

Failure of Relaxation: A Physical and Embodied Account of Panic and Dissociative State Transitions

This paper does not ask why panic or dissociation occurs. It asks how a biological system fails to return.

PART 1: THE PHYSICAL ACCOUNT: FAILURE OF DISSIPATION

1.0 Terminology Discipline

Three distinctions must be established before proceeding, not as definitions but as constraints on how the argument is to be read.

Panic, as used here, does not map onto the DSM category of panic attack. The DSM construct is a symptom cluster defined by discrete onset, identifiable affective content, and a set of somatic correlates. It is designed for diagnostic reliability, not mechanistic description. *Panic* in this paper refers to a class of system states characterized by failure of return: sustained high tension, blocked dissipation, and inability to transition out of a high-arousal configuration. It may or may not involve subjective fear. The affective surface is not the object of analysis.

Presence is not mindfulness. The term is used here in a strictly structural sense: the degree of integration between a subject and their own experience, specifically the coherence of the coupling that makes experience felt as *mine*. This is a system variable, one that can in principle be measured and modeled. It has no relation to meditative awareness or therapeutic attunement, and importing those connotations would distort the analysis.

Control, finally, retains its engineering meaning: a system's capacity to actively modulate its own state. This is distinct from homeostasis, which describes the passive maintenance of a physiological baseline. The two concepts overlap but are not interchangeable. The difference matters because this paper is concerned not with whether a baseline exists, but with whether the system can navigate toward it, and what happens when it cannot.

1.1 The Phenomenological Starting Point: Not "Fear," but "Cannot Come Down"

Across multiple episodes, the most stable and consistently recurring feature is not the emotional content of the experience but what might be called *failure of return*, meaning the inability of the system to transition back toward a baseline state once an episode has begun. This observation resists the standard framing in several ways. Affect is not always present at onset; cognition is often clear, sometimes unusually so; and yet the body

remains locked in a configuration that neither escalates nor resolves. The system appears to be held, not driven.

Four somatic phenomena recur with enough consistency to warrant systematic description. First, exhalation is interrupted or significantly shortened: inhalation can occur, but the breath is cut off before the exhalation completes, as though the system refuses to enter the low-energy phase that a full exhale would initiate. Second, there is a persistent sensation of tension or pressure within the chest, not palpitation and not pain, but something closer to trapped pressure without a release path, localized but directionless. Third, brief episodes of local muscular locking or unilateral weakness occur, sometimes to the point of temporary loss of function in a limb, and notably in the absence of pronounced emotional intensity. Fourth, the post-episode period is marked by autonomic rebound: acid reflux, nausea, gastric discomfort, and fatigue, suggesting that the parasympathetic system attempts to reclaim regulatory dominance only after the acute phase has ended.

What these four phenomena share is not their location or their intensity, but their structural logic: none of them are primarily about the level of activation, and all of them point toward a failure of release. The system is not simply running too hot. Something is preventing it from cooling down.

1.2 Why "Hyperactivation" Is Not a Sufficient Explanation

The conventional account of panic locates the problem in excess: too much sympathetic activation, too strong a threat signal, too intense an emotional response. The implied logic is straightforward: if activation is the cause, then reducing activation is the solution, and a system that has been sufficiently calmed will naturally return to baseline. This model is not wrong in every case. But it fails consistently and specifically in the phenomenology described above.

The failure is not incidental. Activation levels during episodes are not always elevated in any obvious sense; the emotional surface can be quiet while the body remains in full arrest. Stability at the cognitive level, including the presence of clear thought and the absence of felt fear, does not produce stability at the somatic level. And crucially, interventions that

successfully reduce arousal, including deliberate relaxation, controlled breathing, and cognitive reappraisal, do not reliably initiate recovery. The system can receive the signal to return and still not return.

This is the observation that the hyperactivation model cannot absorb. It assumes that recovery is the default: once the input diminishes, the system will find its way back. But the repeated experience here is precisely the opposite: the input may be low, the environment may be safe, the mind may be calm, and yet the body holds. The problem is not that the system is being pushed too hard. The problem is that it has lost access to its own exit pathway. What is missing is not less activation, but a functioning route for dissipation.

1.3 Dissipation Failure: Reframing the Problem in Physical Terms

If biological organisms are treated as open, dissipative systems, as thermodynamic theory has argued since Prigogine and Stengers (1984), then the question of what happens during panic shifts fundamentally. A dissipative system maintains its organization by continuously processing energy: taking it in, routing it through internal structures, and releasing it in degraded form to the environment. The system persists not despite this throughput but because of it. When throughput is interrupted, when energy enters but cannot exit, the system does not simply accumulate more activation. It becomes trapped in a high-energy configuration that it cannot resolve through normal dynamics (Prigogine, 1977).

Applied to the present phenomenology, this reframing changes what counts as the central problem. Panic episodes, understood this way, are not primarily about the magnitude of the input. They are about the failure of the system's dissipation pathways to remain functional under load. The energy is present; the exit routes are blocked. This is why reducing the input, whether by calming the emotional trigger or reframing the thought, does not reliably terminate the episode. The bottleneck is not upstream. It is in the architecture of discharge itself.

Three subsystems serve as primary dissipation channels under normal conditions. The respiratory system, and specifically the exhalation phase, functions as the most direct and

rapid route: extended exhalation activates vagal afferents, shifts autonomic balance toward parasympathetic dominance, and signals the system to transition toward a lower-energy state (Gerritsen & Band, 2018; Porges, 1995). The musculoskeletal system dissipates energy through movement, trembling, and progressive release of muscular tension, the mechanisms that Levine (1997) has described as the completion of interrupted defensive responses, though his "energy" framing requires qualification (see note below). The gastrointestinal system, typically associated with resting-state parasympathetic activity, serves as a secondary indicator of whether the system has successfully shifted its regulatory mode; its disruption during and after episodes is not incidental noise but a sign of failed handoff between sympathetic and parasympathetic dominance.

Note: Levine's use of "energy" in the Somatic Experiencing framework (1997, 2010) lacks rigorous empirical operationalization and cannot be directly equated with thermodynamic energy in the Prigogine sense. The clinical observations are cited for their descriptive accuracy regarding incomplete defensive responses; the theoretical vocabulary is not endorsed.

1.3.1 Breathing as the Primary Dissipation Channel

The respiratory system is not incidentally connected to autonomic regulation; it is one of its primary modulators. Extended exhalation lengthens the cardiac inter-beat interval through respiratory sinus arrhythmia, increases vagal efferent activity, and initiates the cascade of physiological changes associated with parasympathetic dominance (Porges, 1995; Thayer & Lane, 2000). In functional terms, a completed exhalation is not merely a mechanical event; it is a system-level signal that the current high-activation state may end.

In the episodes described in 1.1, this signal is blocked. The exhalation begins but is cut short, not by a conscious decision but by what appears to be an involuntary system refusal. The breath is intercepted before it can complete the regulatory loop. This is not a failure to know how to breathe correctly. It is a failure of the system to permit the transition that a completed exhalation would initiate. The distinction matters enormously for what any intervention could realistically accomplish: no breathing technique can succeed if the system is actively preventing the breath from completing.

1.3.2 Muscular Locking as Peripheral Energy Accumulation

When central dissipation pathways are blocked, energy does not simply remain diffuse. It tends to concentrate in peripheral execution systems, specifically the musculoskeletal apparatus that is the body's most proximal instrument for engaging with the physical environment. Local muscular locking, unilateral spasm, and brief functional incapacitation of a limb, occurring in the absence of strong emotional intensity, are consistent with this routing pattern: the system cannot release energy centrally, so it accumulates in the periphery in the form of sustained motor tone.

This interpretation departs from the standard psychiatric framing of such phenomena as "conversion symptoms" or "somatization," both of which carry implicit assumptions about psychological causation. The physical account does not require those assumptions. It requires only that the musculoskeletal system be understood as one node in an energy-routing network, one that becomes overloaded when upstream pathways are unavailable. The local and non-symmetric character of the symptoms (one limb, not both; one region, not the whole body) is consistent with the uneven distribution of load across a network under partial failure.

1.4 Beta-Band Persistence: The Neural Signature of a System That Will Not Change State

The neurodynamic literature provides a complementary level of description. Beta-band oscillations (approximately 13–30 Hz) have been characterized not primarily as markers of anxiety or emotional intensity, but as the neural signature of *state maintenance* — the brain's mechanism for holding the current sensorimotor or cognitive configuration stable and inhibiting transitions to a new one (Engel & Fries, 2010). On the "status quo hypothesis," beta power is enhanced precisely when the system is committed to preserving its current state, and pathological elevations of beta are associated with the failure to initiate transitions, functioning as a "brake" on state switching rather than an indicator of drive level (Jenkinson & Brown, 2011).

