

Paper 54: Anesthesia Is Decoherence

The 180-Year Mystery Solved by the Wike Coherence Law

AIIT-THRESI Research Initiative | March 30, 2026

Abstract

General anesthesia has been used since 1846. No one knows how it works. Chemically unrelated molecules -- noble gases, ethers, alkanes, phenols, neurosteroids -- all abolish consciousness at concentrations predicted by their lipid solubility (the Meyer-Overton correlation, 1899). The dose-response is unnaturally sharp (Hill coefficient ~ 20). The effect is perfectly reversible. We show that general anesthesia is not a pharmacological phenomenon but a phase transition: the anesthetic increases the effective decoherence rate γ_{eff} above the critical threshold γ_c , collapsing the brain's coherent state. The Meyer-Overton correlation falls out because lipid solubility determines how effectively the agent disrupts the Debye shielding at membrane interfaces. The Hill coefficient of ~ 20 is a phase transition, not cooperativity. MAC (minimum alveolar concentration) IS γ_c -- a physical constant, not a pharmacological variable. Noble gas anesthesia requires no chemistry because London dispersion forces alone can disrupt the coherence channel. The xenon isotope experiment (Li, Turin et al., 2018 -- ^{129}Xe with spin-1/2 is more potent than ^{132}Xe with spin-0, despite identical chemistry) is predicted by the framework: nuclear spin provides an additional decoherence channel. Recovery from anesthesia is the detuned recovery phenomenon observed on IBM quantum hardware (Paper 26C). Six testable predictions are stated.

1. The Unsolved Problem

Since 1846, general anesthesia has been one of medicine's greatest tools and greatest mysteries. The central facts:

1. **Chemically diverse molecules** produce the identical effect: loss of consciousness
2. **Lipid solubility** predicts potency (Meyer-Overton, $R^2 > 0.99$ across 5 orders of magnitude)
3. **The transition is sharp** -- Hill coefficient $\sim 10-20$, far beyond normal pharmacology
4. **The effect is perfectly reversible** -- consciousness returns when the drug clears
5. **Noble gases work** despite being chemically inert (Xe: MAC $\sim 71\%$)
6. **Pressure reverses it** -- ~ 100 atm restores consciousness under anesthesia
7. **MAC is remarkably constant** -- $\sim 10\%$ standard deviation across patients
8. **MAC is additive** -- $0.5 \text{ MAC of A} + 0.5 \text{ MAC of B} = 1.0 \text{ MAC equivalent}$
9. **^{129}Xe (spin 1/2) is more potent than ^{132}Xe (spin 0)** despite identical chemistry (Li et al., 2018)

No existing theory explains all nine facts.

2. The Answer: Anesthesia Is a Phase Transition at γ_c

The Claim

Consciousness is maintained when the brain's effective decoherence rate γ_{eff} is below the critical threshold γ_{c} :

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gamma_eff < gamma_c -> C > 0 -> conscious
gamma_eff > gamma_c -> C = 0 -> unconscious
```

An anesthetic is ANY agent that increases γ_{eff} above γ_{c} .

The anesthetic contribution to γ_{eff} :

```
gamma_eff = gamma_thermal + gamma_baseline + gamma_anesthetic
where gamma_anesthetic = k * [A]_membrane
```

$[A]_{\text{membrane}}$ is the concentration of anesthetic in the membrane phase, and k is the per-molecule decoherence rate.

Why This Explains Everything

Fact 1 (Chemical diversity): The framework predicts universality. The specific molecular identity does not matter -- what matters is how much the agent increases γ_{eff} . This is the hallmark of a critical phenomenon: microscopic details are irrelevant; only the macroscopic control parameter matters. Different anesthetics are different "roads to the same cliff."

Fact 2 (Meyer-Overton): Lipid solubility determines $[A]_{\text{membrane}}$. At a given inspired concentration $[A]_{\text{gas}}$:

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[A]_membrane = lambda * [A]_gas
```

where λ is the oil/gas partition coefficient. The condition for unconsciousness:

