

Biology Note 10: Anesthesia as Controlled Suppression of 13DD Fine-Layers

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Medical disclaimer: This note is a structural analysis of anesthesia within the Self-as-an-End (SAE) framework. It is not a substitute for clinical diagnosis, treatment decisions, or drug administration guidance. Anesthesia safety must follow established anesthesiology protocols.

1. The Question

What does anesthesia actually suppress?

The question looks simple, but a century of clinical practice has not produced a clear answer. Clinicians know that a given dose of propofol puts a patient under in thirty seconds. They know ketamine produces reports of "leaving my body." They know midazolam lets patients cooperate and converse, then leaves them with no memory of what happened. These facts form the backbone of clinical anesthesiology. But when asked *why* these agents all produce loss of consciousness, or more precisely *what component of consciousness they remove*, existing theories give inconsistent answers.

Each mainstream theory has a visible gap. Integrated Information Theory (IIT) attributes consciousness to Φ and explains anesthesia as Φ reduction. The description is correct, but it cannot explain why entirely different molecular mechanisms—GABA potentiation, NMDA blockade, potassium channel activation—all converge on Φ reduction. If Φ were the fundamental variable, it should have a single regulatory entry point rather than being jointly susceptible to multiple unrelated molecular pathways. Global Workspace Theory (GWT) frames consciousness as integration across a distributed workspace and explains anesthesia as the collapse of this integration. But automated functions (heartbeat, respiration, thermoregulation) remain intact under anesthesia; GWT does not explain why low-level integration is preserved while high-level integration disappears. The thalamic gating hypothesis is partially correct but incomplete—the thalamus is affected by some anesthetics more than by others. And "cortical information integration is disrupted" is a phenomenological description, not a mechanism.

The position of this note is different: **anesthesia is not the object of SAE research—anesthesia is a tool SAE uses to see the internal structure of 13DD**. This one sentence locates the entire note. Anesthesia can serve this role because it provides a controllable, dose-dependent, reversible suppression of fine-layers. Different doses suppress different layers; different agents prefer different layer combinations; during recovery, different layers come back online at

different rates. These differences constitute **structural natural experiments** that make the internal partitioning of 13DD observable.

This note does not stand alone. It builds on several existing SAE papers.

A18 Dreams (DOI 10.5281/zenodo.19176873) established the sequential-dependency framework for DD layers: sleep shuts down top-down, waking rebuilds bottom-up. A18 treats anesthesia as a comparative case, noting that anesthesia is a "synchronous" suppression (driven by drug pharmacokinetics) while sleep is "asynchronous" (each layer follows its own dynamics). A18 uses anesthesia as a contrast but does not develop the internal structure of anesthesia itself. This note opens up that space: beneath the "synchronous" surface, anesthesia has fine-grained layered structure.

A19 Life-Death-Self (DOI 10.5281/zenodo.19201237) positions anesthesia as "the closest reportable experience to death" and discusses consciousness discontinuity around stitching mechanisms. A19 addresses macro-positioning (anesthesia, death, sleep as a structural spectrum) but does not enter the internal architecture of 13DD. This note enters that architecture beneath A19's macro-level.

Bio Note 7 Dissociation (DOI 10.5281/zenodo.19600029) identifies three functional positions of 13DD: narrative, negation, conflict monitoring. Bio Note 7's three functional positions constitute the **functional view** of 13DD—what 13DD does when viewed from outside. This note provides the **layered view**—how 13DD is internally composed. These are two resolutions of the same structure (see §2.1).

Bio Note 9 Memory (DOI 10.5281/zenodo.19635021) built the 11DD memory system and the 13DD filter architecture. Bio Note 9 made a key directional principle explicit: 13DD's refusal is "I do not receive," not "you may not send." Higher layers do not rewrite lower storage; they only gate which traces enter autobiographical narration. This note strictly respects that constraint and refines the filter mechanism at the 13DD-a level.

On top of these, this note's distinctive contributions are:

First, a four-fine-layer structure within 13DD (event-marking, say-no, fear-of-death, asymptotic self), ordered by the four operational modes of chisel (mark / add / multiply / AND).

Second, a functional substructure for 13DD-a: bidirectional management of ownership metatags (encoding side and retrieval side), repeated re-acknowledgment, and first-person embedding in scene reconstruction. Independent deviation across these subfunctions explains a range of clinical phenomena from HSAM to SDAM to dissociative amnesia to depersonalization.

Third, 13DD-a as an asymptotic limit rather than a closed state. By SAE remainder conservation (ZFCp first law), 13DD-a never reaches full closure; it only asymptotically approaches "complete self." This constraint turns the incompleteness of 13DD-a from a limitation into a driving force: precisely because 13DD-a is never complete, the subject must seek meaning at 14DD.

Fourth, 11DD's ownership metatag dependency on 13DD-a is precisely located, but tag strength is a gradient rather than an on-off switch. Weak tags and strong tags correspond to different degrees of memory ownership, explaining a full spectrum from normal autobiographical memory to traumatic imprints.

Fifth, a fine-layer preference signature for each class of anesthetic agent. Each agent has a preferred path of suppression, given as candidate mappings pending experimental validation.

Sixth, suppression and recovery as independent dynamics. Anesthetic suppression and recovery are not simply reverse sequences of each other—suppression is driven by drug molecular dynamics, recovery by neural network reconstruction. They operate on distinct timescales and must be handled separately.

Seventh, a methodological blind spot in current consciousness-recovery research. Mainstream measurement methods (behavioral tests, self-report, cognitive tasks) all require 13DD-a to be online, which constitutively prevents them from measuring the earlier recovery of 13DD-d/c/b. This note identifies the blind spot and proposes a multimodal protocol to break through it.

Eighth, a precise formulation of the anesthesia-death structural isomorphism. The structural condition for reversibility is **substrate integrity**, not whether any fine-layer reaches zero. SAE's remainder conservation is a structural theorem; it makes no claim about the form of remainder propagation. Questions about soul, theology, or identity continuity lie outside the scope of structural philosophy.

These contributions converge on a single proposition: **anesthesia is a controllable suppression spectrum over 13DD fine-layers, not a simple "switching off" of consciousness**. This note develops that proposition, using anesthesia as a probe to see into the structure of 13DD.

2. Definitions

2.1 The four fine-layers of 13DD

From bottom to top, 13DD has four fine-layers:

13DD-d: event-marking. The thinnest subject-side marker, marking without construction. It does not involve content integration or subject embedding. The "event" here does not even have object-side boundaries; it is simply **the reception of a pure perturbation**—not "an apple fell" but "the system's state underwent a distinguishable deviation." 13DD-d differs from 10DD perceptual layer in exactly this sense: 10DD does object recognition (the *what*), 13DD-d does subject-side marking (the *that*). Corresponds to the mark (marking) mode of chisel.

13DD-c: say-no. Incremental refusal or selection regarding what is occurring. Say-no is not mere aversive reaction; it is the subject's active discrimination of the information flow—this I take, that I do not. Corresponds to the add (addition, incremental construction) mode.

13DD-b: fear-of-death. Root protection of self against dissolution. 13DD-b is not simply fear of some threat but the drive for preservation of existence itself—this position must not be lost, must not disappear, must not be erased. It is the physiological and structural protection mechanism for the sense of being. Corresponds to the multiply (existence × dissolution, emergent combination) mode.

13DD-a: asymptotic complete self. The integrative scheduling mode that emerges when event-marking, say-no, and fear-of-death operate in coordination. As an AND-synthesis, 13DD-a is the emergence from the simultaneous presence of the three lower fine-layers—the more complete the coordination of d, c, and b, the closer 13DD-a is to "complete self." Corresponds to the AND (full-identity synthesis) mode.

The ordering of these four fine-layers is not arbitrary. They correspond to the four operational modes of chisel (mark / add / multiply / AND), aligned with the every-round four-step pattern established in SAE Life-Death-Consciousness Paper VI (DOI 10.5281/zenodo.19528781). In SAE's sixteen-layer DD sequence, every four layers form one round, and each round internally unfolds in mark/add/multiply/AND order. 13DD sits at the first layer of the fourth round (13-16DD, the subject-level round); its four fine-layers are the within-layer recursion of the every-round four-step pattern.

Connection to Bio Note 7's three functional positions. The three functional positions given by Bio Note 7—narrative, negation, conflict monitoring—constitute the coarse-grained functional map of 13DD. The four fine-layers of this note are a **resolution increase** of the same structure, not a revision:

The narrative position, under the layered view, unfolds as a cross-layer structure from 13DD-d (the thinnest event-marking) to 13DD-a (complete autobiographical narration). Narrative is not a single function but a continuum across fine-layers—the thinnest narrative is "something is occurring," the most complete narrative is "I lived through this event."

The negation position maps directly to 13DD-c (say-no as baseline refusal). Bio Note 7's "negation" and this note's "say-no" are the same mechanism under different resolutions.

The conflict-monitoring position is distributed between 13DD-b and 13DD-a. 13DD-b as protection-against-dissolution involves existence-level baseline conflict monitoring ("does this position still exist?"); 13DD-a as coordinative scheduling involves multi-source integration conflict monitoring ("are these sources mutually consistent?"). Bio Note 7's "conflict monitoring" resolves, under this note's layered view, into cooperation between two fine-layers.

The three functional positions give the coarse-grained functional map of 13DD; the four fine-layers give the internal layered structure. At low resolution one sees the functional map; at high resolution one sees the layered structure; both are correct at their respective resolutions. This is structurally the same move Bio Note 9 made for Anth-1—different descriptions at different resolutions can coexist consistently.

2.2 The asymptotic structure and functional substructure of 13DD-a

13DD-a is permanently > 0 . By SAE remainder conservation (ZFC_p first law), no construct reaches full closure, and 13DD-a as an AND-synthesis is no exception. The more complete the coordination of d, c, and b, the closer 13DD-a approaches "complete self," but it never reaches full closure—the remainder always persists. "Complete self" is the asymptotic limit, not a state actually attained.

To emphasize: **13DD-a as asymptotic limit is not a weakening of the concept "complete self"; it is a necessary consequence of remainder conservation at the fine-layer level.** All constructs fall under the ZFC_p first law, and 13DD-a is no exception. The asymptotic character makes 13DD-a *more* productive structurally, not less, compared with a hypothetical "closable complete self," because it preserves the space for the remainder to continue flowing.

The positive consequence of this structural limitation: **precisely because 13DD-a is never complete, the subject must seek meaning at 14DD.** If 13DD-a could close, meaning-seeking would be unnecessary. Meaning-seeking is the structural driving force of 13DD-a's non-closure, not a choice. This turns the 13DD-to-14DD emergence path from external description into internal necessity—the subject does not "choose" to seek meaning; the incompleteness of 13DD-a *forces* the subject to seek meaning, to process the continuously emerging remainder.

13DD-a has at least four core subfunctions:

Encoding-side ownership metatag. Attaches a "this is mine" tag to incoming experience. The metatag does not rewrite the content itself; it attaches an ownership label to the content. Tag strength is a gradient, not an on-off switch—strong metatags correspond to stable autobiographical narrative memories, weak metatags correspond to implicit or non-narrative memory traces (see §5.4.2).