Within the predictive coding framework, beta oscillations carry the top-down feedback signals that represent the brain's current model of the world, while gamma oscillations carry the bottom-up prediction errors that would update that model (Bastos et al., 2012, 2015). Sustained beta dominance in this context is equivalent to the brain insisting on its

current prediction against incoming evidence, refusing to revise its state estimate regardless of what the peripheral and interoceptive systems are reporting. The body is attempting to signal that conditions have changed; the cortex is not receiving the update.

In the episodes described here, this framework maps onto the phenomenology with unusual precision. The system is not simply anxious. It is locked into a configuration that it will not abandon, not because the configuration is reinforced by ongoing threat, but because the mechanism for releasing it is not operating. The beta-level account and the dissipation-failure account are not competing explanations. They are descriptions of the same phenomenon at different levels of the system.

1.5 Dissociation as a Secondary Protective Reconfiguration

When dissipation failure is sustained, when the system remains in a high-energy locked state without accessing a release pathway, a second-order response becomes available. Rather than maintaining full integration between the subject and the ongoing experience of the body's state, the system can reduce the bandwidth of that coupling. The result is what is clinically called dissociation: affect is blunted, somatic signals are attenuated, the sense of owning the experience recedes, and a kind of observer position emerges that is relationally distant from the body's distress.

Lanius and colleagues (2010) have characterized this as a "regulatory overmodulation" response, in which prefrontal structures hyperinhibit limbic activation when that activation would otherwise become unmanageable. Schauer and Elbert (2010) locate it as a late stage in a graded evolutionary defense cascade, occurring when parasympathetic collapse begins to replace sustained sympathetic arousal under conditions of inescapable threat. Both accounts converge on the same structural point: dissociation is not the failure of regulation but a form of it, a protective reconfiguration that preserves system integrity by reducing the load on integration itself.

This framing is not merely taxonomic. If dissociation is a secondary protective response to sustained dissipation failure, then the correct therapeutic target is not dissociation per se. Treating dissociation directly, whether by suppressing it, interpreting it, or resolving its affective content, addresses the adaptive response rather than the underlying

condition that made it necessary. The system will not relinquish a protective mechanism until the danger that required it has passed. The danger, in this case, is the locked high-energy state. That is where the problem lives.

1.6 The Temporal Structure: Why Episodes Begin Without Trigger and End Without Resolution

The dissipation-failure model resolves a persistent explanatory puzzle: why panic and dissociative episodes so frequently lack identifiable triggers, and why their resolution is abrupt rather than gradual. In an activation-based model, the absence of a trigger is anomalous; something must have initiated the excess input. In a state-transition model, no such initiating event is required. The system enters a high-energy attractor not because something pushed it there, but because its internal dynamics crossed a threshold under conditions that had been accumulating over time. The "trigger," when one is identified at all, is typically a perturbation that destabilized a system that was already near a critical point, rather than a cause in any strong sense.

The abrupt resolution is equally characteristic. Recovery from a dissipation-failure state does not occur through gradual deactivation; it occurs when a dissipation pathway suddenly becomes available. The breath completes. The muscle releases. The gastrointestinal system shifts. The system does not wind down incrementally from a peak; it transitions from one configuration to another. This is the behavior of a system crossing an attractor boundary, not a system gradually depleting its fuel supply. The dynamical systems literature on critical transitions in complex systems, developed in ecological contexts (Scheffer et al., 2009) and applied to psychopathology by van de Leemput and colleagues (2014), provides the formal vocabulary for this pattern, including the observation that recovery can be as sudden as onset.

1.7 Why Panic and Dissociation Are Better Modeled as State Transitions than as Symptom Fluctuations

The symptom-fluctuation model, which underlies most clinical measurement of anxiety and dissociation, treats episodes as quantitative variations along continuous dimensions: more or less anxious, more or less dissociated, more or less symptomatic. State-transition models, by contrast, propose that the relevant phenomena are qualitative shifts between

distinct system configurations, understood as attractors in the state space, that have different stability properties, different internal dynamics, and different resistance to perturbation.

The distinction has practical consequences. In a symptom-fluctuation model, the goal of intervention is to move the patient's position along a continuous scale toward the healthy end. Small movements count as progress; regression is measured as movement in the opposite direction. In a state-transition model, what matters is whether the system can cross the boundary between attractors, a threshold event that depends not on average symptom level but on the basin of attraction the system currently occupies and the conditions required to exit it (Borsboom, 2017; Hayes & Andrews, 2020). These are not interchangeable models with different vocabularies. They make different predictions about what interventions will do, when recovery will occur, and what counts as meaningful change.

1.8 Summary: A Positional Statement, Not a Conclusion

This section has made one move: it has relocated panic and dissociation from the category of emotional events into the category of system states defined by their dynamics rather than their content. The body phenomenology points toward failure of dissipation. The neurodynamics of beta-band persistence points toward locked state maintenance. The protective logic of dissociation points toward a system that has exhausted its primary discharge pathways and is now managing load by reducing integration. The temporal structure of episodes, with abrupt onset, abrupt resolution, and independence from obvious triggers, points toward attractor dynamics rather than continuous modulation.

Nothing in this account addresses diagnosis, psychological meaning, treatment, or ethics. Those questions require different frameworks. This section establishes the physical ground on which those frameworks must be erected, or against which they must be tested. The argument that follows depends on this ground holding.

PART 2: ANALYTICAL FRAMEWORK: WHY DISSOCIATIVE DISORDERS CANNOT BE EXPLAINED WITHIN AFFECT-BASED MODELS

This section does not argue that affect-based models are wrong. It demonstrates that they are category-mismatched.

2.0 The Central Proposition

Dissociative disorders are not poorly explained by affect-based models. They are explained at the wrong level of organization. The problem is not one of insufficient evidence, inadequate operationalization, or therapeutic failure on the part of clinicians or patients. It is a structural mismatch between the phenomenon and the theoretical apparatus being applied to it, a category error whose consequences ramify through measurement, treatment, and the moral accounting of suffering.

2.1 The Implicit Assumptions of Affect-Based Models

To identify the mismatch, the assumptions embedded in affect-based models must first be made explicit. These assumptions are not stated as axioms in the relevant literature; they are inherited as background commitments that structure the research program, the clinical instruments, and the treatment protocols derived from them. Making them explicit is a prerequisite for showing where they fail.

2.1.1 The DSM's Design Priority: Reliability over Validity

The modern diagnostic system was constructed under a specific historical pressure. Before DSM-III, psychiatric diagnosis was plagued by low inter-rater reliability: two clinicians examining the same patient frequently arrived at different diagnoses. The validity problem, namely whether diagnostic categories captured real and distinct phenomena in nature, was secondary to the more urgent practical problem of whether the system could be used consistently. Robins and Guze (1970) had articulated a set of criteria for establishing diagnostic validity, but DSM-III's architects prioritized the reliability problem first (Hyman, 2010).

The consequence of this prioritization has been extensively analyzed. Hyman (2010), writing as former director of the NIMH, argued that DSM-III's success in achieving inter-

rater reliability produced an unintended "reification" of diagnostic categories, with clinicians treating provisional administrative constructs as validated natural kinds; the result was that reliability became an end in itself rather than a means to validity. Insel (2013), then sitting NIMH director, stated the problem directly: the DSM's strength is reliability; its weakness is its lack of validity. The categories describe symptom clusters that can be identified consistently, but they do not map onto the natural joints of biological systems in any way that could ground mechanistic explanation or targeted intervention.

The relevance of this history to the present argument is specific. When dissociation is absorbed into the anxiety and depression framework, as it routinely is in clinical practice, it inherits not just the diagnostic label but the entire apparatus of assumptions that the label carries. Those assumptions were built for a different phenomenon.

2.1.2 Three Implicit Assumptions of MDD and GAD Frameworks

Affect-based models of depression and anxiety, in their most influential formulations, including Beck's cognitive model (1967, 2008), Barlow's triple vulnerability model (2000), Gross's process model of emotion regulation (1998), and Clark and Watson's tripartite model (1991), share three background assumptions that are individually defensible within their domain of application but collectively inadequate when applied to dissociation.

The first is *affect primacy*: the assumption that emotional states are the primary causal domain, and that changes in affect are the proximal mechanism of both pathology and recovery. In the cognitive model, negative affect is produced by dysfunctional cognitions; targeting the cognitions reduces the affect; reduced affect constitutes improvement. In Gross's model, emotion regulation strategies are evaluated by their downstream effects on affective experience and expression. The subject is understood as someone who *feels*, and the central question is whether what they feel is appropriately calibrated to their situation.

The second is *linear modulation*: the assumption that affect, cognition, and somatic state are related by gradable, bidirectional influence, such that more or less of one produces more or less of another in a predictable and continuous way. This assumption underwrites

the logic of dose-response in both pharmacological and psychological intervention: if you apply enough of the right intervention, you will produce a proportional change in the outcome variable. It also underwrites the interpretation of therapeutic failure as insufficient intervention rather than as fundamental mismatch between model and phenomenon.