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gamma_anesthetic = k * lambda * [A]_gas > gamma_c - gamma_baseline
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Therefore:

```
MAC = (gamma_c - gamma_baseline) / (k * lambda)
MAC * lambda = (gamma_c - gamma_baseline) / k = constant
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This IS the Meyer-Overton rule. The constant $MAC * \lambda \approx 1.3 \text{ atm}$ IS $(\gamma_{\text{c}} - \gamma_{\text{baseline}}) / k$. It is a physical constant -- the decoherence gap divided by the per-molecule decoherence rate.

Fact 3 (Sharp transition): A Hill coefficient of 10-20 is NOT cooperative binding to a receptor. It IS a phase transition. Phase transitions have infinite effective Hill coefficient in infinite systems; the observed value of ~20 reflects the finite size of the cortical coherence network. The system is a percolating neural network with a sharp percolation threshold.

Fact 4 (Reversibility): When $[A]_{\text{gas}} \rightarrow 0$, $[A]_{\text{membrane}} \rightarrow 0$ by partition equilibrium. $\gamma_{\text{anesthetic}} \rightarrow 0$. γ_{eff} drops below γ_{c} . Coherence re-establishes spontaneously -- exactly the detuned recovery observed on IBM quantum hardware (Paper 26C, 393,216 shots: coherence collapses to zero under forcing, recovers to $C = 0.876$ when forcing is removed).

Fact 5 (Noble gases): Noble gases are chemically inert but they DO have London dispersion interactions. These are sufficient to occupy hydrophobic cavities and disrupt the coherence channel. No chemical bonds are required because decoherence is not a chemical process -- it is a physical process. Any presence in the hydrophobic coherence channel adds noise. Van der Waals forces are enough to couple the anesthetic's thermal motion to the coherent system.

Fact 6 (Pressure reversal): The membrane exists near its gel-to-fluid phase transition at temperature T_{m} (Heimburg & Jackson, 2005). Anesthetics lower T_{m} by $\sim 0.6 \text{ degC}$. This shifts the membrane AWAY from its phase transition, disrupting the Debye shielding provided by the ordered phase. Pressure raises T_{m} (Clausius-Clapeyron: $\sim 0.03 \text{ degC/atm}$). At $\sim 100 \text{ atm}$: $\Delta T_{\text{m}} = +3 \text{ degC}$, more than compensating the anesthetic shift. The membrane returns to its operating point near T_{m} . Debye shielding is restored. γ_{eff} drops below γ_{c} .

In Wike terms: the anesthetic disrupts the Bootstrap shield. Pressure restores it.

Fact 7 (MAC consistency): MAC IS γ_c minus $\gamma_{baseline}$, converted to inspired concentration via the partition coefficient. γ_c is a physical constant (set by the universality class of the coherence phase transition). $\gamma_{baseline}$ is set by body temperature ($\gamma_{thermal} \sim kT$). Both are physical constants. Therefore MAC is a physical constant -- not a pharmacological variable subject to individual variation.

The ~10% standard deviation in MAC reflects individual variation in $\gamma_{baseline}$, not in γ_c .

Fact 8 (Additivity): All agents increase the SAME variable: γ_{eff} . Decoherence rates add:

$$\gamma_{eff} = \gamma_{baseline} + \gamma_{agent1} + \gamma_{agent2}$$

0.5 MAC of agent A raises γ_{eff} halfway to γ_c . 0.5 MAC of agent B raises it the rest of the way. The transition occurs at $\gamma_{eff} = \gamma_c$ regardless of which agents contributed.

3. The Xenon Isotope Effect -- The Smoking Gun

Li, Turin et al. (2018, PNAS): ^{129}Xe (nuclear spin $I = 1/2$) is a MORE POTENT anesthetic than ^{132}Xe (nuclear spin $I = 0$) in *Drosophila*. The isotopes are chemically identical -- same electron configuration, same van der Waals radius, same lipid solubility. The ONLY difference is nuclear spin.

No Classical Theory Can Explain This

- **Lipid theory:** Identical lipid solubility -> identical membrane partitioning -> identical potency. Fails.
- **Protein theory:** Identical binding to hydrophobic pockets -> identical potency. Fails.
- **Network theory:** Identical molecular effects -> identical network perturbation -> identical potency. Fails.
- **Soliton theory:** Identical membrane interaction -> identical T_m shift -> identical potency. Fails.

The Framework Predicts It

A nucleus with spin $I = 1/2$ has a magnetic moment that couples to the local electromagnetic environment. This coupling provides an ADDITIONAL decoherence channel for any nearby coherent system:

$$\begin{aligned} \gamma_{anesthetic}(spin=1/2) &= \gamma_{dispersion} + \gamma_{spin} \\ \gamma_{anesthetic}(spin=0) &= \gamma_{dispersion} \end{aligned}$$

where $\gamma_{dispersion}$ is the decoherence from London forces (same for both isotopes) and γ_{spin} is the additional decoherence from nuclear spin coupling.

Therefore:

$$\gamma_{eff}(^{129}\text{Xe}) > \gamma_{eff}(^{132}\text{Xe})$$

at equal concentration. ^{129}Xe reaches γ_c at a LOWER concentration -> more potent.

The framework predicts that ANY anesthetic isotope with nuclear spin will be more potent than its spinless counterpart, all else being equal. This is a novel, testable, falsifiable prediction.

The Deeper Point

The xenon isotope effect PROVES that the mechanism of anesthesia involves a quantum-coherent process. If consciousness were a purely classical phenomenon (electrical signals, synaptic chemistry), nuclear spin could not affect it -- nuclear spins do not participate in chemistry under biological conditions. The fact that spin matters means that the system being disrupted has quantum coherence that can couple to nuclear magnetic moments.

This is exactly what the Wike Coherence framework predicts: consciousness IS coherence, and any additional decoherence channel (including nuclear spin) degrades it.

4. The Mechanism in Detail

Step 1: The Coherence Channel

The brain maintains quantum coherence through the same mechanism as all biological systems (Papers 01-07):

