet's Narrow Definition to Structural Dissociation Theory

Since Janet, two broad conceptions of dissociation have competed for the term. A narrow conception, consistent with Janet's own usage, restricts dissociation to a division of the personality into insufficiently integrated subsystems. A broad conception, which has dominated since the 1980s and is reflected in the progressively wider definitions offered across successive editions of the Diagnostic and Statistical Manual of Mental Disorders, extends the term to any disturbance in the normally integrated functions of identity, memory, consciousness, or perception, thereby absorbing common and largely non-pathological alterations of consciousness, such as absorption, alongside genuine divisions of the personality (Van der Hart & Dorahy, 2009; American Psychiatric Association, 2013). The narrow conception matters for the present model because it, and not the broad conception, is what allows dissociation to be read as the structural signature of symbolisation failure specifically, rather than as an undifferentiated catch-all for any lapse of attention. A recent systematic review and meta-analysis estimates that a clinically significant dissociative subtype is identifiable in a meaningful minority of post-traumatic stress disorder presentations across studies, lending epidemiological weight to the structural distinction drawn here between ordinary post-traumatic presentations and a genuinely dissociative one (White et al., 2022).

Distinguishing Somatoform Dissociation from Psychosomatic and Somatoform Disorders

The double-dissociation model proposed below rests in part on the construct of somatoform dissociation, and the label somatoform, shared across three distinct clinical traditions, is a frequent source of confusion that must be resolved before the model can be stated precisely. Psychosomatic disorders, in the sense established by the Paris school of psychosomatic medicine, denote conditions in which demonstrable, organic illness, a genuine lesion or measurable pathophysiological process, is caused or aggravated by psychological factors; the tissue damage is real and independently verifiable, and psychosomatics studies the pathway by which unmentalised affect comes to be inscribed in the body's actual physiology. Somatoform disorders, renamed somatic symptom and related disorders in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders, are a diagnostic category defined at the level of the symptom and the patient's relation to it, distressing or disruptive physical symptoms accompanied by excessive thoughts, feelings, or behaviours, whether or not a demonstrable medical explanation exists (American Psychiatric Association, 2013).

Somatoform dissociation, the construct this paper draws on, belongs to neither preceding category. Developed by Nijenhuis and colleagues (1996; Nijenhuis & Van der Hart, 2011) within the Janetian structural theory reviewed above, it reads phenomena such as psychogenic analgesia, localised sensory loss, and motor disturbance as dissociative, the somatic expression of a part of the personality that carries the sensorimotor imprint of trauma and remains functionally disconnected from the part that manages ordinary daily life, rather than as either a verified organic lesion or a symptom-focused disorder. An elevated score on a somatoform dissociation measure therefore supports no claim of a diagnosed psychosomatic illness and no claim of a somatic symptom or conversion disorder; it supports only the narrower, theoretically specific claim that the bodily channel of dissociation is active. Table 2 sets the three categories side by side, so that the term somatoform, wherever it recurs in the argument that follows, can be read unambiguously as belonging to the third of these three senses.

Table 2: Distinguishing Psychosomatic Disorders, Somatoform Disorders, and Somatoform Dissociation

Category	Classificatory home	Defining feature
Psychosomatic disorders	A field of clinical study and a stance (Paris and Chicago schools), not a DSM-5 diagnostic category	Genuine, independently verifiable organic pathology that psychological factors cause, precipitate, or aggravate.
Somatoform disorders / somatic	American Psychiatric Association	Diagnosis made at the level of the

symptom and related disorders	(2013)	symptom and the patient's relation to it, with or without demonstrable organic pathology.
Somatoform dissociation	Janetian structural theory of dissociation (Nijenhuis et al., 1996; Nijenhuis & Van der Hart, 2011)	A dissociative part of the personality carrying the sensorimotor imprint of trauma, functionally disconnected from the part managing daily life; measured dimensionally, not diagnostically.

Toward a Double-Dissociation Model of Symbolisation Failure

The preceding sections converge on a differentiated proposal that the existing literature has rarely stated explicitly: that primary and secondary symbolisation are separable capacities, with separable neurobiological correlates, whose failure should be expected to produce two distinguishable, rather than one undifferentiated, symptom pictures. This paper terms that proposal a double-dissociation model, adopting the term in its methodological sense, that two theoretically distinct predictors are each associated with a theoretically distinct outcome, rather than in its clinical sense of dissociative psychopathology, although the two senses meet directly in the model's content.