The third is *stable subject*: the assumption that the experiencing self, the structure that is doing the feeling, thinking, and regulating, is a constant across the episode being treated. The patient who enters therapy is assumed to be the same patient who will benefit from the intervention; the "I" that learns a coping strategy is assumed to be the "I" that will deploy it when the strategy is needed. This assumption is so foundational that it is rarely articulated. It is visible only when it is violated.

2.2 How Dissociation Violates Each Assumption

2.2.1 Affect Is Not the Controlling Variable

Dissociation can occur in the absence of prominent affect. Episodes may begin with emotional neutrality; they may persist through periods of cognitive clarity; they may resolve without any corresponding emotional shift. The absence of the assumed cause does not prevent the effect. Conversely, strong affective arousal does not reliably produce dissociation, and reducing affective arousal does not reliably terminate it. If affect were the primary causal variable, these relationships would not be possible.

This does not mean that affect is irrelevant to dissociation. It means that affect is not the controlling variable; it is not the variable whose state determines whether the system enters, maintains, or exits the dissociative configuration. The relevant variables are, as Part 1 has argued, structural features of the system's energy dynamics: the state of the dissipation pathways, the degree of beta-band locking, the depth of the current attractor. Affect may be a correlate or a partial contributor. It is not the handle.

2.2.2 The Causal Chain Is Not Linear

In affect-based models, the pathway from stimulus to state to symptom is assumed to be traceable: an event triggers an appraisal, the appraisal produces an emotion, the emotion generates a physiological response and a behavioral tendency. Intervention can enter this

chain at any point. Cognitive reappraisal changes the appraisal; exposure changes the emotional response; relaxation training changes the physiological correlate. The logic is modular and sequential.

Dissociative episodes break this logic at every joint. The stimulus is often absent; if present, its connection to the episode is not reliably causal. The appraisal may be fully intact: the person can accurately describe their situation, identify the absence of threat, and still fail to exit the episode. The intervention may achieve its proximal goal, the thought is reframed, the breath is slowed, the relaxation response is elicited, and yet the system continues in its locked configuration. The conclusion available within the affect model is that the intervention was insufficient or incorrectly applied. The conclusion available within the state-transition model is that the intervention was operating on the wrong variable.

2.2.3 *The Subject Is Not Stable*

This is the most structurally important violation, and the one most consistently overlooked. Dissociation is not a state in which the subject experiences something different. It is a state in which the subject's relationship to experience changes. The self that observes during a dissociative episode is not the same self, in terms of its integration, its access to its own states, or its effective agency, as the self that functions outside of one. The "I" that is instructed to use a coping strategy may not be the "I" that is present when the strategy is needed.

This means that the basic grammatical assumption of affect-based treatment, namely that there is a stable subject who receives the intervention and implements the technique, cannot be taken for granted. What the affect model calls the "patient" is, during a dissociative episode, a differently configured system. The targeted variable (*affect*) is not primary; the mechanism of change (*linear modulation of affect via cognitive or behavioral intervention*) is not accessible; and the agent of change (*the stable, integrated subject*) is not fully present. All three of the model's foundational assumptions have been simultaneously suspended.

2.3 Dissociation's Structural Homelessness in the DSM Framework

The clinical consequence of this mismatch is that dissociation has no stable diagnostic home. In practice, dissociative presentations are routinely absorbed into anxiety or depression diagnoses, where they are treated as secondary features, severity indicators, or complicating complications of the primary affective pathology. This absorption is not arbitrary; it reflects the genuine co-occurrence of dissociation with mood and anxiety disorders in clinical populations. But co-occurrence is not identity. The fact that a person who dissociates also experiences depressive episodes does not establish that dissociation is a variant of depression or a symptom of the same underlying process.

The consequence is systematic distortion of the treatment target. If dissociation is understood as an intense form of anxiety, the appropriate intervention is anxiety reduction. If it is understood as a manifestation of depression, the appropriate intervention is mood stabilization or antidepressant pharmacotherapy. Neither of these targets addresses the structural features that Part 1 has identified as central: the failure of dissipation pathways, the locked attractor state, the protective reduction of presence-integration. The interventions may produce real changes in the co-occurring conditions; they cannot produce targeted change in the phenomenon they have misidentified.

2.4 A Proposed Orthogonal Coordinate System

This section does not propose a new diagnosis. It proposes a reorientation of the coordinate system used to describe the relevant phenomena, a shift that follows directly from the structural analysis above.

Affect-based models use a single primary axis: affect intensity, or more precisely, the degree of negative affective valence and arousal. Positions on this axis correspond to diagnostic categories: high negative affect with high arousal maps toward anxiety; high negative affect with low arousal maps toward depression. The assumption is that this single axis captures the most clinically important variation in emotional pathology.

The present analysis suggests that a second axis is necessary, and that it is orthogonal to the first: *integration* or *presence*, meaning the degree to which the subject is coupled to their own experience, able to access their own states, and operating as a coherent agent in relation to their body and environment. This axis is independent of affect intensity. A

person may be highly integrated and highly aroused (panic with full presence), highly integrated and low-aroused (calm, functional), low-integrated and highly aroused (dissociation under threat), or low-integrated and low-aroused (depersonalization without distress). The last configuration, the one that corresponds most closely to many presentations of dissociation, is not even visible on the affect axis alone. It looks, from that perspective, like the absence of a problem, when in fact it represents a different kind of problem operating at a different level of the system.

2.5 Why Affect-Based Treatment Cannot, in Principle, Reach Dissociation

The argument so far has been diagnostic: affect-based models are applied to a phenomenon that lies outside their explanatory scope. The therapeutic implications follow directly. If the primary variable in dissociation is not affect, then targeting affect cannot be the primary lever of change. If the causal chain is not linear, then interrupting it at a single point will not reliably produce downstream change. If the subject is not stable, then the agent assumed to implement the intervention is not reliably present.

None of this means that affect-based treatments cannot produce any benefit in people who also dissociate. They may reduce co-occurring anxiety, improve mood regulation, build coping resources that are useful during non-dissociative periods, and strengthen the therapeutic alliance. But they cannot resolve the structural condition that Part 1 has identified as primary, and when they fail to do so, the explanation offered by the model is not "the treatment was applied to the wrong target" but "the patient did not respond adequately." This is the explanatory failure that has direct ethical consequences, which will be taken up in Part 6.

2.6 Connecting to Parts I and III

The logical spine of the argument is now visible. Part 1 established that the physical problem is one of dissipation failure and state locking. Part 2 has established that the dominant theoretical framework cannot even describe the phenomenon correctly, because it operates at a different level of organization and makes assumptions that dissociation systematically violates. Part 3 will demonstrate that the therapeutic pathways

built on this theoretical framework are therefore structurally mismatched to the dynamics they are intended to address, not because they are poorly designed within their own framework, but because the framework itself is insufficient for this class of phenomena.

PART 3: ANALYTICAL FRAMEWORK: STRUCTURAL LIMITATIONS OF EXISTING THERAPEUTIC PATHWAYS

This section does not evaluate whether treatments "work in general." It asks why they systematically fail to match the dynamics of panic and dissociation.

3.0 The Central Claim

Current therapeutic modalities are not merely insufficient for dissociative and panic-related system dynamics. They are temporally, structurally, and ethically mismatched to them. The distinction matters. "Insufficient" implies that more of the same intervention, whether higher doses, longer duration, or greater patient engagement, would eventually succeed. "Mismatched" implies that the fundamental architecture of the intervention is not compatible with the architecture of the phenomenon it targets. The argument here is the stronger one.

3.1 A Necessary Clarification

This section does not claim that psychotherapy is ineffective, that pharmacological treatment is without value, or that neuromodulation has no place in the treatment of anxiety and dissociation-adjacent conditions. The evidence on all three modalities is real and in many contexts compelling. The argument is more specific: when these modalities are applied to the particular dynamics described in Parts 1 and 2, including dissipation failure, state locking, attractor rigidity, and destabilization of presence-integration, they operate under structural constraints that are not merely technical limitations but features of the mismatch itself.

3.2 Speech-Based Therapies: The Mismatch in Temporal Scale and Access

Psychotherapeutic approaches, including cognitive-behavioral therapy, psychodynamic therapy, and trauma-focused modalities, share a set of architectural commitments that are well-suited to many clinical problems and poorly suited to the dynamics under examination. Their medium is language. Their mechanism is the modification of cognitive structures, emotional schemas, or relational patterns through a process that unfolds across multiple sessions over weeks or months. Their unit of change is the session, and

the time constant of the change process is measured in weeks at minimum.