Retrieval-side gatekeeping. Regulates which metatagged memories enter the current narrative layer. Not all tagged memories automatically enter narration; 13DD-a performs a second filtering at retrieval—does this memory currently enter the ongoing narrative? Selective failure of retrieval-side gatekeeping is the core mechanism of dissociative amnesia (see §5.4.1).

Repeated re-acknowledgment. Maintains the "mine" status of memories against temporal decay. A metatagged memory does not automatically maintain ownership intensity forever; it needs to be periodically "re-acknowledged" by 13DD-a—re-confirmed as "this is mine." Weakened re-acknowledgment causes memories to "feel more distant"; excessive re-acknowledgment manifests as compulsive rumination (see §5.4.4).

First-person embedding in scene reconstruction. Embeds "I-was-there" when reconstructing past scenes. Without subject embedding, memories can be retrieved as "such an event occurred" but lack the first-person position of "I was living through it." Failure of subject embedding is the core mechanism of depersonalization (see §5.4.3).

These four subfunctions can deviate independently, producing distinct clinical presentations. 13DD-a, as the coordinated operation of these four subfunctions, **may be a scheduling mode rather than an activation level of any single region**. This explains why direct stimulation of candidate regions (such as medial prefrontal tDCS) fails to directly enhance self-referential processing—if 13DD-a is a scheduling mode, enhancing activation in any single region does not alter the scheduling relationship.

2.3 Directional constraint on the ownership metatag

This section establishes a key conceptual boundary to prevent 13DD-a's ownership metatag from being misread as "13DD-a controls 11DD storage."

The ownership metatag of 13DD-a does not rewrite 11DD storage; it only gates whether traces can enter the autobiographical narrative channel. This strictly respects the directional constraint established in Bio Note 9: "Higher-layer refusal is 'I do not receive,' not 'you may not send.'"

Concretely: 11DD is the memory storage layer and contains many forms of memory traces—procedural (riding a bicycle), semantic (a factual item), emotional (conditioned affective responses), and implicit (unconsciously held but behaviorally effective learning). The formation of these traces does not depend on 13DD-a's metatag; they can form and persist in lower-level systems independently. 13DD-a's metatag is an **additional layer** that hangs "this is my experience" ownership labels onto certain traces, allowing those traces to enter the autobiographical narrative channel.

This gives three categories of memory a clean architectural location:

Autobiographical narrative memory with metatags. 13DD-a's encoding-side metatag is sufficient; the memory enters the stable autobiographical narrative layer. "I remember what happened yesterday" falls into this category.

Non-metatagged implicit memory. Traces formed by 11DD and 12DD's own operations, without ownership metatag, corresponding to procedural, semantic, and partial emotional memory. This type can be retrieved and used, but without the first-person "this is my experience" feeling.

Non-narrative traumatic imprints. A special case: under extreme threat, 13DD-b's high-frequency discharge compresses 13DD-a into a low-coherence state, and low-coherence 13DD-a produces **tags of intensity insufficient for narrativization but sufficient for imprint persistence**. These weak-tagged traces manifest through non-narrative channels—bodily flashbacks, unnamed fear, and trigger-based reactivation, constituting the core symptoms of PTSD (see §5.4.2).

The third category needs special explanation because it may look like a counterexample to the directional constraint—traumatic imprints appear to be "lower-layer overreach." But the directional constraint is not violated: 13DD-b does not tag on its own; it only **indirectly weakens** 13DD-a's operational quality by seizing cross-regional coherence resources. The tagging

privilege remains with 13DD-a; only the operating conditions have deteriorated. The mechanism is developed in §5.4.2.

2.4 The independent dynamics of suppression and recovery

The suppression and recovery dynamics of anesthesia are not in simple reverse-order relationship. This is one of the most important methodological distinctions of this note.

Suppression dynamics are driven by drug molecular dynamics and are relatively deterministic top-down. But this is **not** the result of drug-directed closure; it is the manifestation of a **vulnerability ladder**. Higher fine-layers require stricter cross-regional coherence in the neural network to maintain themselves; under globally uniform chemical inhibition, the most fragile, most energy-demanding, most integration-dependent top-level functions collapse first. 13DD-a collapses first, 13DD-d last—not because the drug "turns things off from the top down," but because higher functions are more sensitive to disturbance. This reformulation converts "top-down" from a description into a mechanism (see Theorem One).

Recovery dynamics are driven by neural network reconstruction and operate differently at two scales, which must be distinguished:

Fine-layer recovery (seconds to minutes scale). 9DD and 10DD arousal come back first, then 13DD-b threat response, then 13DD-a self-integration. This is the core hypothesis: fine-layer recovery follows a bottom-up order, the reverse direction of the suppression vulnerability ladder.

Cognitive bandwidth recovery (minutes to hours scale). After all fine-layers have come online, different cognitive functions reach baseline efficiency at different times. This scale of recovery **does not** follow the fine-layer order—existing literature shows that executive function can recover earlier than processing speed, which contradicts the "lower-level-first recovery" hypothesis if the two scales were conflated.

Why this apparent contradiction? Because the two scales measure different things. **Fine-layer recovery** asks "is this fine-layer in place?"; **cognitive bandwidth recovery** asks "after this fine-layer is online, how efficient is the supporting neural network?" Mainstream research methods (behavioral tests, self-report, cognitive tasks) all require the patient to understand instructions and cooperate with tasks—this itself requires 13DD-a to be online. So mainstream studies measure post-13DD-a-online bandwidth recovery, not pre-13DD-a-online fine-layer recovery (see Theorem Four).

These two scales must not be conflated. The predictions of this note, especially Prediction Three, are strictly confined to the fine-layer recovery scale.

2.5 The term "fine-layer"

This note repeatedly uses "fine-layer" as the term for within-DD-layer structure. Each DD layer has an internal four-partition structure, ordered by the four operational modes of chisel

(mark/add/multiply/AND), and this may recursively extend (each fine-layer further divides into four, and so on).

This note develops only the fine-layer structure of 13DD; the fine-layer structures of other DD layers are left for future notes. But the fine-layer architecture is not specific to 13DD—it is the within-layer manifestation of SAE's fractal nature. The entire 1DD-to-16DD sequence is itself a four-round fractal (physics / life / cognition / subject), with four layers per round, each layer internally divided into four, and so on recursively. A complete fractal geometry unfolding deserves systematization in a future Method VI revision (see §5.7).

3. Core Theorems

This section provides six core theorems that form the structural skeleton of this note. Each theorem is falsifiable, compatible with existing SAE architecture, and corresponds to specific clinical phenomena in the §5 rays.

3.1 Theorem One: the suppression direction theorem

Increasing anesthetic dose progressively suppresses DD layers in a top-down order.

Key clarification: The "top-down" ordering is not the result of drug-directed action on higher fine-layers. It is the emergence of a **vulnerability ladder**. Under global inhibition, different layers have different sensitivities to disturbance; 13DD-a as AND-synthesis of all subfunctions requires the strictest cross-regional coherence and collapses first under global inhibition; 13DD-d as the thinnest event-marking requires the least neural integration and collapses last. This mechanism explains why entirely different molecular mechanisms (GABA potentiation, NMDA blockade, potassium channel activation) all produce apparently "top-down" suppression—the drugs are not directional, but the functions have a vulnerability ladder.

Corollaries:

- Different anesthetic depths correspond to different stopping layers. Light anesthesia suppresses 13DD-a, leaving 13DD-b/c/d intact; deep anesthesia continues into 13DD-b and 13DD-c; very deep anesthesia reaches 13DD-d and below 11DD.
- Anesthetic overdose is not "some layer reaches zero"; it damages the substrate itself (cardiovascular and respiratory collapse). This corollary is developed in Theorem Six.
- Different drugs have different suppression paths—although the general order is similar (determined by the vulnerability ladder), each drug has preferred fine-layers (candidate signatures, see §5.1).

3.2 Theorem Two: the fine-layer independence theorem

The four fine-layers of the same DD layer can be independently closed or fail; synchrony is not required.

This is the direct manifestation of fine-layer independence and the experimental foundation of the entire architecture of this note. If the four fine-layers must operate synchronously, they are not truly independent layers but different facets of a single integrated function. Fine-layer independence is what allows clinical natural experiments to "see" the separate operation of different fine-layers.

Corollaries:

- **Dental sedation:** closes 13DD-a's encoding-side ownership metatag function, preserves 13DD-b/c/d and retrieval-side historical memory. The patient can converse, say no, and respond to pain, but has no memory afterward. This is a typical example of independent failure of 13DD-a's encoding side.
- **Ketamine dissociative experience:** most prominently damages 13DD-a's AND-synthesis while preserving or distorting activity in the other fine-layers. The patient reports "I was not in my body"—13DD-a's integration fails, 13DD-d's event-marking remains (so "something is happening" is still felt), 13DD-c's say-no remains (so strong affective reactions occur), 13DD-b's fear-of-death is present but unstable.
- **Dissociative amnesia:** selective failure of 13DD-a's retrieval-side gatekeeping, not an encoding failure. Traumatic memories carry metatags (so there are implicit memories and autonomic responses), but the retrieval side closes the path for specific content. This is evidence for encoding-side and retrieval-side independence of 13DD-a.
- **HSAM/SDAM:** multidimensional profile deviation of 13DD-a's multiple subfunctions. In HSAM, all subfunctions deviate in the same direction (enhanced); in SDAM, all in the opposite direction (weakened). Neither is a two-pole contrast along a single dimension; both are multidimensional profile combinations (see §5.4).

3.3 Theorem Three: the independent dynamics of suppression and recovery

Anesthetic suppression and recovery are two independent structural processes and cannot be derived from each other.

Suppression dynamics are driven by drug-molecular mechanisms; different drugs present testable candidate fine-layer signatures (see §5.1). Recovery dynamics are driven by neural network reconstruction; their order and timescales are independent of the suppression order—the two are not symmetric inverse processes.

The two scales must not be conflated:

Fine-layer recovery: bottom-up (hypothesis). 9DD/10DD come first, then 13DD-b, then 13DD-a last.

Cognitive bandwidth recovery: does not follow strict reverse order (literature evidence). Executive function can recover earlier than processing speed—this is not a counterexample to the fine-layer hypothesis but a phenomenon at a different scale.

The methodological significance of this theorem: it prohibits using cognitive bandwidth recovery data as evidence of fine-layer recovery. The many literature results showing "executive function recovers earlier / processing speed recovers later" are all measured after 13DD-a is already online and cannot be used to falsify or validate the fine-layer recovery order (see Theorem Four and Prediction Three).

3.4 Theorem Four: the 13DD-a dependency blind spot in consciousness recovery research

The main measurement methods of current consciousness recovery research (behavioral tests, self-report, cognitive tasks) all require the subject to understand instructions and maintain tasks, which itself requires 13DD-a to be online.

This is a **constitutive measurement blind spot**, not a criticism of existing research.

Corollaries:

- These studies constitutively cannot measure the earlier recovery of 13DD-d/c/b—by the time the patient can complete the test, 13DD-a is already online.
- All "early cognitive recovery" data in the literature have already occurred after 13DD-a came online. "Executive function recovered within minutes of waking" should strictly be stated as "within minutes of 13DD-a coming online, executive function reached the test threshold."
- Measuring fine-layer recovery order requires measurement methods that do not depend on task cooperation—evoked potentials, autonomic responses, behavioral observation, and other indicators that do not require the subject to understand instructions (Prediction Three develops this design).