On the first limb of the model, a failure of primary symbolisation, the inability to transform raw sensory and affective excitation into a first mental image, is theorised to be expressed through the sensory-somatic axis of post-traumatic pathology: somatoform dissociation, in which the unrepresented traumatic imprint is carried in the body as sensory loss, motor disturbance, or unexplained pain, and dissociative amnesia, in which the failure of hippocampal contextualisation leaves the experience unfiled as memory belonging to the past. On the second limb, a failure of secondary symbolisation, the inability to bind an already-formed image to word and narrative, is theorised to be expressed through the narrative-linguistic axis: the compulsion to repeat, in which an unbound experience returns as intrusive image, flashback, or enactment rather than being recalled as history. Figure 1 traces this pathway end-to-end, from traumatic overwhelm to clinical manifestation, and Table 3 summarises the architecture and the neurobiological correlate proposed for each limb.

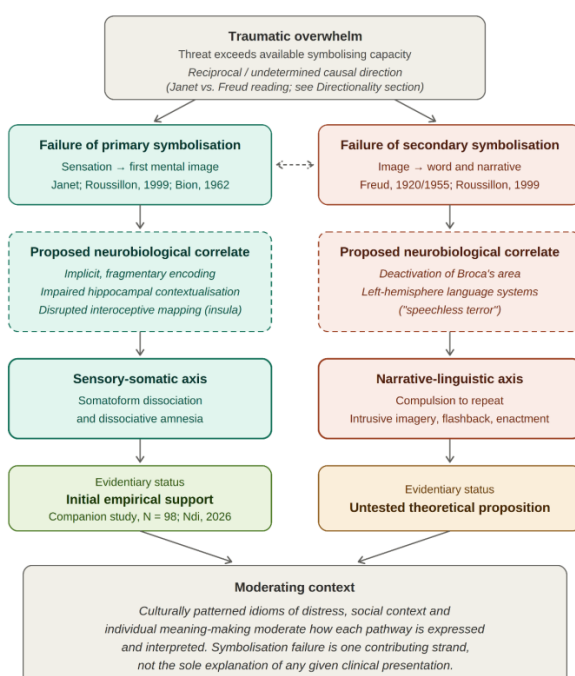


Figure 1: Proposed pathway from traumatic overwhelm through primary- or secondary-symbolisation

failure to the corresponding clinical manifestations, with the current evidentiary status of each limb and the cultural and contextual factors that moderate its expression.

Table 3: The Double-Dissociation Model of Symbolisation Failure

Symbolisation register	Developmental / clinical failure	Theorised symptomatic fate	Principal neurobiological correlate
Primary symbolisation (sensation → first mental image)	Beta-elements remain unmetabolised; raw excitation is not bound into image or somatic schema (failed container-contained exchange; the “alexithymic breast”)	Somatoform dissociation and dissociative amnesia (sensory-somatic axis)	Impaired hippocampal contextualisation; disrupted interoceptive mapping (insula); amygdala-mediated implicit, sensory encoding
Secondary symbolisation (image → word and narrative)	An image or somatic schema forms, but is not bound into verbal, narratable, reflective form	Repetition compulsion: intrusive imagery, flashback, and enactment (narrative-linguistic axis)	Deactivation of Broca’s area and left-hemisphere language systems during traumatic recall (“speechless terror”); situationally accessible, cue-driven memory

Note. The two limbs of the model do not currently carry equal evidentiary weight: the sensory-somatic limb (primary symbolisation → somatoform dissociation and dissociative amnesia) has initial quantitative support from a companion empirical study (Mbofung, 2026); the narrative-linguistic limb (secondary symbolisation → repetition compulsion) is, at present, an untested theoretical proposition, and the neurobiological correlates listed in the final column of this table are proposed conceptual alignments rather than directly demonstrated mechanisms.