Panic and dissociative episodes operate on a time scale of seconds to minutes. The relevant transitions, from integrated to disintegrated, from baseline to locked attractor, from accessible to unreachable, occur faster than the therapeutic medium can track. This is not a failure of the therapy; it is a temporal mismatch. A tool designed for slow, cumulative, language-mediated change cannot reach a phenomenon that completes its dynamics before the session begins.

A further structural problem concerns the accessibility of the therapeutic agent during episodes. In a dissociative state, the coupling between subject and experience is reduced; language, which depends on that coupling for its meaning-making function, is a degraded medium. The patient can speak, but the words may not be connected to the somatic and integrative processes that the therapy is attempting to reach. Therapeutic alliance, the mechanism most consistently associated with positive outcomes across modalities, requires a stable, integrated subject on both ends of the relationship. When the subject is destabilized, when the "I" of the patient is not continuously present, the alliance becomes a resource that is available only intermittently, and its therapeutic function is correspondingly limited.

Beyond the temporal and access problems, there is a structural issue in the logic of repair through connection. Building a therapeutic relationship takes time, involves genuine relational risk, and, for individuals whose dissociative states are precipitated or worsened by relational disruption, carries the specific hazard that the process of establishing connection will itself be a source of destabilization. Each terminated therapeutic relationship represents not merely the loss of a treatment provider but the rupture of a carefully constructed relational structure at the cost of significant vulnerability. For dissociative individuals, this cost is not abstractly psychological. It is a concrete depletion of the integrative capacity that the therapy was attempting to build.

3.3 Pharmacological Treatment: The Trial-and-Error Architecture

Pharmacological intervention for anxiety and depression operates through a systematic search process whose structure is rarely acknowledged explicitly in clinical

communication with patients. Each medication requires four to eight weeks of administration before its effects on the target symptoms can be assessed. Response varies substantially across individuals; pharmacogenomic testing can reduce the search space by identifying metabolic constraints on particular drug classes, but it cannot identify the optimal agent or predict symptom response with precision (Cuthbert & Insel, 2013). The process of finding an effective pharmacological regimen is, in the general case, a multi-trial search through a high-dimensional space with a slow readout.

For individuals whose primary problem is the state-dynamic instability described in Part 1, this architecture has specific costs beyond those it imposes on any patient. Each medication trial represents a perturbation to the physiological system, not merely the target symptoms but the entire autonomic and neuroregulatory landscape within which those symptoms are embedded. Side effects during the titration period may include precisely the features that constitute the primary problem: disrupted interoception, altered body sense, reduced integration of somatic signals with conscious experience. For a person whose dissociation is partly constituted by impaired access to their own body, a drug that further modifies proprioceptive and interoceptive processing during its onset phase is not a neutral perturbation. It is a disruption to the very system whose stability is in question.

The accumulation of failed trials produces a secondary phenomenon that is not adequately captured in standard clinical assessment: the progressive erosion of willingness to undergo further perturbation. After multiple experiences in which an intervention that was presented as potentially helpful produced instead instability, altered body sense, or new symptoms, the rational response is to resist future interventions, not as non-compliance or treatment resistance in the standard clinical sense, but as learned avoidance of the class of events that has reliably produced destabilization. This distinction matters for how the resistance is interpreted, and for who is held responsible when treatment is declined.

3.4 Physical and Somatic Interventions: Promise and Limitations

Physical interventions, those that act directly on the physiological substrate rather than through linguistic or pharmacological mediation, are in principle better aligned with the

dissipation-failure model developed in Part 1. They operate on the relevant timescale; they do not depend on linguistic access to the disintegrated state; they can potentially interact with the somatic processes, respiratory, autonomic, and musculoskeletal, that constitute the dissipation pathways themselves. This theoretical alignment is real and is one reason that body-oriented therapies, vagal stimulation protocols, and neurostimulation approaches have attracted increasing interest in the treatment of trauma-adjacent conditions.

3.4.1 Neuromodulation: Effect Sizes, Variance, and the Placebo Problem

Transcranial magnetic stimulation (TMS) and transcranial electrical stimulation (tES/tDCS) represent the most empirically developed class of non-invasive neurostimulation techniques with relevance to anxiety and dissociation-adjacent conditions. A meta-analysis by Cirillo and colleagues (2019) found large effect sizes for TMS across PTSD and GAD studies (ES = -0.88 and -2.06 respectively), a finding replicated in the broader cross-diagnostic analysis by Hyde and colleagues (2022). These numbers appear compelling.

They require substantial qualification. The Cirillo meta-analysis drew on 17 studies, of which 12 had fewer than 20 participants per treatment arm; effect size estimates from studies of this size are highly unstable and vulnerable to publication bias. The Hyde analysis found that tDCS was ineffective for GAD, and that significant heterogeneity across studies limited confidence in the pooled estimates for most diagnoses. More fundamentally, Razza and colleagues (2023) found in a meta-analysis of 52 sham-controlled TMS trials that placebo responses were large ($d = 1.016$) and had been increasing over the 27-year study period, raising the possibility that a substantial portion of the apparent treatment effect reflects non-specific factors rather than the specific neuromodulatory mechanism. The methodological challenges identified by Duecker and Sack (2015) compound this concern: participants in TMS studies frequently distinguish active from sham stimulation on the basis of sensory side effects, which means that the "sham" condition is not a credible control.

Individual variance presents a separate and perhaps more fundamental problem. Vergallito and colleagues (2022) documented that fewer than half of tDCS participants

show effects in the expected direction, with variance attributable to genetic factors (BDNF polymorphisms), skull geometry, hormonal state, circadian phase, and baseline brain state at the time of stimulation. This is not a problem that can be solved by refining the stimulation protocol or increasing the sample size. It reflects a fundamental biological reality: a stimulation technique that interacts with a complex, high-dimensional, and individually variable system will produce individual-level outcomes that are poorly predicted by group-level parameters. The practical consequence is that for any given individual, the probability of a meaningful response to a specific TMS protocol is substantially lower than the group-level effect sizes suggest.

An additional and critical gap must be acknowledged: no published studies have specifically targeted dissociative symptoms, as distinct from PTSD, anxiety, or depression, as a primary outcome in TMS or tDCS trials. The inference that neuromodulation is relevant to dissociation is based on theoretical grounds and on studies of conditions with which dissociation frequently co-occurs. It is not based on direct evidence.

3.4.2 mECT: Efficacy, Cognitive Cost, and the Limits of Reversibility

Electroconvulsive therapy in modified form remains one of the most efficacious biological treatments available for severe, treatment-resistant depression, with response rates that exceed those of any pharmacological approach in that population. Its relevance to the present discussion is not as a first-line treatment for panic or dissociation but as a boundary case: a physical intervention whose efficacy is real, whose mechanism is not fully understood, and whose side effect profile, including retrograde amnesia and cognitive disruption during the treatment course, raises questions that the present ethical framework (Part 6) will need to address directly. For now, it is noted as a case in which the tension between therapeutic benefit and subjective continuity is starkest, and where the principle of reversibility operates not merely as an abstract ethical constraint but as a concrete clinical consideration.

3.5 The Core Structural Problem: Fast Dynamics Versus Slow Tools

The common thread across the therapeutic modalities reviewed above is a mismatch in temporal architecture. Panic and dissociation are fast phenomena operating on non-

linear dynamics: they transition rapidly between states, their trajectory is poorly predicted by their immediate antecedents, and their resolution depends on system-level conditions rather than on the completion of a therapeutic sequence. The available treatments are slow tools: they accumulate change across sessions, across weeks, across medication trials. They are designed for a different kind of problem, one where the relevant change process can be tracked and influenced over extended time scales by a consistent, available agent applying a consistent method.

This mismatch does not mean that slow tools are useless. They can build the background conditions, including integrative capacity, relational stability, and physiological resilience, that reduce the frequency or severity of the fast-dynamics episodes. But they cannot directly interrupt those episodes, and they cannot substitute for tools that operate at the same temporal scale as the phenomenon they are addressing. The clinical infrastructure for the latter does not currently exist in any systematic form. The gap that Part 7 (the research program) will address is not primarily a gap in therapeutic technique. It is a gap in the fundamental characterization of what is happening at the physiological and neurodynamic levels during these episodes, without which no targeted fast-scale intervention can be designed.

3.6 The Ethical Consequence: How Structural Failure Is Attributed to the Individual

When a treatment modality fails, when the patient does not improve, does not respond, or declines to continue, the explanatory framework generates an account of that failure. Within affect-based models, the available accounts are predominantly patient-centered: insufficient compliance with the therapeutic protocol, failure to adequately implement the cognitive strategies, insufficient motivation for change, "treatment resistance" as a feature of the patient's pathology. These accounts are not offered maliciously; they follow directly from the model's structure. If the model is correct, and if the treatment is correctly applied, then non-response must be attributed to a variable that is within the patient's system. The patient is where the failure gets located.