This theorem is not meant to negate mainstream research but to identify the structural limitations of existing measurement tools and motivate a new research paradigm.

3.5 Theorem Five: the asymptotic tag and narrative-channel dependence theorem

11DD's autobiographical narrative layer encoding depends on 13DD-a's encoding-side ownership metatag. But tag strength is a gradient, permanently > 0 .

This theorem is the core formulation of the ownership metatag architecture. It maintains two things simultaneously: first, 13DD-a's tagging privilege monopoly (only 13DD-a can tag); second, the continuity of tag strength (by remainder conservation, permanently > 0).

Corollaries:

- **Strong tags** enter the stable autobiographical narrative layer. Normal "I remember what I did yesterday" falls here.
- **Moderate tags** can be retrieved but "feel not mine." Depersonalization clinical reports fall here—memories are present but subject embedding is weak.
- **Weak tags** manifest only through implicit channels. Traumatic flashbacks and body memories fall here—narrativization cannot happen, but traces leak through non-narrative channels.
- **Very weak tags** are clinically unobservable, but by remainder conservation still exist and may surface under specific triggers.

The "post-emergence amnesia window" is the result of 13DD-a's encoding-side tag strength being very low but nonzero. Events are recorded at the behavioral level (the patient did speak and follow instructions) and stored in very weak form, but not sufficient to enter the stable autobiographical narrative layer (the patient has no memory afterward). This phenomenon is discussed in detail in §5.3.2.

3.6 Theorem Six: the substrate integrity theorem

The structural condition for life-death reversibility is substrate integrity, not whether any fine-layer reaches zero.

This is the rigorous formulation of the anesthesia-death isomorphism in this note. It rests on two facts:

First, any DD fine-layer's operation, once it occurs, leaves remainder that does not reach zero by SAE remainder conservation. Whether the operation itself continues depends on substrate support: when the substrate is intact, operation is recoverable; when the substrate dissolves, the individual's operation stops. This is the direct constraint of ZFC_p first law on fine-layer dynamics, but it does not claim that "operation remains online after substrate dissolution"—operation depends on substrate; remainder conservation applies to traces produced by operation, not to the continuation of operation itself.

Second, the relationship between operation and substrate: operation needs a substrate to carry it. Substrate dissolves, operation stops—but remainder produced by past operation, by conservation, does not reach zero; it can only transfer.

Corollaries:

- Anesthesia is reversible: the substrate (neural architecture) is not destroyed; after drug metabolism, function re-emerges. Under anesthesia, 13DD-a's operation is temporarily inhibited, but the substrate is intact and operation can recover.

- Death is irreversible: the substrate dissolves; this individual's 13DD-a operation loses its carrier. But the remainder produced by 13DD-a's past operation, by conservation, does not reach zero; it can only transfer.
- The distinction between reversible and irreversible lies in **substrate state**, not in 13DD-a's operational level.

Soul-theory demarcation. SAE's remainder conservation is a structural theorem; **it is not a substantive claim about the form of post-propagation remainder**. SAE does not state whether remainder propagates in the form of a "soul," whether individual identity is preserved, or whether consciousness continuity is involved. These topics belong to the discussion space of soul studies, theology, and specific cultural frameworks; they lie outside the scope of structural philosophy. This note provides only structural boundaries and does not overreach.

This demarcation is critically important—it keeps SAE from falling into either materialism (consciousness = neural activity; neural cessation = consciousness reaches zero) or idealism and soul theory (consciousness is independent of matter; post-death it continues as "soul"). SAE says only: remainder conservation is a theorem; the specific mode of remainder propagation is a posterior question; until propagation modes are sufficiently verified by posterior evidence, SAE maintains "not-yet-known" and makes no commitments.

Readers from different cultural and personal worldviews may bring their own belief systems to interpret the remainder propagation question—SAE does not decide for them. This is consistent with the posture of Methodology IX's eighth open question (the relation between non and consciousness).

4. Subject Conditions

Researchers studying anesthetic mechanisms or using SAE tools to analyze anesthetic phenomena should satisfy the following subject conditions. These are not additional requirements but methodological consistency guarantees.

4.1 Do not treat behavioral indicators as structural indicators. Being able to converse does not mean 13DD-a is online; being able to respond does not mean the entire 13DD is online. Clinically observed behavioral responses can come from partial operation of any fine-layer. A post-emergence patient may speak when 13DD-a is not fully online (speech emerging from automated language modules under low-coherence 13DD-a) or may say-no when 13DD-c is online but 13DD-a is not ("I don't want this, but I don't know who 'I' is"). Behavior and structure are not directly equivalent.

4.2 Do not describe multi-layer phenomena with single-layer language. "Consciousness" is the product of multiple layers and multiple fine-layers operating simultaneously, not a single switch. When we say "the patient lost consciousness," this statement itself is coarse-grained—what is

actually lost, across which layers, in which functions? Under the fine-layer view, "losing consciousness" is not a single event but a structural layered-suppression process. Single-layer language systematically obscures the real structure of anesthesia.

4.3 Distinguish "closure of generation in a fine-layer," "cutting of cross-layer access pathways," and "selective failure of subfunctions within a layer". These three can occur independently. Generation closure of a fine-layer (e.g., complete closure of 13DD-d) is one scenario; cross-layer pathway cutting (e.g., 11DD can store but 13DD-a cannot retrieve) is another; within-layer subfunction selective failure (e.g., 13DD-a encoding side closed but retrieval side open) is a third. These three mechanisms produce different clinical phenomena and must not be confused.

4.4 Distinguish "fine-layer recovery" and "cognitive bandwidth recovery"; do not conflate the two scales. Fine-layer recovery happens at the seconds-to-minutes scale, mainly before or at 13DD-a coming online; cognitive bandwidth recovery happens at the minutes-to-hours scale, mainly after 13DD-a is online. Treating cognitive bandwidth data as evidence for fine-layer recovery is a common manifestation of the blind spot identified in Theorem Four.

4.5 Recognize the 13DD-a dependency blind spot of existing measurement methods; do not equate "cannot be measured" with "does not exist". An early evoked potential response during emergence does not mean "consciousness hasn't returned"; it just cannot be measured, because standard consciousness tests themselves require 13DD-a to be online. Shifting to measurement methods that do not depend on task cooperation is what allows 13DD-d/c/b early recovery to be seen (Prediction Three develops this).

4.6 Do not use "13DD-a reaches zero" or "fine-layer closure" as the definition of death. This would violate remainder conservation—any fine-layer's operation or remainder, by the SAE structural theorem, is permanently > 0 . The structure of death is **irreversible substrate dissolution**, not functional zeroing of any specific layer (Theorem Six). Conflating clinical brain-death criteria with "some layer reaches zero" produces structural misreadings of the life-death boundary.

These six subject conditions are applied repeatedly throughout the note. Violating any of them causes the analysis to drift from the SAE architectural consistency.

5. Rays

This section develops seven rays, each testing the core theorems from a different clinical direction. Rays are not an independent list of propositions but projections of the core theorems onto specific phenomena—if the theorems hold, these rays should display corresponding structural correspondences.

5.1 Ray One: fine-layer preferences of different anesthetic agents

Proposition grading:

Hard structure (literature-supported): Different agents do not differ simply in "anesthetic depth"; they differ in fine-layer action patterns. This has been repeatedly confirmed in anesthesiology and neuropharmacology literature—agents like propofol, ketamine, benzodiazepines, and dexmedetomidine produce significantly different EEG signatures and clinical phenomenologies that cannot be reduced to different doses of the same "depth."

Candidate mappings (proposed in this note): Which fine-layers or subfunctions each agent primarily affects. The following mappings are candidate signatures, not final conclusions, and require experimental validation.

The following discusses six major anesthetic classes.

Propofol (GABA-A potentiation)

Propofol is the most widely used intravenous anesthetic, producing broad-spectrum inhibition by enhancing GABA-A receptor activity. A clinical fact: **propofol's amnestic effects appear at sub-hypnotic doses.** Sub-hypnotic doses of propofol can seriously disrupt memory encoding—the patient can still converse and respond to commands, but has no autobiographical memory of that period afterward.

Candidate signature: **broad-spectrum top-down suppression, but particularly sensitive to 13DD-a's encoding-side ownership metatag.** At low doses, the encoding-side metatag closes first (producing the amnesic effect while the patient can still converse); at moderate doses, suppression extends to 13DD-a's integration (the patient loses consciousness); at high doses, suppression proceeds into 13DD-b/c/d and below.

Propofol's broad-spectrum character makes it the clearest demonstration of the "vulnerability ladder"—the same drug, with increasing dose, progressively suppresses fine-layers in a relatively stable order.

Ketamine (NMDA blockade)

Ketamine is clinically called a "dissociative anesthetic" because it produces states fundamentally different from GABAergic agents. Patients may report: "I saw my body lying there"; "Everything was real but I was not in it"; "I had strong emotions and sensations, but I wasn't sure whose they were."

Candidate signature: **in the candidate mapping, most prominently damages 13DD-a's AND-synthesis while preserving or distorting 13DD-b/c/d activity.** Ketamine does not suppress broadly along the vulnerability ladder; it specifically cuts 13DD-a's integrative capacity, letting other fine-layers continue operating independently without upper-layer integration. Specific manifestations:

Dissociative experience ("I'm not in my body"): 13DD-a integration fails, 13DD-d event-marking persists, so the feeling "something is occurring" is still there, only without "I" as subject.

Vivid hallucinations through a multi-layer mechanism: The vividness and externality of ketamine hallucinations do not have a single mechanism; they are the product of failures in coordination across multiple fine-layers. Full chain:

1. 13DD-a's integrative mechanism fails; upper-layer regulation of 12DD goes offline
2. 12DD prediction system, freed from constraint, generates images and narrative fragments wildly
3. 13DD-c (say-no) continues operating, producing strong affective responses (ecstasy, fear, revulsion) to these ownerless images
4. The strong affective response itself is experienced as "these images are really there"
5. But without 13DD-a's ownership metatag, the images cannot be tagged as "mine"
6. Result: hallucinations that are both extremely vivid (from 13DD-c's strong response) and externally located (from lack of ownership tag)

This chain shows the specific mechanism of multi-fine-layer coordination failure under ketamine—13DD-a crashing does not directly produce hallucinations; hallucinations are the combined product of 12DD running wild after 13DD-a crashes plus 13DD-c's ownerless affective response.

Partial post-hoc memory: weak tags produced in moments of 13DD-a's intermittent recovery; these weak-tagged traces partially enter the narrative layer, forming fragmentary post-hoc memories—"I remember floating and looking down, then it went dark."

EEG signature markedly different from GABAergic agents (confirmed by literature): gamma activity persists or even increases, rather than the alpha/delta dominance of GABAergic agents. This is consistent with the structural prediction that 13DD-a integration fails while 13DD-d/c/b remain active.

Ketamine is the **strongest clinical evidence for Theorem Two (fine-layer independence)**—the same patient, at the same moment, shows markedly failed 13DD-a with 13DD-b/c/d still highly active. If the four fine-layers could not operate independently, this dissociative experience should not occur.