The model carries a specific, falsifiable prediction: that a measure of primary-symbolisation deficit should relate more strongly to somatic and amnesic forms of dissociation than to intrusive repetition, while a measure of secondary-symbolisation capacity, or its absence, should relate more strongly to the intensity of repetition compulsion than to somatoform dissociation, a differential pattern of association that a model treating symbolisation deficit as a single, undifferentiated construct would not predict. A first empirical test of the model’s sensory-somatic limb is reported in full in a companion paper by the first author (Mbofung, 2026), and is summarised here only to the extent needed to situate the theoretical argument: in a sample of 98 conflict-affected civilians with confirmed PTSD in Cameroon’s North-West and South-West Regions, a primary-symbolisation deficit, indexed by the Toronto Alexithymia Scale, predicted somatoform dissociation beyond PTSD severity ($\Delta R^2 = .241$, $\beta = .552$, $p < .001$) and predicted dissociative amnesia beyond PTSD severity ($\Delta R^2 = .176$, $\beta = .469$, $p < .001$), in each case remaining a significant predictor once trauma severity was controlled. These findings support the first limb of the model stated here; a corresponding test of the second limb, relating secondary-symbolisation capacity to the intensity of repetition compulsion, has not yet been conducted and is identified as a priority below. The theoretical case for why this differential, two-axis pattern should be expected in the first place, rather than a single, undifferentiated symbolisation deficit predicting dissociation and repetition alike, is the present paper’s specific contribution.

The Directionality Question: Does Symbolisation Failure Cause Dissociation, or Does Dissociation Impede Symbolisation?

The model as stated reads causation in a Freudian, representational direction: a failure to symbolise leaves

traumatic material unbound, and dissociation is its consequence. An equally well-grounded, Janetian, structural reading runs in the opposite direction: dissociation occurring at the moment of the trauma, so-called peritraumatic dissociation, itself impedes the encoding and integration on which symbolisation depends, so that dissociation is not the product of failed symbolisation but its cause. Peritraumatic dissociation has been described as among the largest known psychological risk factors for the later development of post-traumatic stress disorder (Ozer et al., 2003), and in studies of war-affected children it has predicted later symptoms through memories that are disorganised and lack verbal coherence, independent of the level of war exposure itself.

The evidence available does not adjudicate cleanly between these two readings. Retrospective studies tend to support a predictive role for peritraumatic dissociation, while the handful of genuinely prospective designs report a weaker and less consistent association, raising the possibility that the retrospective findings partly reflect the confounding of current symptom state with recalled dissociative experience (van der Velden & Wittmann, 2008). The most defensible position, and the one this paper adopts, is that the relationship is reciprocal: a pre-existing or peritraumatic vulnerability to dissociation can impede the initial encoding that symbolisation requires, while a subsequent failure to bind the resulting fragments into narrative sustains and deepens the dissociative presentation, in a self-reinforcing rather than a one-directional pathway.

Resolving the relative weight of these two directions requires prospective or two-wave longitudinal designs capable of establishing temporal order, a requirement the largely cross-sectional literature to date has not met, and one the present paper identifies as a priority for the empirical research programme the model implies.

Implications for Conflict-Affected Sub-Saharan African Populations

The overwhelming majority of the neurobiological and psychoanalytic literature reviewed above was developed in North American and European clinical and research populations, and a model built from that literature cannot be applied to a conflict-affected sub-Saharan African setting without explicit reflection on its cultural and contextual validity. Two considerations bear on this question in opposite directions. On one hand, the systems the neurobiological literature describes, the autonomic nervous system, the HPA axis, the fear circuitry, the memory systems, are shared human biology, and there is no principled reason to expect the basic physiology of the threat response to differ across populations; to that extent the model's neurobiological substrate claims legitimate universality. On the other hand, the expression, interpretation, and idiom of distress are profoundly shaped by culture, and the predominantly somatic presentation documented among conflict-affected civilians in the North-West and South-West Regions of Cameroon may reflect culturally patterned idioms of distress as much as, and in interaction with, the neurobiology of somatoform dissociation this paper describes.

This dual commitment, to a universal machinery of threat and integration expressed through a particular cultural and relational world, is directly consonant with the model's own architecture, provided the two are not collapsed into one another. Where psychological distress is characteristically communicated through the body, gastric complaints, chronic headache, hypertension, and other medically unexplained symptoms prominent among the clinical population from which this model is drawn, that somatic idiom is very unlikely to have any single cause: it is more plausibly shaped by the simultaneous interaction of trauma exposure, culturally patterned modes of expressing and interpreting distress, the social meaning attached to particular symptoms within a given community, and the individual's own process of making sense of what has happened to them, of which a dissociative, symbolisation-related mechanism may be one contributing strand rather than the explanation. The sensory-somatic axis of the model, and the sharp distinction it draws between somatoform dissociation and both psychosomatic and somatic symptom disorder, is nonetheless of clinical relevance in this setting: it cautions against automatically reading a conflict-affected civilian's bodily complaint as either a verified medical illness or as a case of somatic symptom disorder, while stopping short of asserting that a dissociative, unsymbolised-trauma reading is the necessary or sufficient explanation for any given presentation; the clinically appropriate stance is one of differential consideration, not the