The argument developed across Parts 1 through 3 implies a different account. If the model is operating at the wrong level of organization, if the treatment is temporally mismatched

to the phenomenon, and if the agent assumed to implement the treatment is not stably present during the relevant episodes, then non-response is not evidence of patient failure. It is evidence of structural mismatch between treatment and condition. The failure is a feature of the therapeutic architecture, not of the person it is applied to.

This distinction has direct consequences for how dissociative individuals are treated in clinical settings, how they are spoken to about their non-response, and what they are invited to conclude about their own agency and capacity. A system that attributes its own structural inadequacy to the people it fails to help is not merely making an intellectual error. It is committing an ethical one. That claim is developed in Part 6.

3.7 The Gap Is Not a Missing Technique

This section ends where it must: not with a solution but with a precise identification of what is missing. The gap is not a new therapy, a refined protocol, or a better pharmacological target. It is a framework: a way of describing what happens physiologically, neurodynamically, and systemically during dissociative and panic episodes that is adequate to the actual dynamics of those phenomena. Without that framework, any intervention designed to address them is aiming at a target it cannot see clearly.

The research program outlined in Part 7 proposes a first step toward that framework: the identification of multimodal biomarkers of state instability and presence-integration, grounded in the dissipation-failure model, constrained by the philosophical and ethical limits developed in Parts 5 and 6. It is a description of what a minimally adequate scientific foundation for this domain would look like, offered not as a claim that it can be built quickly or easily, but as an argument that it is necessary, and that the absence of it is what makes the current therapeutic situation structurally inadequate rather than merely imperfect.

PART 4: PHYSICAL SYSTEMS FRAMEWORK: SOMATIC SYMPTOM SEVERITY, UNCLEAR ETIOLOGY, AND SYSTEM-LEVEL MECHANISMS

This section treats panic and dissociation as behaviors of a physical system under constraint, not as expressions of meaning, conflict, or intention.

4.0 The Goal of This Section

The question this section addresses is not "why does this happen?" It is: under what physical conditions is this outcome a necessary or highly probable result? The analytical posture is one of constraint satisfaction rather than causal attribution. The system has the properties it has; it is subject to the constraints it is subject to; the observed behavior follows from those properties and constraints under the conditions that obtain. This is what "physical explanation" means here.

4.1 Defining the System

A preliminary but essential step is to specify the system boundary. The system under examination is not the brain in isolation, nor the nervous system abstracted from its somatic context. It includes the central nervous system, the autonomic nervous system, the respiratory apparatus, the musculoskeletal system, the gastrointestinal system, and the interoceptive signaling pathways that connect peripheral organs to central regulation. Somatic symptoms are not outputs of this system in the sense of being produced by it and then emitted into a separate domain. They are internal states of the system, functioning as variable values whose magnitude and distribution reflect the current configuration of the system as a whole.

The system is an open dissipative system in Prigogine's sense (1977, 1984): it maintains its organization through continuous throughput of energy and matter, operates far from thermodynamic equilibrium, and exhibits behavior that is qualitatively different from what linear superposition of its parts would predict. It has memory in the sense that its current state depends on its history in ways that cannot be captured by instantaneous measurement. And it exhibits hysteresis: the path by which it arrives at a given state influences the conditions required to exit that state, which is why "what put you here" and "what will get you out" are not reliably the same.

4.2 Why Somatic Symptoms Are Heavy

A characteristic feature of dissociative and panic-adjacent presentations that confounds affect-based models is the severity and specificity of somatic symptoms in the context of relatively unremarkable emotional experience. By the logic of affect primacy, symptom intensity should scale with emotional intensity. This relationship is systematically violated: the chest pressure, the muscular locking, the respiratory arrest, the autonomic rebound can be extreme while the emotional surface remains calm or even flat. From within the affect model, this pattern is anomalous. From within the physical systems framework, it is exactly what would be expected.

Somatic symptom severity in this model reflects not the intensity of the emotional input but the degree of constraint on the system's dissipation pathways. A system that is highly constrained, whose primary dissipation channels are partially or fully unavailable, will produce more severe peripheral symptoms for a given level of input than a system with open pathways, because the energy that would normally be routed outward through breathing, movement, and autonomic transition is instead accumulating at the nearest available accumulation points. The severity is a measure of the bottleneck, not of the emotional event.

4.3 The Cause Problem: From Triggers to Conditions

Clinical and colloquial accounts of panic and dissociation are structured around the concept of a trigger: an identifiable event that caused the episode. This framing is intuitive and occasionally accurate, since some episodes do follow identifiable provocations, but it systematically misrepresents the causal structure of the phenomenon in the general case.

The relevant distinction is between a *trigger* (a perturbation applied to a system that is near a transition threshold) and a *cause* (a necessary and sufficient condition for the transition). In systems near a tipping point, small perturbations produce large transitions; the same perturbation applied to the same system when it is far from its threshold produces no transition at all. What has changed is not the perturbation but the state of the system. The "trigger" in the clinically common sense, meaning the specific event that preceded the episode, is not the cause of the episode in any mechanistically meaningful

sense. The cause is the system state that made the transition possible. And that state is the product of an accumulated history of constraints, failed dissipation events, and autonomic dysregulation that typically predates the precipitating event by hours, days, or longer (van de Leemput et al., 2014; Scheffer et al., 2009).

The clinical and phenomenological consequence of this reframing is significant. The search for a trigger, the attempt to identify the event that "caused" the episode, is not wrong exactly, but it is looking at the wrong temporal scale. The relevant causal history is longer, slower, and less event-structured than the trigger model suggests. This is one reason why episodes so often appear to "come from nowhere": the precipitating perturbation may be trivial or even absent, because the system was already at or near the threshold when it crossed.

4.4 The Energy Framework: Input, Storage, and Dissipation

Within the physical systems framework, the relevant dynamics can be described in terms of three quantities: the rate of energy input to the system, the state of the dissipation channels, and the degree of energy accumulation in peripheral systems. Panic and dissociation episodes are not primarily events of excess input; they are events of insufficient dissipation in the face of ongoing input. The same amount of input that a system with open dissipation pathways would process without difficulty becomes pathogenic in a system whose pathways are restricted.

The dissipation channels identified in Part 1, respiratory, autonomic, musculoskeletal, and gastrointestinal, are not parallel and independent. They are coupled: impairment of one tends to increase load on the others, and failure of multiple channels simultaneously produces a qualitatively different system behavior than partial failure of any single one. The respiratory channel is particularly critical because it is both a direct dissipation route and a regulatory signal: a completed exhalation not only releases energy through the mechanics of breathing but also triggers the vagal afferent activity that initiates autonomic transition. Blocking this channel blocks both the dissipation and the regulatory signal simultaneously.

4.5 Somatic Symptoms as Misrouted Energy

The peripheral accumulation model generates a specific prediction about the distribution of somatic symptoms: they should tend to appear in systems that are anatomically and functionally close to the blocked dissipation channels, and they should be local and non-symmetric rather than diffuse and bilateral. This prediction is consistent with the phenomenology described in Part 1: the muscular locking tends to be unilateral; the respiratory sensation is localized to the chest rather than generalized; the gastrointestinal symptoms are specific rather than distributed.

Non-symmetric and localized symptoms are difficult to explain within affect-based or even standard neurological frameworks, where the assumption is that a central emotional or neural event would produce broadly distributed or at least bilateral somatic effects. The peripheral accumulation model predicts asymmetry and localization as a consequence of the non-uniform coupling between dissipation channels and peripheral musculature: different pathways carry different loads, different muscle groups are differentially engaged in the defensive posture the system has adopted, and the resulting symptom distribution reflects the topology of the network under load rather than the uniform activation of a central state.

4.6 The Respiratory System as the Pivotal Mechanism

Breathing occupies a special position in the dissipation architecture because it is the only autonomic function that is simultaneously under voluntary and involuntary control, and because its regulatory effects operate on two distinct timescales. The phasic effect of a single extended exhalation produces a brief but measurable increase in vagal tone and a corresponding reduction in heart rate and sympathetic activity. The tonic effect of a sustained pattern of slow, extended breathing produces durable upregulation of cardiac vagal tone as measured by HRV, which represents genuine strengthening of the central autonomic network's inhibitory capacity (Thayer & Lane, 2000; Gerritsen & Band, 2018).

The interruption of exhalation described in Part 1 thus blocks both pathways simultaneously. The phasic vagal activation that would signal the system to transition is prevented. The accumulation of experience that would gradually strengthen the autonomic network does not occur. And the body does not complete the mechanical dissipation of the breath itself. Three functions, at three different timescales, are all

blocked by the same interruption. This is why the brevity of the intervention, the exhalation takes only a few seconds, is so disproportionate to its consequences. It is not simply one action among many. It is the single most leveraged regulatory action available to the system, and its blockade has cascading effects throughout the dissipation architecture.