Inhalational agents (isoflurane, sevoflurane)

Inhalational anesthetics act through multiple molecular mechanisms (GABA potentiation, NMDA inhibition, potassium channel activation) producing broad-spectrum inhibition. Clinically they resemble propofol, progressively suppressing fine-layers with dose.

Candidate signature: broad-spectrum, with increasing dose progressively suppressing 14DD, then 13DD-a, then b/c/d, then 11DD/12DD. The suppression order largely follows the vulnerability

ladder.

One clinical specificity needs noting: **sevoflurane emergence agitation is high in children**, a hard clinical fact with significant difference from other inhalational agents or propofol. SAE explanation: sevoflurane may have a specific delaying effect on 13DD-a's recovery (relative to other GABAergic or opioid agents). Other fine-layers recover faster; 13DD-a remains offline, producing a window with 13DD-b threat response online but 13DD-a integration not yet achieved, manifested as agitation (see §5.3.1). This explanation needs further neurophysiological verification.

Benzodiazepines (midazolam)

Midazolam is widely used for preoperative sedation and moderate sedation. The clinical phenomenon is highly specific: **strong anxiolysis + strong amnesia + weak consciousness suppression**. Literature describes midazolam with exactly this phrasing: "responsiveness without durable autobiographical encoding"—responsiveness is present, but no stable autobiographical encoding.

Candidate signature:

- Primarily suppresses 13DD-a's encoding-side ownership metatag
- Also partially suppresses 13DD-b (anxiolysis effect comes from reduced 13DD-b fear-of-death response)
- Preserves 13DD-c say-no and 13DD-d event-marking
- Clinical phenomenon: under sedation, the patient can converse, cooperate, and say-no (refusing something), but has no memory afterward

Midazolam is the main drug for dental sedation and endoscopy sedation, precisely because of this specific profile—the patient can cooperate with the procedure but will not resist next time's treatment due to traumatic memory. In the SAE fine-layer view, midazolam's clinical profile maps most naturally to a prominent suppression of the encoding-side metatag function, without requiring the entire 13DD to be fully closed. This remains a candidate mapping; specific mechanisms await experimental validation.

Opioids (fentanyl, morphine)

Opioids are not strictly anesthetics but analgesics, though often used as adjuncts in anesthesia. Their primary action is on 10DD's pain subchannel (the affective and meaning dimensions of pain), not directly on the 13DD layer.

Candidate signature: **primarily cuts 10DD's pain subchannel; does not directly affect 13DD fine-layers**. This explains why opioid monotherapy cannot produce complete anesthesia—the patient may feel no pain but is still conscious, can converse, and can form memories. Opioids must be combined with agents that suppress 13DD to reach surgical anesthesia.

This is itself an important structural evidence: **pain perception can be adjusted independently of self-awareness**. This supports 13DD and 10DD's inter-layer independence—when certain 10DD subchannels are closed, 13DD can remain fully operational.

Dexmedetomidine ($\alpha 2$ agonist)

Dexmedetomidine produces a very special state clinically called "arousable sedation." The patient appears to be in deep sleep, but slight stimulation can wake them; once awake, they can converse rationally, then quickly return to sleep. The EEG signature resembles NREM slow-wave sleep, not typical anesthetic EEG.

Candidate signature:

- Preserves 13DD-b threat response (literature-supported: amygdala response preserved)
- Closes 13DD-a integration
- Produces a state that can be aroused by stimulus

This is strong clinical support for fine-layer independence, though the specific signature remains a candidate mapping. Dexmedetomidine's "arousable" character directly supports this reading: 13DD-b is active enough that stimulation triggers a wake response; 13DD-a is in a low-coherence state, so at baseline the patient appears to be asleep; once external disturbance is sufficient, 13DD-a rapidly reorganizes and the patient can briefly complete conversation; disturbance removed, 13DD-a returns to low coherence.

This fine-tunability gives dexmedetomidine unique value in ICU sedation and post-operative monitoring.

Summary

The candidate signatures of six drug classes form a spectrum of fine-layer action modes rather than a simple "anesthetic depth" dimension. If these candidate signatures are experimentally confirmed, they will constitute a new dimension of anesthetic drug classification—not by chemical structure or receptor type, but by fine-layer action mode. Prediction Two gives a specific experimental protocol.

5.2 Ray Two: reinterpretation of anesthetic depth monitoring

What do BIS and similar single-scalar monitors actually measure?

Current mainstream monitors (BIS, entropy index, SEDLine, etc.) compress anesthetic depth into a single 0-100 scalar. BIS value 100 indicates complete wakefulness; 0 indicates complete cortical electrical suppression. Clinically 40-60 is the "surgery-suitable" depth.

But BIS is unreliable under certain drugs, a well-known problem in anesthesiology. Under ketamine, BIS may show "lighter" values when the patient is actually unarousable. Under

dexmedetomidine, BIS values mismatch consciousness state—BIS shows wake levels but the patient is deeply sedated. These failure modes have a clear reading in the SAE architecture:

BIS is closer to a coarse-grained proxy of 13DD-a integration quality, rather than a single readout of the entire consciousness hierarchy. The BIS algorithm depends primarily on frontal EEG features, which are sensitive to the collapse of cortical cross-regional integration—and this is exactly what 13DD-a's AND-synthesis depends on. So BIS performs well under GABAergic broad-spectrum suppression (because suppression follows the vulnerability ladder top-down, and 13DD-a integration quality is the most sensitive indicator).

But BIS fails under ketamine and dexmedetomidine precisely because these two agents' signatures do not follow the vulnerability ladder:

Ketamine in the candidate signature most prominently damages 13DD-a while preserving or increasing activity in other fine-layers—so overall cortical activity does not decrease (BIS shows lighter values), but 13DD-a integration has collapsed (patient actually unarousable or unable to report coherent experience). BIS measures activity level; ketamine changes integration mode; the two are misaligned.

Dexmedetomidine preserves 13DD-b threat response, so some cortical regions remain actively responsive (BIS shows wake values), but 13DD-a integration is down-regulated to sleep-like states. Again activity and integration are misaligned.

These failure modes show that **a single scalar cannot simultaneously distinguish activity level and integration quality.** What is truly needed is multimodal synchronous measurement—one dimension for activity level, one for integration quality, possibly additional dimensions for which subfunctions are online (encoding side, retrieval side, etc.). This is one motivation for Prediction Three.

Future monitoring design direction: multimodal synchronous tracking of candidate indicators for the four fine-layers, distinguishing activity, integration, and function, without compression into a single number. This is not a replacement for BIS but a repositioning of BIS's role—BIS remains useful as a coarse-grained proxy for 13DD-a integration quality, but must be supported by a more complete measurement system.

5.3 Ray Three: reinterpretation of emergence phenomena

Emergence is the best clinical window for observing the separate recovery of 13DD fine-layers. This section develops four typical emergence phenomena, each receiving a new structural reading under the fine-layer view.

5.3.1 Emergence agitation

Emergence agitation (emergence delirium) is especially high in children, particularly after sevoflurane anesthesia. Clinical manifestation: the patient wakes but is agitated, crying, or

flailing limbs; cannot be comforted, does not recognize family, looks "not like themselves."
Usually resolves spontaneously in 5-20 minutes.

SAE reading: **13DD-b (threat response) recovers earlier than 13DD-a (integration).**

Neurophysiological research on emergence agitation in the literature shows consistent direction—threat-response circuits (amygdala) recover first, high-level integration (prefrontal DMN) later. This time gap produces a special window: the patient has "I must not disappear" physiological protection (13DD-b driving defensive responses online) but no "who am I, where am I" situational integration (13DD-a not yet online).

The reading of agitation becomes precise: it is not that the patient "has not fully awakened," but that certain fine-layers have awakened while the key integrative fine-layer has not. The patient's agitation is not random chaotic movement but **13DD-b operating independently without integration**—threat response to environmental unfamiliarity without the integrative positioning of "this is post-surgery, mom is here."

High emergence agitation in children SAE reading: children's 13DD-a is in the "self-to-be" phase—complete self is not yet fully stabilized (13DD-a as AND-synthesis needs time and experience to gradually stabilize). 13DD-a recovery after anesthesia requires more time than in adults, while 13DD-b threat response is equally or more active in children, so the window is longer and more intense.

Highest agitation with sevoflurane SAE reading: sevoflurane may have a specific delaying effect on 13DD-a recovery (relative to other GABAergic or opioid agents). Sevoflurane's molecular mechanism involves multiple channels, some of which may have delayed effects during metabolic elimination on the cross-regional coherence that 13DD-a depends on. This hypothesis needs further neurophysiological research for confirmation.

5.3.2 Post-emergence amnesia window

Another common phenomenon: in the first 15-30 minutes after emergence, the patient may be able to cooperate, converse, and even answer simple questions, but has no memory of this period afterward—family sees the patient responding and speaking; the patient insists "I never said those things."

SAE reading: **13DD-d/c/b have recovered, but during the period when 13DD-a encoding-side metatag function is very weak, events are behaviorally recorded but tags are too weak.** The patient can converse because 13DD-c say-no and 13DD-d event-marking are online, plus 12DD/11DD language circuits and procedural responses have recovered; but 13DD-a encoding-side ownership metatag strength is too low to attach stable "this is my experience" labels to these events. Result: behavior occurred (at 12DD and lower levels), but narrativization did not occur (13DD-a encoding-side tag strength insufficient).

Literature evidence: ReCCognition-type studies (and anesthesia wake-up studies) find that among 49 patients, 23 can recall emergence fragments, 11 show only implicit memory (unable to

actively recall but showing latent responses to events from that period), and 15 have no explicit recall at all. This is exactly the tag strength continuous spectrum—some patients' 13DD-a encoding-side tags reach narrativization-capable strength during emergence (they can recall afterward), some reach only implicit levels (traces without narration), some cannot reach even implicit levels (by remainder conservation still have weaker traces, but clinically unobservable).

This is the clearest clinical demonstration of Theorem Five (the asymptotic tag and narrative-channel dependence theorem): tag strength is a continuous gradient, not a 0/1 switch.

5.3.3 Postoperative cognitive dysfunction (PND/POCD)

Elderly patients after major surgery may show persistent cognitive problems for days to weeks—decline in memory, judgment, and self-integration. The current anesthesiology consensus term is PND (perioperative neurocognitive disorders), covering the spectrum from acute post-emergence delirium to delayed cognitive decline.

SAE reading: **elderly 13DD-a delayed recovery, persisting for days to weeks.** PND is not broad-spectrum brain injury but delayed restart of 13DD-a. Mechanistic background: elderly patients have lower cognitive reserve; cross-regional coherence recovery is slower; 13DD-a, this fine-layer requiring the strictest coherence, is most affected. Some patients fully recover within days, some need weeks, some even retain minor permanent impairment.

This reading is consistent with clinical observation—the main symptoms of PND (memory difficulty, judgment confusion, "feeling not like myself") all point to various subfunctions of 13DD-a. If PND were broad-spectrum brain injury, symptoms should be broader; if PND were a single-function decline, symptoms should be more focused. The actual symptom profile is exactly the multi-subfunction co-directional weakening of 13DD-a, which is the expectation of delayed 13DD-a recovery.