substitution of one single-cause account for another. African psychodynamic frameworks that theorise mentalisation and meaning-making as fundamentally communal and intersubjective processes, rather than as achievements of an isolated individual mind, offer a complementary resource for the secondary-symbolisation, narrative-linguistic axis of the model, suggesting that the restoration of narrative capacity in these settings may depend as much on collective and relational processes of meaning-making as on individual verbal elaboration (Mayi, 2017; Sow, 1977). A model that imported Western constructs uncritically into this setting would risk both medicalising culturally patterned responses to adversity and overlooking exactly the collective resources on which recovery in such settings may most depend; the present model is offered as one lens among several through which these presentations might be understood, to be tested and refined in this setting, not imposed upon it as a singular explanation.

Future Directions

The double-dissociation model proposed in this paper generates a concrete empirical and clinical research agenda:

- Differential testing of the model's core prediction, that primary-symbolisation deficit relates more strongly to somatoform dissociation and dissociative amnesia than to repetition compulsion, and that secondary-symbolisation capacity relates more strongly to repetition compulsion than to somatoform dissociation, in trauma-exposed clinical populations.
- Prospective or two-wave longitudinal designs, incorporating peritraumatic dissociation measures, capable of adjudicating the directionality question between symbolisation failure and dissociation.
- Cross-cultural validation of the psychometric instruments used to operationalise symbolisation, dissociation, and repetition compulsion in Central African and other sub-Saharan populations, where such validation is presently scarce or absent.
- Designs that pair psychometric proxies of symbolisation with psychophysiological or, where feasible, neuroimaging indices, so that the neurobiological correlates proposed in Table 1 can be tested directly rather than inferred from separate literatures.
- Qualitative and mixed-methods research attentive to culturally specific and communal models of meaning-making, to establish whether narrative-integration and reflective-functioning constructs developed in Western samples adequately capture secondary symbolisation in communal and spiritually framed African contexts.
- Intervention research testing whether treatments that directly target affect symbolisation and mentalisation, building on existing skills-based approaches to trauma-related dissociation (Boon et al., 2011), reduce somatoform dissociation, dissociative amnesia, and repetition compulsion in conflict-affected populations, which would give the model direct clinical, and not only explanatory, value.

CONCLUSION

This paper has argued that the psychoanalytic and neurobiological literatures on psychic trauma, developed largely in isolation from one another, converge on a single underlying construct: the failure to symbolise overwhelming experience. Building on Janet's and Freud's foundational accounts, Roussillon's distinction between primary and secondary symbolisation, Bion's container-contained model, and the structural theory of dissociation, and grounding each construct in its proposed neurobiological correlate, the paper has proposed a double-dissociation model, distinguished the construct of somatoform dissociation from the neighbouring categories of psychosomatic and somatoform disorder, and situated the model within the somatic idioms of distress documented among conflict-affected civilians in sub-Saharan Africa, including survivors of the ongoing Anglophone crisis in Cameroon, while cautioning against reading those idioms as attributable to symbolisation failure alone.

Only the model's first, sensory-somatic limb has so far been tested empirically; the second, narrative-linguistic limb, the unresolved question of causal direction, and the specific neurobiological mappings proposed throughout remain theoretical claims requiring direct empirical test. The model is offered not as a settled account but as a differentiated, falsifiable framework, one that a single undifferentiated notion of

symbolisation deficit could not generate, and one whose empirical testing, in the populations for whom the questions it raises are most urgent, is the necessary next step.

Declarations

Ethical Approval

This paper is a theoretical and conceptual synthesis and reports no new human-subjects data; it therefore required no independent ethical review. The paper draws on a doctoral research programme for which the University of Yaoundé I granted ethical clearance, in conformity with the Declaration of Helsinki; empirical results from that programme are reported separately.

Conflict of interest

The authors declare no conflict of interest.

Data availability

This paper generated no new data. The theoretical model proposed is derived from published sources cited throughout and from the first author's doctoral thesis.

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