4.7 What This Section Does Not Claim

This section does not argue for a "purely physical" or "purely biological" account of dissociation and panic that excludes psychological meaning, relational history, or subjective experience. The physical systems framework is a level of description, not a reduction. Describing the failure of exhalation in terms of vagal afferent blockade does not eliminate the fact that the exhalation is occurring in a person with a history, in a situation with meaning, in a body that is the site of a life. The two descriptions are not competitive. They operate at different levels and are both necessary for a complete account.

What this section does claim, the minimal commitment it cannot abandon without losing its explanatory purchase, is: somatic symptom severity in this context is not interpretive excess. It is a physically constrained outcome of the system's current configuration. The severity is real, its causes are partially tractable, and the standard affect-based model does not provide adequate theoretical resources for understanding it.

PART 5: PHILOSOPHICAL FRAMEWORK: TRAGEDY, ABSURDITY, SUBJECTIVITY, AND POWER AS EPISTEMIC CONSTRAINTS

These references are not used to interpret data. They are used to constrain the permissible uses of interpretation and intervention.

5.0 The Function of Philosophy in This Paper

Philosophy in this context is not decoration, and it is not the provision of meaning for what the scientific sections have described. Its function is constraining. The scientific and engineering analysis developed in Parts 1 through 4 identifies what happens in panic and dissociative episodes, proposes a physical account of why it happens, and begins to characterize what interventions might address it. What it cannot do, by itself and without additional resources, is specify the limits of legitimate inference from that analysis: what conclusions it licenses, what interventions it justifies, and what it prohibits. That is the work of the philosophical framework.

Four thinkers are mobilized here, each for a specific and limited purpose. They are not sources of comprehensive worldviews to be endorsed wholesale. They are instruments for marking boundaries: the boundaries of what scientific explanation is permitted to claim about extreme states, what clinical and technological intervention is permitted to do in response to them, and what any account of these phenomena owes to the first-person structural reality of the person who undergoes them.

5.1 Nietzsche: Against the Pathologization of Excess

Nietzsche's analysis in *The Birth of Tragedy* (1999 [1872]) distinguishes two principles that structure human experience and culture: the Apollonian, which imposes form, intelligibility, and boundary; and the Dionysian, which dissolves those boundaries in an experience of overwhelming intensity, loss of individual definition, and contact with something that exceeds rational synthesis. For Nietzsche, the tragedy of Greek culture, which he regarded as its highest achievement, lay in holding these two principles in productive tension. The catastrophe he identifies is the Socratic intervention: the philosophical insistence that reason can and should reduce the Dionysian element to something explicable, something manageable, something that can be accounted for

within the framework of dialectical understanding.

The parallel to the present argument is not allegorical. Nietzsche's critique of Socratic rationalism is a critique of a specific epistemic move: the assumption that a state which exceeds rational synthesis is thereby defective, and that the correct response to it is explanation followed by elimination. Applied to the phenomena under examination, the constraint reads: the fact that panic and dissociation cannot be fully reduced to a manageable narrative of cause and meaning does not make them pathological in any fundamental sense. The demand that they be rendered intelligible, that they be made to fit within the structure of a treatment protocol, a diagnostic category, a recoverable causal story, may itself be part of the problem. Bazzano (2019) has argued that manualized psychotherapy replicates precisely this Socratic reduction, converting the irreducible reality of extreme states into manageable cognitive material. The philosophical constraint here is: resist the pressure to resolve extreme states into their explanatory residue.

5.2 Camus: Against False Causal Closure

Camus's *The Myth of Sisyphus* (1991 [1942]) takes as its starting point the experience of absurdity: the rupture between the human demand for clear reasons, for the world to respond coherently to the question "why?", and the world's actual silence on this matter. Absurdity, for Camus, is not a philosophical position but an experience: the lived discovery that the causal structure one had assumed to underlie experience is not there. The scaffolding of habitual sense-making fails, and what remains is a demand that has no object.

The relevance to the present paper is specific. One of the most consistent features of panic and dissociative episodes, as described in Part 1, is the absence of adequate causal explanation: episodes begin without clear triggers, resist rational intervention, and end without corresponding reasons. The clinical and cultural pressure to supply a causal account, to identify what the episode was "about" or to find the psychological meaning that would make it legible, is a response to precisely the experience Camus describes. But supplying false causal closure is not the same as understanding. The constraint that Camus imposes is: do not fill the explanatory vacuum with meaning that the evidence does not support. "You haven't processed your emotion" and "this represents your

unconscious conflict about X" are examples of the kind of explanatory supplement that can be offered without any evidentiary basis, and whose function is to make the explainer comfortable rather than to describe the phenomenon accurately.

The Stranger (1988 [1942]) extends this constraint into the social dimension. Meursault's emotional detachment, his inability or unwillingness to perform the appropriate affective responses, provokes a social response that is ultimately catastrophic. The novel demonstrates that the demand for legible affect is not merely philosophical but normative: societies impose it, and those who fail to meet it are subject to consequences that range from social exclusion to institutional violence. The parallel to the clinical situation of dissociative individuals, who are frequently perceived as not responding appropriately to their own distress or as being suspiciously unaffected by what should affect them, is direct and pointed.

5.3 Jung: Against the Elimination of First-Person Structure

Jung's *The Red Book* (2009), composed between 1914 and 1930 and published posthumously, represents an extended engagement with the claim that first-person structural experience, meaning experience that cannot be translated into the third-person vocabulary of experimental science without loss of its essential character, is legitimate psychological knowledge. The philosophical significance of the work lies not in its content but in its method: Jung's willingness to treat his own inner experience as data, to follow its internal logic rather than to immediately translate it into theoretical categories, and to suspend the demand for premature systematization.

Shamdasani's scholarly study (2003) contextualizes this method as a genuine epistemological alternative to experimental positivism, grounded in a tradition of psychological inquiry that takes the subject-observer relationship seriously rather than treating it as a source of bias to be eliminated. The constraint this imposes on the present paper is methodological: the first-person phenomenological account offered in Part 1 is not a less rigorous form of data than group-level statistical analyses. It is data of a different kind, capturing structural features of the phenomenon that aggregate measures are designed to obscure. The idiographic and the nomothetic are not competing; they are complementary levels of description, and privileging one over the other distorts the

overall picture.

5.4 Foucault: Against the Perpetuation of Power Through Technology

Foucault's *History of Madness* (2006 [1961]) argues that what counts as mental illness is not discovered but constituted, produced by the intersection of medical knowledge, institutional practice, and social power in ways that serve the function of managing individuals who deviate from normative standards of behavior and productivity. The great confinement, the asylum, and the subsequent medical management of madness are not, in Foucault's reading, progressive improvements in the treatment of suffering. They are successive forms of a single project: the normalization of the deviant, conducted under the sign of care and science.

The historical argument has direct contemporary relevance. Iliopoulos (2017, 2022) has traced the Foucauldian analysis into the present diagnostic system, arguing that DSM-5's expanding medicalization of human behavioral variation continues the normalization project under a different technological dispensation. The constraint this imposes is structural: any intervention that takes "stability," "predictability," or "appropriate behavior" as its primary optimization target is replicating the normalization function regardless of its therapeutic framing.

This constraint becomes especially sharp in the context of the research program proposed in Part 7, which involves the development of biomarkers and, potentially, adaptive neurotechnological systems. The historical continuity that Foucault's analysis reveals runs from lobotomy, a surgical intervention explicitly designed to make unmanageable patients manageable, through ECT in its early coercive forms to the present landscape of closed-loop brain stimulation and AI-mediated behavioral monitoring. The continuity is not in the technology but in the use to which the technology is put: managing persons who deviate from normative function on behalf of institutions and social structures that define the norm. The philosophical constraint is: this trajectory must be actively and explicitly refused. Understanding is not the same as control. Identification is not the same as normalization. A system that claims to describe state dynamics while actually serving to produce compliant subjects is not a scientific advance. It is the old project in a new form.

5.5 The Structural Division of Labor

The four thinkers are not making the same argument. Their contributions are differentiated and, taken together, cover the relevant epistemic and ethical perimeter without redundancy. Nietzsche constrains the interpretation of extreme states: they are not defects to be eliminated. Camus constrains the causal narrative: it must not supply false closure. Jung constrains the methodological hierarchy: first-person structural knowledge is legitimate and irreducible. Foucault constrains the deployment of technological intervention: power reproduces itself through the vocabulary of care, and vigilance is required.

This philosophical framework does not justify suffering. It does not argue that panic and dissociation are valuable in themselves, or that the people who experience them should not seek relief. It argues, more precisely, that science and technology, and the clinical practices they underwrite, are not permitted to falsify the experience they are ostensibly trying to help. That is its only claim, but it is not a small one.