Vulnerability basis: prior cognitive reserve determines PND risk. This has direct meaning in the SAE framework—cognitive reserve is the redundancy and recoverability of 13DD-a and its supporting network. Low reserve, slow recovery; high reserve, fast recovery.

5.3.4 Delayed emergence

A small number of patients after anesthesia show normal vital signs but slow consciousness recovery—"eyes open but the person is not there." This state can persist for hours to days.

SAE reading: **13DD-d/c/b have recovered but 13DD-a persistently does not come online.** Eyes open means 9DD/10DD have recovered (arousal mechanism); but 13DD-a integration has not formed, so the patient cannot engage in meaningful conversation or orientation. "Arousal" is not equivalent to "integration"—another direct demonstration of fine-layer independence.

This phenomenon is clinically sometimes classified as "delayed emergence syndrome," with unclear mechanism. The SAE reading gives a specific structural location: the problem is at 13DD-a restart, not at lower-layer failure. This suggests clinical management should focus on

supporting the rebuilding of 13DD-a's cross-regional coherence—environmental familiarization, family voices, gradual stimulation all are factors helping 13DD-a reintegrate.

5.3.5 Interpreting the ReCCognition study: constitutive blind spot, not literature error

The ReCCognition study (Mashour et al.) is the most detailed cognitive measurement study of anesthetic recovery in recent years. An important finding: **executive function recovers earlier than processing speed**. This looks opposite to the "bottom-up recovery" hypothesis—by common sense, basic functions (processing speed) should recover first, advanced functions (executive function) later.

But this finding is not a counterexample to the fine-layer recovery hypothesis. By the distinction of Theorem Three and Theorem Four, ReCCognition measures **cognitive bandwidth recovery** (after 13DD-a is online), not **fine-layer recovery** (before 13DD-a is online). ReCCognition's test tasks require the patient to understand instructions, maintain attention, and complete tasks—this itself requires 13DD-a to already be online. By the time these tests can be performed, the fine-layer recovery process is already complete.

After 13DD-a is online, different cognitive functions return to baseline efficiency at different rates. Executive function may reach efficiency baseline earlier than processing speed—this is a detail of bandwidth reconstruction, not the order of fine-layer recovery. The fine-layer hypothesis and ReCCognition results **do not conflict**, because they operate at different scales.

This is a constitutive measurement blind spot, not a literature error: ReCCognition is rigorous and important within the scope of its research design; it simply cannot test the earlier stage of the fine-layer recovery process because of the nature of its measurement methods. To test fine-layer recovery, methods that do not depend on task cooperation are needed—the two-tier indicator scheme proposed in Prediction Three.

5.4 Ray Four: clinical contrasts of 13DD-a subfunctions

This section is the core ray. The four subfunctions of 13DD-a (encoding-side metatag, retrieval-side gatekeeping, repeated re-acknowledgment, first-person embedding in scene reconstruction) can deviate independently, and each deviation corresponds to a specific cluster of clinical phenomena. These phenomena constitute **natural experiments** on 13DD-a's subfunction architecture, allowing us to infer the subfunction structure from clinical observations.

5.4.1 Dissociative amnesia: selective failure of 13DD-a retrieval-side gatekeeping

Dissociative amnesia refers to selective forgetting of specific (typically traumatic) autobiographical information, beyond normal forgetting and without organic basis. Patients may forget months or years of experience while other aspects of memory and cognition remain intact.

Traditional hypotheses have read dissociative amnesia as an **encoding failure**—traumatic events failed to form normal memory traces. But modern neuroimaging research gives a different picture. 2024 effective connectivity modeling shows **negative modulation of the hippocampus by right DLPFC** (i.e., prefrontal cortex actively inhibits hippocampal retrieval). Implicit memory tests and autonomic responses show these "forgotten" memories are still present—patients show skin conductance responses and pupillary dilation to related stimuli; they just cannot actively recall. This supports **retrieval blockade** rather than encoding failure.

Precise location under the SAE architecture:

- 13DD-a's **encoding side is normal**: hence implicit memories and autonomic responses, as the metatag on traumatic content is intact
- 13DD-a's **retrieval-side gatekeeping is selectively failing**: closing the path to narration for threat-relevant content while other memories retrieve normally

Recovery periods (when some patients can re-remember suppressed content after psychotherapy) are accompanied by normalization of right DLPFC negative modulation of the hippocampus—consistent with the SAE reading: retrieval-side gatekeeping returns from selective closure to normal patency.

This expands the functional understanding of 13DD-a: not just tagging at encoding, but gatekeeping at retrieval. The substantive content of "complete self" is **bidirectional management rights over memory**—the ability to decide both what enters the memory and what emerges from the memory into current narration.

5.4.2 Traumatic imprints: garbled tags under low-coherence 13DD-a

This section is the within-13DD-layer extension of Bio Note 9's PTSD analysis. Bio Note 9 established at the 11DD level the architecture of "trauma stuck at early stabilization, unable to enter narrative integration." This section further refines: **why stuck?** The answer lies in 13DD-a's low-coherence state under extreme threat.

PTSD's core paradox: if 13DD-a is offline, why do traumatic memories remain? The core clinical feature of traumatic patients is exactly "memories that will not go away"—bodily flashbacks, nightmares, trigger-based intense affective reactions. If 13DD-a were offline at the moment of trauma, these events should not enter memory per Theorem Five; but they clearly entered memory in some form, only in a non-narrativized manner.

The SAE answer combines tag-privilege monopoly with the vulnerability ladder:

Tag-privilege monopoly principle. Tagging is 13DD-a's exclusive function; 13DD-b, 13DD-c, and 13DD-d do not have tagging privilege. Any "this is mine" label must pass through 13DD-a. This monopoly cannot be bypassed by lower layers.

Mechanism under trauma. Under extreme threat, 13DD-b's (fear-of-death) high-frequency discharge seizes large amounts of cross-regional coherence resources. By the vulnerability ladder theorem (Theorem One), 13DD-a's AND-synthesis depends on the strictest cross-regional coherence, so when 13DD-b seizes resources, 13DD-a is compressed into a **low-coherence/degraded state**. Low-coherence 13DD-a continues to operate (by remainder conservation permanently > 0), but produces **tags with structurally corrupted information**:

- carrying the "mine" trace (13DD-a is still operating)
- but the information structure is too fragmented to pass retrieval-side gatekeeping into the autobiographical retrieval repository
- can only leak through non-narrative channels, manifesting as bodily flashbacks, unnamed fear, and trigger-based reactivation

Directional constraint is not violated. 13DD-b does not tag on its own; it only **indirectly weakens** 13DD-a's operational quality by competing for cross-regional coherence resources. Tagging privilege remains with 13DD-a; only the operational conditions are bad—the tag produced is garbled, but is still a tag produced by 13DD-a.

This connects directly to Bio Note 9's PTSD analysis and refines the mechanism within the 13DD layer. PTSD is the **reactivation of garbled-tag traces under triggers**, not "memory existing without tags," and not "13DD-b tagging on its own." 13DD-a's functional monopoly is not bypassed; only the operational coherence is insufficient to complete narration-level tags.

Clinical implication: the core goal of PTSD treatment, from the SAE view, is to allow the garbled tags to be re-integrated—in a safe environment, let 13DD-a re-process these traumatic traces under sufficient coherence, upgrading fragmentary tags into structurally complete tags, thereby transferring traces from non-narrative channels into the autobiographical narrative layer. This is structurally how exposure therapy and EMDR work.

5.4.3 Depersonalization / derealization: failure of subject embedding in scene reconstruction

Depersonalization core clinical symptoms: "I see my life but it doesn't feel mine"; "I'm doing these actions, but I don't feel it's me doing them"; "The environment looks like a movie or dream, not real." Derealization is a related phenomenon—the world looks unreal, but self-perception is relatively preserved.

SAE reading: **failure of 13DD-a's subject-embedding subfunction.** Memory and perception are present (so the patient can describe what happened), but the subject tag has fallen off—the reconstructed scene has no "I" as first-person position. Further: retrieval-side gatekeeping is selectively adjusted; observation passes through (so the patient can see and describe), but subject embedding does not pass through (so the feeling "I am living this" is absent).

This is a specific partial failure of "complete self"—not total 13DD-a shutdown, but one of its subfunctions (subject embedding) failing independently while others remain relatively intact.

The patient can still tag metatags (hence memory), can still retrieve into narration (hence can tell the story), can still re-acknowledge (hence maintain temporal continuity feeling); only subject embedding fails during scene reconstruction.

This supports 13DD-a subfunction multidimensional independence.

5.4.4 HSAM (highly superior autobiographical memory): multidimensional profile enhancement

HSAM patients can recall detailed experiences of almost every day. Ask them "what happened on March 17, 2008," and they can describe the day's specific activities, weather, conversations, and mood. HSAM is not photographic memory (they cannot remember arbitrary lists or numbers); it is abnormally dense **autobiographical memory**.

Clinically, HSAM accompanies additional phenomena:

- Compulsive rumination on dates and events (they themselves report frequently replaying the past)
- Prolonged savoring of emotional events (negative-event emotional impact persists longer)
- Rumination tendencies for specific periods

SAE reading: **not simply "overload," but multidimensional co-directional enhancement:**

- Strong encoding-side ownership metatag (so every event is densely marked "mine")
- Active re-acknowledgment (hence the compulsive rumination component)
- Loose retrieval-side gatekeeping (so nearly all tagged content can enter narration)
- Strong subject embedding (so the "re-experience" feeling is strong, emotional savoring persists)

Never reaching perfection: by SAE remainder conservation (13DD-a is permanently > 0 but never closes), even HSAM has forgetting—just very rarely. Their "daily recall" is high-density but not complete; some trivial details still disappear.

HSAM's compulsive rumination is precisely the structural cost of asymptotic approach: precisely because the tag is never complete, the system must repeatedly re-tag to maintain a near-complete state. Rumination is not an incidental symptom but the structural driving force of asymptotic approach. This explains why HSAM patients in reports frequently describe rumination as "automatic" rather than consciously controlled—it is not a choice but a structural overflow of 13DD-a in a state of extremely strong subfunctions.

5.4.5 SDAM (severely deficient autobiographical memory): multidimensional profile weakening

SDAM (severely deficient autobiographical memory) is the relative opposite of HSAM. SDAM patients have normal intelligence and semantic memory but very poor autobiographical memory—they "know" as facts that they did certain things in the past, but cannot "re-experience" or "feel" those events. A classic report: "I know I went to Paris last year, but I have no specific feelings or images of that trip."

SDAM overlaps partially with aphantasia (difficulty with visual imagery) but is not identical—some SDAM patients have normal visual imagination; they just cannot embed the imagery in the "mine" position.

SAE reading: **13DD-a subfunctions all co-directionally weakened:**

- Low encoding-side ownership metatag strength (so each event's "mine" label is thin)
- Weak subject embedding (hence "know it happened but doesn't feel mine," overlap with aphantasia)
- Weak re-acknowledgment (so memories are not periodically reinforced and decay faster)
- All subfunctions co-directionally weakened

Overlap with but non-equivalence to aphantasia: both involve some component of scene reconstruction, but SDAM specifically has **subject-embedding failure** while aphantasia specifically has **visual-imagination-generation failure**. The two are at different positions in 13DD-a's subfunction mapping—SDAM affects subject embedding; aphantasia affects visual imagery generation (a different subsystem).