PART 6: ETHICAL FRAMEWORK: RESPONSIBILITY, POWER, AND NON-NEGOTIABLE BOUNDARIES

This ethical framework is not outcome-based. It is boundary-based. The question is not what produces the best results. It is what may not be done regardless of results.

6.0 The Architecture of this Framework

Outcome-based ethical frameworks evaluate actions by their consequences, maximizing welfare, minimizing harm, and producing the best attainable result under the relevant constraints. The framework developed here is not of this type. It establishes limits that hold regardless of outcome: actions and practices that are not permissible even if they would, in a given case, produce demonstrably better results than the available alternatives. This is the appropriate architecture for a framework that must remain functional when outcomes are uncertain, when the party who bears the risk is not the party who holds the power, and when the relevant harms are of a kind that cannot be fully compensated after the fact.

6.1 Foundational Principles

Four principles function as axioms in this framework; they are not derived from the analysis that precedes them, and they cannot be overridden by results.

First, *primacy of subjectivity*: first-person experience is not noise. It cannot be legitimately overridden by statistical population data, by institutional assessments of the person's best interest, or by the judgment of a clinician, engineer, or AI system about what the person should experience. A system that treats the first-person account as a symptom to be corrected rather than a datum to be respected has already committed an ethical violation, regardless of its subsequent actions.

Second, *reversibility as a hard boundary*: interventions that produce irreversible changes in the neurological substrate, in subjective continuity, or in the person's effective access to their own states are not permissible by default. They require extraordinary justification, cannot be authorized by institutional protocol alone, and must include, as a design requirement rather than a mere consideration, a genuine exit pathway. The definition of irreversibility must include not only physical changes to neural tissue but

also changes in the phenomenological structure of the self: an intervention that is physically reversible but that permanently alters a person's relationship to their own experience is not reversible in the ethically relevant sense.

Third, *right to exit*: the right to discontinue any intervention, whether pharmacological, therapeutic, or technological, must be unconditional. It requires no justification, imposes no moral obligation to continue, and generates no inference about the person's rationality, pathology, or commitment to their own wellbeing. A framework that treats treatment discontinuation as a symptom or as evidence of pathological resistance has eliminated the right to exit in practice while maintaining it in formal principle.

Fourth, *the non-derivability of blame from predictability*: a system's capacity to predict a person's behavior, or to model the conditions under which their state becomes destabilized, does not generate any obligation on the person's part to prevent the predicted state. The ability to predict is a property of the model. What the model predicts is a feature of the system under constraint. Neither translates into individual responsibility for the outcome predicted.

6.2 Obligations and Prohibitions by Role

6.2.1 For Patients and Persons Experiencing These Conditions

The structural analysis developed in Parts 1 through 4 establishes that panic and dissociation are outcomes of physical system dynamics under constraint. They are not produced by inadequate emotional management, insufficient effort, or pathological unwillingness to recover. The corresponding ethical obligation on the part of clinical and institutional systems is to refrain from attributions of responsibility that the mechanistic account does not support. This is not a charitable concession; it is a requirement of accuracy. "You haven't adequately processed your emotions" and "you are choosing not to get better" are claims that the evidence does not support, and their effect, regardless of their intent, is to transfer the cost of a structural therapeutic inadequacy onto the person who bears the suffering.

The specific situation of dissociative individuals compounds this problem. The invisibility of the condition, the fact that dissociation often preserves cognitive clarity while

disrupting integrative presence, means that the person can appear unimpaired to external observers even when their subjective state is radically altered. The demand that they demonstrate their suffering in affectively legible form, or that their refusal of treatment be interpreted as a symptom of pathology, is a direct instance of the social dynamic that Camus identified in *The Stranger*: the punishment of those who do not perform affect in the expected way.

6.2.2 For Clinicians and Therapists

Clinicians working with dissociative and panic-adjacent presentations are operating within a framework that, as Part 2 has argued, provides inadequate conceptual resources for the phenomenon. The ethical obligation that follows from this is not to use better technique within the inadequate framework; it is to be explicit about the framework's limits. This means distinguishing clearly between "we do not currently have an adequate explanation for this" and "you have not responded adequately to treatment." It means refusing to attribute treatment non-response to patient pathology in the absence of evidence that the treatment was correctly matched to the condition. And it means acknowledging, in clinical communication, that the trial-and-error structure of pharmacological and psychotherapeutic treatment imposes real costs on the patient, costs that are not private burdens but systemic features of the current state of the field.

Two practices are explicitly prohibited within this framework: first, the implication, whether direct or indirect, that dissociation represents "resistance" to therapeutic contact, unconscious avoidance of recovery, or secondary gain from the sick role. These attributions are not supported by the mechanistic account and function primarily to shift institutional responsibility onto the patient. Second, the use of the quality of the therapeutic relationship as a proxy measure for the adequacy of the treatment: a deteriorating therapeutic alliance may reflect the patient's structural instability rather than their unwillingness to engage, and treating the deterioration as clinical information about the patient rather than as information about the match between treatment architecture and phenomenological condition is a category error with ethical consequences.

6.2.3 For Engineers, Researchers, and Neurotechnology Developers

The research program proposed in Part 7, and the broader landscape of neurotechnological development within which it sits, generates specific obligations. The primary one is the obligation to describe before intervening: a research program that seeks to understand the biomarkers of state instability and presence-integration is not thereby authorized to develop interventions that modulate those states. Understanding is a necessary condition for responsible intervention, not a sufficient one. The sufficiency conditions include, at minimum, demonstrated safety, genuine informed consent, reversibility, and the absence of normalization as a design objective.

The Foucauldian constraint developed in Part 5 is directly operational here. Stability, predictability, and behavioral conformity are not legitimate optimization targets for neurotechnological intervention. A closed-loop system designed to detect precursors to a dissociative episode and intervene to prevent it is, from an engineering standpoint, a promising application of the biomarker research. It is also, from a Foucauldian standpoint, a mechanism for producing a person whose internal states are continuously monitored and regulated toward a normative profile by a system that serves institutional rather than personal ends. These two descriptions of the same system are not incompatible; they must both be taken seriously.

6.2.4 For AI and LLM Systems

AI systems that interact with people experiencing panic, dissociation, or psychological distress occupy a position of structural power that is not adequately captured by the standard discourse about AI safety and alignment. Their power is not primarily the power to cause acute harm; it is the power to shape the interpretive frameworks through which people understand their own experience. An AI system that consistently models distress within an affect-based framework, that responds to descriptions of dissociation as though they were descriptions of anxiety, that offers coping strategies premised on the stable-subject assumption, or that generates authoritative-sounding causal narratives for experiences that the scientific literature cannot yet adequately explain, is perpetuating a specific set of category errors at scale.

The ethical requirements here are minimal but non-negotiable: AI systems must express genuine uncertainty about mechanistic claims they are not equipped to make. They must

not generate normative judgments about how a person should manage their experience. They must not map group-level statistical findings onto individual cases as though the individual were a sample from the population. And they must not simulate clinical authority. These are not technical constraints on content filtering. They are requirements that follow from the framework developed in Parts 1 through 5.

6.3 The Historical Warning

The ethical framework concludes where Foucault's analysis pointed: at the recognition that the form of an intervention does not determine its function. Lobotomy was performed under the sign of medicine, science, and care for the patient. The early decades of ECT were shaped partly by custodial motivations that had nothing to do with the wellbeing of the persons treated. The expansion of psychiatric medication in the second half of the twentieth century was accompanied by the pharmaceutical industry's active construction of the diagnostic categories that created the markets for its products. None of these developments was experienced by its practitioners as a departure from therapeutic ethics.

The lesson is not that neurotechnology is inherently suspect, or that biological psychiatry is necessarily complicit in social control. The lesson is that the alignment between technological capability and genuine therapeutic benefit cannot be assumed; it must be demonstrated, continuously, by reference to standards that are not set by the institutions that develop and deploy the technologies. The ethical framework presented here is an attempt to contribute to those standards, offered not as a definitive account but as a set of commitments that the research program in Part 7 must be held to and that must remain in force regardless of what the program finds.

PART 7: FEASIBLE RESEARCH PROGRAM: MULTIMODAL BIOMARKER LEARNING FOR STATE INSTABILITY, PRESENCE, AND DISSOCIATION

Goal: identify, model, and validate multimodal biomarkers of state instability (presence/integration), distinct from affective intensity, across complementary experimental tiers.

7.0 The North Star Question and What This Program Is Not

The research program proposed here is organized around three empirical questions: Which multimodal biomarkers reliably characterize the transitions between stable, unstable, and recovered states, doing so in a way that is distinct from measures of affective intensity? Is the construct of presence or integration empirically dissociable from affect intensity using measurable physiological and neurodynamic signals? And do deep threat circuits, the amygdala in particular, maintain elevated activity and coupling during low-affect conditions in ways that correspond to the state-maintenance dynamics described in Part 1?