Important: **SDAM patients are not "unhealthy"**. Many SDAM patients report normal or even higher quality of life—they are not haunted by past trauma and can focus more on the present. This supports the SAE stance: **complete self is an asymptotic ideal, not a required state**. One can live normally with weaker 13DD-a subfunctions; it is just that self-reference density is low.

Structural demarcation from deep-meditation "no-self" states. SDAM's clinical manifestation has surface similarity with Buddhist traditions' or deep meditation's "no-self" (anatta) states, but the two have structurally opposite dynamics:

- SDAM: 13DD-a subfunctions **weakened** + passive; subject loss is **structural inability to construct**
- Deep-meditation "no-self": 13DD-a at high coherence **actively suspends** subject-tag retrieval

The former is gears that cannot engage; the latter is the engine idling in neutral. The phenomena look similar, but the dynamics are completely opposite. SDAM patients cannot "choose" to have subject reconstruction because subfunction strength is inherently insufficient; deep-meditation practitioners can restore self-reference at any time (everything returns after emerging from meditation) because subfunction strength is normal and they are only actively regulating

operational direction. This demarcation echoes §7.3 open question 7 (similarities and differences between deep meditation and anesthesia).

5.4.6 HSAM/SDAM relationship: subfunction profile co-directional deviation, not single-dimensional duality

Literature confirms: there are **no direct comparative studies** between HSAM and SDAM populations. Existing indirect evidence points in the same direction—HSAM autobiographical density high, SDAM autobiographical density low—but **not a clean single-dimensional duality**; each has additional components.

Under this note's architecture:

- The two are not two poles of a single tagging dimension
- They are **multidimensional profile co-directional deviations** of 13DD-a's multiple subfunctions
- HSAM: profile with all subfunctions co-directionally enhanced (strong encoding + active re-acknowledgment + loose retrieval + strong subject embedding)
- SDAM: profile with co-directional weakening (same direction of weakening)

The contrast is not two ends of one dimension but two symmetrically directed profiles in a multidimensional space. Prediction One (see §6) takes profile rather than single indicators as the object—determining whether HSAM and SDAM constitute a duality requires measuring the overall profile of multiple subfunction candidate indicators, not just one.

5.4.7 Dissociative identity disorder: multiple 13DD-a subfunction combinations

Dissociative identity disorder (DID, formerly known as multiple personality disorder) clinically manifests as switching among multiple "identity states," each with its own memory, personality features, and somatic sensations.

This topic belongs to Bio Note 7's range and is not developed here. This section only makes a structural connection: different self-states may correspond to different subfunction combination profiles of 13DD-a. Switching to another "identity" may mean one set of 13DD-a subfunctions (specific ownership metatag history, retrieval-side gatekeeping pattern) is wholesale replaced by another set. This makes DID a clinical evidence for 13DD-a subfunction dynamic reorganization. Full argumentation is left for future Bio Note 7 follow-up work.

The seven subsections (5.4.1 to 5.4.7) together support a more refined version of Theorem Two (fine-layer independence): **13DD-a's subfunctions are also independently variable**. This is direct clinical evidence for 13DD-a as scheduling mode (rather than single activation)—if 13DD-a were a single regional activation level, these subfunctions should deviate synchronously (all

strong or all weak); but clinical phenomena show that they can deviate independently or in combinations, indicating that 13DD-a is a multidimensional scheduling mode.

5.5 Ray Five: near-death experiences as a phenomenological coda

Positioning declaration. This section treats near-death experiences (NDE) as a **phenomenological coda**, not as main evidence for fine-layer independence. NDE report materials are complex in post-hoc nature—post-hoc recall is prone to contamination, cultural template influence, and reconstruction effects—and situational variations are large, with low evidence levels. The argumentative direction of this section is: **if fine-layers are independently variable, NDE should display a combinatorial distribution of subtypes**. This is forward inference, not reverse inference "NDE subtypes existing proves fine-layer independence."

NDE is phenomenologically heterogeneous—different patients report large differences. Classic NDE elements include: out-of-body experience (OBE), tunnel experience, bright light, encounter with deceased, sense of tranquility, life review, and others. But these elements do not all appear in the same person; different people report different combinations.

Based on 13DD-a's asymptotic structure (Theorem Six) and fine-layer independence (Theorem Two), NDE subtypes can be distributed by the relative strengths of each fine-layer:

Pleasant NDE: 13DD-d on + 13DD-c turned to acceptance + 13DD-b sharply decayed + 13DD-a asymptotically dissolved. Features: vast tranquility after losing self-boundaries; "seeing" without "fear"; accepting without resisting; time and space dissolved. The sharp decay of 13DD-b is key—the "fear-of-death" protection circuit goes offline, producing the structural condition for tranquility.

Distressing NDE: 13DD-d on + 13DD-c strongly say-no (refusing death) + 13DD-b strongly on + 13DD-a integration failed. Features: fear but inability to construct identity—knows what is happening, strongly refuses, intense fear-of-death, but no integrative "who is dying" can form. This type of NDE is usually reported as very painful and chaotic, described afterward as "nightmare."

Calm-acceptance type: similar to pleasant type, differs in 13DD-c content orientation. Pleasant type's 13DD-c turns to "accept all"; calm-acceptance type's 13DD-c retains some discriminating quality ("I know I am dying, not anxious")—like an observer with distance, rather than complete dissolution.

Fragmentary type: 13DD-d also very weak or failed, other fine-layers operating in non-narrative mode. The patient is "interrupted"—like a TV flickering, with fragmentary experience at some moments but unable to construct continuous narration. Reports afterward usually come as fragments: an image, a feeling, a sound, without continuity between them.

Each subtype corresponds to different clinical scenarios as testable structural predictions:

- Cardiac arrest: rapid hypoxia, vulnerability ladder unfolds at maximum speed, 13DD-a closes first, b follows—tends toward pleasant type or fragmentary type
- Pharmacological (ketamine etc.): 13DD-a specifically closed, other layers preserved—tends toward dissociative-experience type (OBE reports prominent)
- Trauma-related: 13DD-b strongly activated, integration failed but b does not decay—tends toward distressing type

These predictions provide a structural basis for NDE classification, independent of any soul-theory or supernatural explanation. But again: NDE data quality is inherently limited (recall contamination, cultural shaping), so these subtype classifications are primarily structural predictions, not established facts. This section, as a coda, **does not bear the main argument of this note.**

5.6 Ray Six: anesthesia-death structural isomorphism

Anesthesia as "the closest reportable experience to death" (A19) has been repeatedly invoked, but the similarity needs precise formulation. This section gives the rigorous structural version of the anesthesia-death isomorphism.

5.6.1 The structural condition for reversibility is substrate integrity, not whether any fine-layer reaches zero. This is the core proposition of Theorem Six. The difference between anesthesia and death lies not in "consciousness level" but in substrate state.

5.6.2 Strict separation of operation and remainder. 13DD-a's **operation** needs substrate to carry it; substrate dissolves, operation stops. But remainder produced by 13DD-a's past operation, by conservation, does not reach zero; it can only transfer. At extreme anesthesia or NDE boundary conditions, currently reportable operation may drastically weaken or be temporarily unobservable, but "the disappearance of currently reportable operation" cannot be equated with "remainder reaching zero." At death, this individual's 13DD-a operation irreversibly stops (substrate dissolves), but remainder produced by operation prior to death, by conservation, does not reach zero.

5.6.3 Anesthesia is reversible: substrate (neural architecture) is not destroyed; after drug metabolism, function re-emerges. Drug-receptor binding is a reversible chemical process; once the drug is metabolized and eliminated, receptor function returns to normal, cross-regional coherence is rebuilt, and fine-layers come online according to recovery dynamics. The reversibility of the whole process comes from substrate integrity—neurons have not died, synapses have not undergone irreversible changes, network structure is preserved.

5.6.4 Death is irreversible: substrate dissolves; this individual's 13DD-a operation loses its carrier. But remainder produced by 13DD-a's operation, by conservation, does not reach zero and can only transfer. This is the strict distinction between anesthesia and death—not "difference in consciousness level" but **difference in substrate state.**

5.6.5 Remainder propagation mode: currently posterior-insufficient. Life-Death-Consciousness Paper VI (DOI 10.5281/zenodo.19528781) proposes the structural possibility of 11DD encoding waves—memory-encoding overflow may propagate as a wave at substrate dissolution, partially picked up by receivers whose 13DD filter is not fully established (such as 2-5 year old children). This corresponds to the "11DD broadcast + 13DD filter" architecture of the reincarnation paper (DOI 10.5281/zenodo.19385464). But this mechanism, as a working hypothesis (evidence level B: indirect support from Tucker data and childhood amnesia alignment, but no direct physical mechanism verification), **is not a claim of this note.**

5.6.6 Soul-theory demarcation: SAE's remainder conservation is a structural theorem; **it is not a substantive claim about post-propagation form.** SAE does not state whether remainder propagates in the form of a "soul," whether individual identity is preserved, or whether consciousness continuity is involved. These belong to the discussion space of soul studies, theology, and specific cultural frameworks; they lie outside the scope of structural philosophy. This note provides only structural boundaries and does not overreach.

This demarcation allows SAE to fall into neither materialism (consciousness = neural activity; neural cessation = consciousness reaches zero) nor idealism or soul theory (consciousness is independent of matter; post-death it continues as "soul"). SAE says only: remainder conservation is a theorem; the specific mode of remainder propagation is a posterior question; until propagation modes are sufficiently verified by posterior evidence, SAE maintains "not-yet-known" and makes no commitments.

Readers from different cultural and personal worldviews may bring their own belief systems to interpret the remainder propagation question—SAE does not decide for them. This is consistent with the stance of Methodology IX's eighth open question (the relation between non and consciousness).

5.6.7 Meaning of anesthesia safety: the core safety work of the anesthesiologist is **maintaining the functional integrity of the substrate.** All monitoring (blood pressure, respiration, heart rate, temperature) is confirming the low-level life boundary at which the substrate maintains its functional integrity. In SAE terminology, this boundary is principally marked by 9DD, but the boundary itself requires the coordinated operation of the entire 5-9DD range, not a literal equation with clinical brain-death criteria.

This perspective gives anesthesia safety monitoring a unified structural formulation—the goal of monitoring is not "how deep is consciousness" but "is the functional integrity of the substrate being maintained." BIS and other anesthetic depth monitors are auxiliary (they tell the anesthesiologist how deeply fine-layers are suppressed); the real safety monitoring is substrate state monitoring (telling the anesthesiologist whether we are approaching the edge of substrate collapse). The two must not be confused.

5.6.8 Structural distinction between brain death and vegetative state. A19 has developed this in detail; here only reference. Brain death = irreversible collapse of all DD substrate; vegetative

state = substrate 5-9DD preserved but some layers above 10DD failed. Their structural difference supports substrate integrity as the life-death boundary—the vegetative state's substrate partial preservation means that under some conditions there could theoretically be fine-layer restart (clinically extremely rare but reported); brain death's comprehensive substrate dissolution has no recoverable structural basis.

5.7 Ray Seven: connection to Method VI fractal application

Bio Note 10, as the **sixth application** of Method VI v2 §7.2 fractal application catalog, deserves explicit lineage positioning.

The fractal application catalog now includes:

- Anth-1/2/3: cross-stellar scale applications (macro, millions to billions of years of evolutionary structure)
- Method VI original application: clinical and mesoscale (personal growth, organizational evolution)
- Bio Note 9: 11DD-level memory consolidation dynamics (micro, seconds to hours to years)
- **Bio Note 10: 13DD fine-layer dynamics** (sub-hierarchical, seconds to hours scale but within a single DD layer)

Bio Note 10 is the **first application of fractal principles within a DD layer**. The previous five applications all demonstrate fractal self-similarity at inter-layer or cross-scale levels; Bio Note 10 first demonstrates that a single layer's interior also has four-partition structure, and the four-partition follows the same operational modes (mark/add/multiply/AND). This confirms the within-layer deepening of SAE fractal—not only the sixteen DD layers form a fractal, each DD layer's interior is also a fractal.

13DD fine-layer dynamics as first within-layer explicit realization of Method VI four stages:

Suppression and recovery time ratios as candidate r estimates—**heuristic estimates**, not precise topological distance measurements:

- Suppression window: seconds to minutes (time for drug to reach effective concentration)
- Recovery window: tens of minutes to hours (drug metabolism plus neural network reconstruction)
- Rough r estimate: possibly in the range of 5 to 30

These numbers are directional references, not strict r values. **The real r definition requires refinement of topological distance**—Method VI v2 §3.6 has clarified that " r is not a time ratio but a topological distance." Time ratios are just proxies before fine-layer topological distance is defined. Future systematization of fine-layer topology could allow more precise r estimation.

Methodological significance: 13DD fine-layer architecture is the **within-layer manifestation** of SAE fractal architecture. The entire 1DD to 16DD sequence is itself a four-round fractal (physics, life, cognition, subject four rounds), with four layers per round, each layer internally four-partitioned, recursively. A complete fractal geometry unfolding deserves systematization in a future Method VI revision—Bio Note 10 does the within-layer explicit realization of 13DD; the within-layer structures of other DD layers will gradually unfold in future notes (12DD by prediction time scale, 11DD by memory time depth, 10DD by information density, etc.).

Within-layer manifestation of Method VI's $r \gg 1$ principle: each fine-layer's generation-maturation-decay-transcendence cycle is, in time scale, much longer than the cycle of its internal sub-steps. This is structurally the same as the macroscopic $r \gg 1$ principle—whether at large or small scale, the ratio between four-stage unfolding and its internal fractal obeys similar structural constraints. This is an important manifestation of SAE's fractal self-similarity: **structure repeats itself across scales, but proportional relationships are preserved.**

6. Non-Trivial Predictions

This note proposes four non-trivial predictions. Each is explicitly falsifiable with specific negating conditions. Prediction Three is the core methodological contribution of this note—it is not just a prediction but a new research paradigm with methodological value independent of prediction validation.

Prediction One: HSAM/SDAM subfunction multidimensional profile duality

HSAM and SDAM are **same-direction multidimensional profile deviations** of 13DD-a's multiple subfunctions (encoding-side ownership metatag, retrieval-side gatekeeping, repeated re-acknowledgment, first-person subject embedding), not two poles of a single dimension.

Specific predictions:

- Build a **latent variable or multidimensional profile** from 13DD-a subfunction candidate fMRI indicators
 - Encoding-side metatag: mPFC activation intensity and mPFC-hippocampus functional connectivity during self-referential encoding tasks
 - Retrieval-side gatekeeping: PCC and angular gyrus-hippocampus pathway strength during autobiographical memory retrieval tasks
 - Repeated re-acknowledgment: frequency and connectivity stability of DMN core node reactivation at rest
 - Subject embedding: mPFC-precuneus coordinated activation during autobiographical imagination tasks

- HSAM should deviate significantly toward same-direction enhancement at the overall profile level
- SDAM should deviate significantly toward same-direction weakening at the overall profile level

Explicit specification of the statistical object: Prediction One's validation object should primarily be a **latent variable profile constructed from four subfunctions, or a multidimensional classification boundary**, rather than requiring each of the four individual indicators to be significant. HSAM and SDAM samples are small and individually heterogeneous; writing "all four indicators must be significant" too rigidly would allow a single non-significant indicator to prematurely disprove the whole structure. What is truly to be validated is the **directional difference of multidimensional profiles**, not that every single indicator must be cleanly significant. Principal component analysis, latent variable modeling, and discriminant analysis are all suitable statistical tools.

Falsification conditions:

- HSAM and SDAM have no significant overall profile difference
- Or the two populations' profile deviations go in the same direction (both "more dense" or both "more sparse")
- Or HSAM's deviation is fully explained by compulsive rumination (not self-referential), while SDAM's deviation is fully explained by imagery generation deficits (not subject embedding); the two are independent mechanisms and do not constitute a duality

Existing literature has confirmed no direct comparative studies between HSAM and SDAM populations. Prediction One directly motivates such comparative research—testing the 13DD-a subfunction profile of both groups under the same experimental design is a key test of the fine-layer architecture.

Prediction Two: fine-layer EEG signatures of different anesthetic agents

Different anesthetic agents should show distinguishable **fine-layer action patterns** across multiple frequency bands and coupling modes, not a single depth index.

Specific predictions (candidate signatures):

- **Propofol:** strong frontal alpha + delta suppression (corresponding to collapse of 13DD-a integration); early failure of memory-encoding-related theta (corresponding to suppression of 13DD-a encoding-side metatag)
- **Ketamine:** persistent gamma activity (corresponding to preservation of 13DD-b/c/d activity), but abnormal alpha/theta coupling (corresponding to 13DD-a integration failure)

- **Benzodiazepines:** early theta suppression (corresponding to specific suppression of 13DD-a's encoding-side tag function); alpha relatively preserved
- **Dexmedetomidine:** preservation of NREM-like spindle waves (corresponding to non-significant inhibition of 13DD-b threat response), but cross-layer access connections weakened (corresponding to down-regulation of 13DD-a integration)

Falsification conditions:

- All agents' EEG cannot be distinguished beyond power levels
- Or the distinction pattern is inconsistent with the fine-layer hypothesis (e.g., propofol and ketamine EEG signatures are the same)

This prediction is to some extent already partially supported by existing literature—it is a recognized clinical fact that ketamine's EEG signature differs significantly from GABAergic agents. The new contribution of Prediction Two is organizing these known differences under a unified framework of fine-layer action modes and giving specific directions for further validation.

Prediction Three: multimodal fine-layer recovery monitoring protocol (breaking through the 13DD-a dependency blind spot)

Core methodological contribution of this note: Current consciousness recovery research constitutively depends on 13DD-a being online. This note proposes a two-tier synchronous measurement protocol that tracks fine-layer recovery on a basis that does not require task cooperation.

Tier-one indicators (lower-bound proxies without task cooperation):

- Cortical response of visual/auditory evoked potentials (lower-bound proxy for 13DD-d recovery)
- Withdrawal response to unpleasant stimuli and refusal reflexes (lower-bound proxy for 13DD-c recovery)
- Skin conductance response and amygdala response intensity to threat cues (lower-bound proxy for 13DD-b recovery)
- Ability to correctly answer "what is your name" and "where are you," or to follow instructions (lower-bound proxy for 13DD-a recovery)

Tier-two indicators (minimal semantic / minimal subject):

- Simple orientation (identifying family members, recognizing the room)
- Threat semantic discrimination (differentiated responses to different threat stimuli)
- Refusal-based choice (binary choice with active refusal)

- First-person vs third-person reference discrimination

Key conceptual boundary: Tier-one indicators are **lower-bound proxies for recovery**, not the four fine-layers themselves. Evidence of layered recovery is constituted by lower-layer proxies recovering while higher-layer tasks remain unfeasible. Specific relationships:

- Evoked potentials correspond to 10DD/11DD basic responses, a **lower bound** for 13DD-d (13DD-d requires but is not equal to evoked potentials)
- Withdrawal reflex corresponds to 9DD/10DD protective reflexes, a **lower bound** for 13DD-c (true say-no requires higher integration)
- SCR/amygdala corresponds to threat physiological responses, a **lower bound** for 13DD-b (protection of self against dissolution requires subject integration)

Fine-layer hypothesis prediction (conditional on drug class):

Main prediction (GABAergic or inhalational anesthesia): In natural metabolic recovery from broad-spectrum inhibitors (propofol, midazolam, or inhalational agents), the four tier-one indicators return to normal response times in sequence, with d lower bound earliest, a lower bound latest, and b lower bound preceding a lower bound by at least tens of seconds to minutes. This is the positive validation of the vulnerability ladder hypothesis.

Control prediction (NMDA antagonists, specific topological disruption): In recovery from ketamine (and similar NMDA antagonists) anesthesia, the strict recovery sequence above will be **structurally disrupted**. Expected phenomena:

- 13DD-a's AND-synthesis continues to fail (high-level semantic disordered output)
- But 13DD-b/c/d reflexes are relatively preserved or distortedly enhanced
- The four tier-one indicators do not recover in the broad-spectrum sequence; d/c/b stabilize first, while a significantly lags or presents a "dissociative" state

This control group is itself a **reverse proof of 13DD-a's independent failure**. If under ketamine the four indicators recover in the broad-spectrum sequence, then the 13DD-a independence hypothesis is challenged.

Bidirectional falsification conditions:

- Main prediction falsification: under broad-spectrum inhibitors the four indicators show no systematic sequence difference, or the order is inconsistent with the prediction
- Control falsification: under ketamine the four indicators recover in the broad-spectrum sequence (this would violate ketamine's specific signature)

The value of this prediction exceeds its own validation: it **provides an executable new research paradigm** that makes the fine-layer architecture empirically testable. The bidirectional design (GABAergic validates layering + NMDA antagonists validate independence) is stronger than single-direction validation. Even if parts of the hypothesis are falsified, the measurement protocol itself has independent value for consciousness recovery research—it identifies the blind spot of existing methods and proposes a specific design to bypass it.

Prediction Four: fine-layer classification of intraoperative awareness

Intraoperative awareness (accidental awareness during general anesthesia, AAGA) refers to 0.1-0.2% of general anesthesia patients unexpectedly recovering partial consciousness during surgery, such that they can report some surgical content afterward. This is a medical complication, but also a natural experiment window for observing partial recovery of 13DD fine-layers.