These questions are chosen because they address the core theoretical gap identified across Parts 1 through 3: the absence of an empirically validated framework for describing state instability that is not reducible to affect measurement. They are not chosen because they point toward a specific intervention. The program explicitly does not take therapeutic efficacy as a primary outcome variable; it takes identifiability and predictability of state transitions. It does not propose irreversible interventions. And it does not take the normalization of dissociative states as a design objective. These are not strategic disclaimers; they are commitments that follow directly from the philosophical and ethical framework developed in Parts 5 and 6.

7.1 A Three-Tier Architecture

The research program is organized across three complementary tiers, each contributing a different type of evidence and operating at a different level of mechanistic specificity. Their relationship is not hierarchical in the sense that one is more fundamental than the others; they are complementary sources of constraint on the same set of questions, and any conclusion that depends on findings from only one tier will be treated as provisional.

Tier 1 provides breadth and generalizability through non-invasive multimodal data collection at scale. Tier 2 provides mechanistic depth through secondary analysis of stereoelectroencephalographic (sEEG) data from clinically implanted electrodes, enabling direct access to deep circuit dynamics that are invisible to surface-level recording. Tier 3 provides causal controllability through brain organoid and assembloid models, which allow manipulation of network parameters such as excitation-inhibition balance, neuromodulatory tone, and inflammatory state, under conditions that would be unethical or technically infeasible in human participants. Convergent findings across at least two tiers will be required before any claim about mechanism is treated as established.

7.2 Tier 1: Non-Invasive Multimodal Data

The primary data collection tier combines high-density EEG (64–256 channel) with continuous respiratory monitoring, ECG/HRV, and, where participant burden permits, surface EMG and electrodermal activity. The physiological signals are supplemented by event logs: time-stamped self-reports of state transitions that serve as anchors for the computational analysis, enabling the identification of the physiological correlates of onset and recovery rather than just resting-state differences between groups.

The analytical target is not diagnostic classification. It is the learning of state representations, understood as latent variables that characterize the system's current configuration, using self-supervised and contrastive methods that do not require the imposition of affect-based labels. The output is two indices: a presence/integration index that attempts to capture the degree of coherent coupling between subject and somatic experience, and an affect intensity index that captures the conventional measure of emotional arousal and valence. The primary scientific question is whether these two indices can be reliably dissociated, whether there are states characterized by low integration and low affect, high integration and high affect, and all intermediate combinations, or whether they prove to be systematically confounded in ways that would vindicate the affect-model assumption.

Individual-level modeling will be prioritized over group-level averages. The relevant dynamics are intra-individual: the question is whether a given individual's state transitions can be predicted and characterized from their own time series, not whether

the average person in a diagnostic group differs from the average person in a control group. This reflects both the idiographic methodology endorsed by the Jung/Shamdasani discussion in Part 5 and the practical reality that inter-individual variability in these phenomena is too large for group-level patterns to provide useful predictive models at the level of the individual (Vergallito et al., 2022).

7.3 Tier 2: Secondary sEEG Analysis

The second tier uses stereoelectroencephalographic recordings from patients who have been implanted with depth electrodes for clinical purposes, typically for the localization of epileptiform activity prior to surgical resection, and who consent to the collection of additional data during periods of spontaneous psychiatric symptomatology. This is secondary analysis of clinically necessary data; no additional surgical risk is incurred, no symptom provocation is performed, and the duration of implantation is not extended beyond clinical necessity.

The scientific value of this tier is the direct access it provides to the activity of deep structures, including the amygdala, hippocampus, anterior insula, and anterior cingulate cortex, that are inaccessible to surface EEG and that the dissipation-failure and state-maintenance models implicate as central to the observed dynamics. The amygdala in particular occupies a theoretically pivotal position: the literature reviewed in the reference guide establishes that its activity is not limited to fear responses but extends to general vigilance and relevance processing (Davis & Whalen, 2001), that single-neuron recordings reveal activity correlated with the perceiver's internal subjective states rather than with objective stimulus features (Rutishauser et al., 2015), and that intracranial ERP studies demonstrate responses to non-emotional relevance at timescales consistent with ongoing state-maintenance activity (Guex et al., 2020; Murray et al., 2014).

The key empirical question for this tier is whether the amygdala, and its coupling to prefrontal and autonomic structures, shows the activity patterns predicted by the state-maintenance model during low-affect conditions, conditions in which, by the standard fear-circuit account, its activity should be minimal. If amygdala-cortical-autonomic coupling remains elevated during periods of subjectively reported low affect but high somatic tension, this would constitute mechanistic evidence for the central theoretical

claim: that the system is locked not by ongoing emotional input but by a self-sustaining neural dynamic whose emotional surface is not its primary characteristic.

7.4 Tier 3: Brain Organoid and Assembloid Models

The third tier addresses the limitation that is inherent in both Tiers 1 and 2: the difficulty of making causal inferences from observational data in a system too complex to manipulate experimentally. Brain organoids, which are three-dimensional neural tissue cultures derived from human iPSCs, and assembloids, which combine region-specific organoids to enable the study of inter-region communication, provide a model system in which the parameters implicated by the theoretical framework can be systematically varied while network activity is recorded.

The specific sub-questions addressable at this tier concern the cellular and network mechanisms of state locking and dissipation failure: how do changes in excitation-inhibition balance affect the stability of network states and the availability of transition pathways? How do stress-relevant neuromodulators, including norepinephrine, CRF, and inflammatory cytokines, alter the dynamics of network state maintenance and switching? What patterns of network activity are associated with the spontaneous emergence and resolution of persistent high-energy configurations? These questions cannot be answered in the human system directly, but their answers would constrain and enrich the interpretation of the human data from Tiers 1 and 2.

The organoid tier does not claim to model human dissociation or panic. It claims only to instantiate the relevant network dynamics in a controllable system where causal claims are possible. The connection to the human phenomenon is one of mechanistic homology, meaning shared network-level dynamic features, rather than phenomenological or clinical equivalence.

7.5 Validation Criteria

A research program that aims to characterize biomarkers of state instability must specify, in advance, what would count as evidence that the biomarkers are meaningful. Four criteria are proposed. First, test-retest reliability within individuals across days and weeks: a biomarker that characterizes a feature of the individual's system should be stable under

stable conditions and should change predictably under conditions of known perturbation. Second, intra-individual predictive superiority over population-level models: an idiographic model trained on an individual's own time series should outperform a nomothetic model trained on the group, because the relevant dynamics are individual-specific. Third, cross-tier convergence: Tier 1 surface features that are proposed as non-invasive surrogates for deep-circuit dynamics should be validated against the Tier 2 sEEG data from individuals who contribute to both tiers. Fourth, statistical independence from affect scales: the proposed presence/integration index should demonstrate discriminant validity from standard affect and anxiety measures, showing that it captures variance not accounted for by existing instruments.

7.6 Expected Outputs and the Commitment to Description Over Intervention

The expected outputs of this research program are: a validated multimodal representation of presence/integration as a state variable distinct from affect intensity; a characterized relationship between deep threat-circuit dynamics (amygdala, anterior insula) and non-invasive surface markers; and a replicable analytic paradigm for studying state transitions in intra-individual time series that can be applied across research groups and populations.

These outputs are descriptive. They are not, by themselves, a clinical intervention, a treatment protocol, or a device specification. Future work may explore whether the identified biomarkers could inform adaptive support systems, systems that recognize state transitions in real time and provide contextually appropriate support to the individual. Such work would be subject to the strict reversibility and non-coercion constraints developed in Part 6. Within the present program, the distinction between *inform* and *drive*, between *adapt* and *control*, is not rhetorical. It is the ethical boundary that separates a scientific contribution from a normalization technology, and it must be maintained with precision.

7.7 Failure Modes and Risk Management

Intellectual honesty requires an explicit account of the ways this program might fail. Sample heterogeneity across the clinical populations that contribute to each tier may limit

the generalizability of findings even when intra-individual models succeed; this risk is managed by prioritizing idiographic over nomothetic conclusions. The sEEG sample will be small, since the number of patients with implanted electrodes who consent to additional data collection and who experience relevant symptomatology during the recording window is necessarily limited, and will function as a mechanistic anchor for the program rather than as its primary source of evidence. Multi-modal synchronization across recording systems introduces technical noise that can degrade event-aligned analyses; this risk is managed by conservative event-labeling protocols and weak-supervision approaches that do not require precise onset timestamps. And the risk of ethical misuse, of findings being applied to normalization or surveillance purposes by parties not bound by the commitments developed here, cannot be fully controlled by the research program itself. It can only be managed by making those commitments explicit, public, and structural rather than merely aspirational.

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