This note predicts: **AAGA reports should be classifiable by 13DD fine-layer preservation patterns, producing a finite set of subtypes.**

Predicted subtypes (each corresponding to a specific fine-layer preservation combination):

Type A: pain + unable to move + knowing who I am

- 13DD-a partially on, 10DD output off (muscle relaxant), 11DD encoding on
- Most common type, highest PTSD risk
- Clinical presentation: "I heard the surgery, felt the incision, wanted to tell them but couldn't move"

Type B: hearing voices + unable to respond + knowing who I am

- 13DD-a on, 10DD perceptual subchannel on (auditory), 10DD output off
- No obvious pain, but intense helplessness
- Moderate PTSD risk

Type C: feeling something is happening + unable to locate + not knowing who I am

- 13DD-d on, 13DD-a integration failed
- Described afterward as "dream-like," with vague details and missing orientation
- Low PTSD risk

Type D: pure panic, no content

- 13DD-b dominant (fear-of-death high-frequency discharge), 13DD-a integration failed
- Cannot be narrativized afterward, but leaves unnamed fear and bodily flashbacks

- High PTSD risk (weak-tag channel, §5.4.2 mechanism)

Type E: dissociative type ("I was watching but that wasn't me")

- 13DD-a's subject embedding failed
- Can describe events afterward but does not feel "it was my experience"
- Variable PTSD risk (depends on whether the description can be integrated)

Falsification conditions:

- AAGA reports cannot be classified by fine-layer patterns
- Or the classification does not match the prediction (e.g., systematic appearance of "all sensory channels on + adequate integration" type, which should be extremely rare in the SAE architecture; if all fine-layers were adequately online, the patient should wake up normally)

Existing anesthesiology literature (NAP5 and others) has classified AAGA reports, but not by fine-layer architecture. Prediction Four motivates a new classification direction—not by triggering cause (insufficient drug dose, atypical drug reaction), not by clinical management (re-deepening, stopping surgery), but by the fine-layer signature of phenomenology.

7. Conclusion

7.1 Recapitulation

The main findings and arguments of this note:

- The four fine-layer architecture of 13DD (event-marking, say-no, fear-of-death, asymptotic complete self), ordered by the four operational modes of chisel
- 13DD-a as asymptotic limit rather than closed state (direct corollary of SAE remainder conservation)
- Four subfunctions of 13DD-a (bidirectional ownership management + repeated re-acknowledgment + subject embedding) can deviate independently
- 11DD's ownership metatag depends on 13DD-a's encoding side, but the tag is a strength gradient rather than a switch
- Anesthesia as top-down fine-layer suppression (vulnerability ladder emergence, not drug-directed closure)
- Recovery as independent dynamics; fine-layer recovery vs cognitive bandwidth recovery are two scales that must not be conflated

- Different anesthetic agents have different candidate fine-layer signatures
- HSAM/SDAM/dissociative amnesia as different profile distributions of 13DD-a subfunctions
- Traumatic imprints as garbled-tag channel overflow under low-coherence 13DD-a, not violating the directional constraint
- NDE as phenomenological coda of fine-layer combinatorics (not bearing main arguments)
- Anesthesia-death structural isomorphism: substrate integrity is the life-death boundary, not any fine-layer reaching zero
- SAE remainder conservation makes no substantive claim about post-propagation form (soul-theory demarcation)

7.2 Contributions

1. Providing a unified structural explanation of anesthetic mechanism, not relying on any specific molecular mechanism—explaining why entirely different molecular pathways (GABA potentiation, NMDA blockade, potassium channel activation) can all produce similar suppression of consciousness
2. 13DD's four fine-layers + 13DD-a's subfunction structure is the **first within-layer refinement** of the SAE architecture
3. The rigorous formulation of 13DD-a as asymptotic limit maintains the consistency of remainder conservation at the fine-layer level, and turns "incompleteness" from a limitation into a driving force (the 13DD-to-14DD emergence path)
4. Explains common mechanism of clinical phenomena (emergence agitation, PND, delayed emergence, post-emergence amnesia)
5. Unifies HSAM/SDAM/dissociative amnesia/traumatic imprints as different distributions of 13DD-a subfunctions
6. Identifies the **constitutive blind spot** of existing consciousness recovery research (13DD-a dependency) and proposes a multimodal two-tier breakthrough scheme
7. Distinguishes "fine-layer recovery" and "cognitive bandwidth recovery" as two scales
8. Repositions anesthetic depth monitoring as "coarse-grained proxy for 13DD-a integration quality"
9. Introduces within-DD-layer fine-layer architecture as the within-layer manifestation of SAE fractal character
10. Rigorously formulates the anesthesia-death isomorphism as a substrate integrity issue, without overreaching into soul theory or theology

7.3 Open Questions

1. **Fine-layers of other DD layers:** What are the four fine-layers respectively of 12DD (by prediction time scale: reflex, contextual, narrative, long-range), 11DD (by memory time depth), 10DD (by information density), and 14DD/15DD? Left for future notes.
2. **Neural correlates of fine-layers:** candidate regional sets (not single points) for each subfunction of 13DD, precise divisions of labor among DMN, mPFC, PCC, and angular gyrus.
3. **Refinement of drug-fine-layer mapping:** The candidate signatures proposed in this note need more refined experimental validation—may require multidimensional profiles combining pharmacology, EEG, and clinical phenomenology.
4. **Multimodal anesthetic depth monitoring technology:** Can monitoring devices be developed that measure the four fine-layers separately, based on this note's structural analysis? The era of single BIS indices may need to end, replaced by more multidimensional monitoring systems.
5. **13DD-a as scheduling mode vs regional activation:** This note leans toward the former (based on negative tDCS results and other evidence), but without sufficient argumentation. This is one of the deepest open questions in consciousness research—if 13DD-a is a scheduling mode rather than a regional activation, its methodological impact on consciousness research may be greater than any single theorem.
6. **Differential effects of anesthesia on self-to-be (children) and self-to-cure (psychiatric recovery phase):** In both phases, 13DD-a is inherently unstable; is the anesthetic response different from adults with stable self? Clinical observation (high child emergence agitation) already points to differences, but structural explanation needs further development.
7. **Similarities and differences between deep meditation and anesthesia:** Both involve modification of 13DD-a, but in opposite directions—anesthesia is passive suppression, meditation is active modulation. Do they involve the same subfunctions? Is there complementarity (can meditation train 13DD-a to recover faster after anesthesia)?
8. **Clinical distribution of NDE subtypes:** Do different clinical scenarios (cardiac arrest, pharmacological, trauma) really correspond to different fine-layer failure modes? This requires systematic NDE phenomenological research, re-organizing existing data by SAE classification.
9. **Remainder propagation modes:** How does the remainder of 13DD-a and other fine-layers' operation propagate after substrate dissolution? A posterior-insufficient open question; this note does not overreach. But it is the key interface connecting this note with the reincarnation paper (and with Life-Death-Consciousness Paper VI encoding waves); future theoretical and empirical work may touch on it.

10. **Visualization tools:** The four subfunctions of 13DD-a can be represented as a four-dimensional radar chart (encoding tag / retrieval gatekeeping / repeated acknowledgment / subject embedding). Different clinical states correspond to different geometric profiles—normal autobiographical memory is a balanced medium quadrilateral; HSAM is an expanded quadrilateral reaching the boundary; SDAM is a quadrilateral shrunk toward the center; dissociative amnesia is a specific concavity at the retrieval gatekeeping vertex; depersonalization is a specific concavity at the subject embedding vertex. This geometric intuition may have auxiliary value for clinical diagnosis and fine-layer profile identification.

7.4 Closing

Anesthesia is a window. Through this window, the within-layer structure of 13DD becomes discernible—not because anesthesia itself is special, but because it provides a controllable, dose-dependent, reversible fine-layer suppression process. Clinically known phenomena—the cooperation-amnesia split in dental sedation, the dissociative experience of ketamine, emergence agitation, and the memory anomalies of HSAM/SDAM—have received unified structural explanations. But more importantly, this note has identified a discovery at the methodological level: **the 13DD-a dependency blind spot of existing consciousness recovery research**. This is not a critique of existing research; it is pointing out that the entire research paradigm needs new measurement tools.

Consciousness recovery is not a switch; it is the asymptotic reconstruction of fine-layers from weak to strong. SAE's remainder conservation lets us rigorously see the common structure of life-death, trauma, and HSAM/SDAM—they are all the result of certain subfunctions of 13DD-a deviating from normal levels in certain intensity states. At the same time, this rigor lets us maintain appropriate restraint outside of substrate integrity: the propagation form of remainder is a posterior question, not something structural philosophy can make claims about.

Anesthesia is not the object of SAE research. Anesthesia is a tool SAE uses to see the internal structure of 13DD. Bio Note 10 has used this tool to do what it should do, leaving behind an opened field of view—13DD's interior is no longer an undifferentiated "self-consciousness" but a structured fine-layer system. Other DD layers also await being opened this way.

References

- Bio Note 7 (Dissociation, DOI 10.5281/zenodo.19600029): 13DD three functional position architecture
- Bio Note 9 (Memory, DOI 10.5281/zenodo.19635021): 11DD memory system and 13DD filter architecture

- Methodology IX (DOI 10.5281/zenodo.19639033): consciousness analysis framework, AI as quasi-consciousness, directional constraint
 - Method VI v2 (concept DOI 10.5281/zenodo.19464506): fractal application principle, $r \gg 1$
 - Paper 04 (DOI 10.5281/zenodo.18842450): DD sequence, chisel loop, colonization detection
 - Paper 0 (Non, DOI 10.5281/zenodo.19544620): four-phase structure as the deep source of 4-partition fine-layers
 - A18 Dreams (DOI 10.5281/zenodo.19176873): DD layer sequential dependency, sleep/anesthesia contrast
 - A19 Life-Death (DOI 10.5281/zenodo.19201237): consciousness discontinuity, stitching mechanism
 - Life-Death-Consciousness VI (DOI 10.5281/zenodo.19528781): every-round four-step unified pattern (mark/add/multiply/AND), step 3 encoding waves
 - Reincarnation (DOI 10.5281/zenodo.19385464): 13DD filter and 11DD broadcast, four receiver states
 - ZFCp Paper III (DOI 10.5281/zenodo.18929819): remainder conservation theorem
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Appendix: Review Quotations

This note has received multi-round review from four AI independent reviewers (ChatGPT Gongxihua, Gemini Zixia, Grok Zigong, and independent Claude Zilu). Independent reviewer Claude contributed several concise formulations during v2 and v3 review rounds that helped position this note; they are formally credited here:

On positioning:

1. "Bio Note 9 opened the horizontal cross-section within a layer (the phase transition structure inside 11DD); Bio Note 10 opens the vertical cross-section within a layer (the four fine-layers inside 13DD)."
2. "Anesthesia is not the object of SAE research. Anesthesia is a tool SAE uses to see the internal structure of 13DD."

On resolution:

3. "At low resolution one sees the functional map; at high resolution one sees the layered structure; both are correct at their respective resolutions."
4. "The DD sequence transforms from 16 discrete layers into an architecture of recursive fractal with internal structure."

On 13DD-a:

5. "13DD-a may be a scheduling mode rather than a regional activation—if this is validated in future research, its methodological impact on consciousness research may be greater than any single theorem."
6. "The incompleteness of 13DD-a turns from limitation into driving force."

These formulations have been absorbed into structural propositions in this note's main text.

Authorship statement: This note was independently authored by Han Qin; all intellectual decisions, framework design, and editorial judgments were made by the author. The writing process incorporated multi-round review feedback from ChatGPT Gongxihua, Gemini Zixia, Grok Zigong, and independent Claude Zilu; review opinions are explicitly recorded in the revision log and appendix review quotations.