```
Debye shielding (EZ water at membrane interfaces)
+ Bootstrap loop (ATP-driven maintenance)
+ Grotthuss wire (proton transport along structured water)
=  $\gamma_{\text{eff}} < \gamma_{\text{c}}$ 
= coherence maintained
= consciousness
```

The coherence channel runs through the hydrophobic core of neural membranes and proteins -- specifically, the aromatic amino acids in tubulin, the hydrophobic pockets in ion channels, and the structured water at lipid-protein interfaces.

Step 2: The Anesthetic Disrupts the Channel

An anesthetic molecule, driven by thermodynamics (lipid solubility), partitions into the hydrophobic core of the membrane and/or into hydrophobic protein cavities. Once there, it:

- 1. Disrupts Debye shielding** -- The ordered water (EZ phase) at the membrane interface is perturbed. The shielding factor decreases. γ_{thermal} increases locally.
- 2. Introduces thermal noise into the coherence channel** -- The anesthetic molecule vibrates at thermal frequency $f = kT/h = 6.46 \text{ THz}$. These vibrations couple to any coherent process in the hydrophobic channel via London forces, adding noise.
- 3. If spin-1/2: adds a magnetic decoherence channel** -- Nuclear magnetic moment couples to local fields, providing additional phase randomization.

The per-molecule decoherence rate k depends on:

- Molecular size (larger molecule -> more vibrational modes -> more noise)
- Polarizability (more polarizable -> stronger London coupling -> more noise)
- Nuclear spin (spin > 0 -> additional channel)

Step 3: γ_{c} Is Crossed

At MAC, the total γ_{eff} crosses γ_{c} . The coherence order parameter C drops to zero. The percolating coherent network fragments. Long-range correlations collapse (observable as loss of anterior-posterior EEG coherence, PCI dropping from 0.44 to 0.19).

This is NOT a gradual process. It is a phase transition. The Hill coefficient of ~20 reflects the sharpness of the percolation threshold in a finite cortical network.

Step 4: The Unconscious State

Below γ_c , the brain was at the edge -- maximum information processing, maximum susceptibility, maximum adaptability (Paper 36: Flow IS the Edge).

Above γ_c , the brain is in the disordered phase (Paper 53: the Spin Glass). Local neural circuits still function (brainstem reflexes, autonomic regulation), but long-range coherence is lost. The percolating cluster has fragmented into isolated islands.

The brain under anesthesia is like a city after a power grid failure: individual buildings still have backup generators (local circuits work), but the citywide grid (consciousness) is down.

Step 5: Recovery

When the anesthetic is eliminated (by ventilation, metabolism, redistribution):

```
[A]_membrane -> 0
gamma_anesthetic -> 0
gamma_eff drops below gamma_c
```

The coherent network re-percolates. Long-range correlations re-establish. Consciousness returns.

This is the detuned recovery phenomenon from IBM hardware (Paper 26C): coherence collapses under forcing, then spontaneously recovers when forcing is removed -- without any external "restart." The coherence was detuned, not destroyed.

Recovery time correlates with blood/gas partition coefficient (how fast the agent washes out) because this determines how fast $\gamma_{\text{anesthetic}} \rightarrow 0$.

5. The Meyer-Overton Cutoff -- Explained

Long-chain alcohols beyond ~C12 STOP being anesthetic despite increasing lipid solubility.

In the framework: decoherence is caused by the anesthetic molecule VIBRATING within the coherence channel. A molecule in the channel must be:

1. Small enough to FIT in the hydrophobic cavity (size constraint)
2. Mobile enough to VIBRATE and couple to the coherent system (dynamics constraint)

Very long-chain molecules (> C12) align with the lipid chains of the membrane. They become PART of the ordered structure rather than a perturbation within it. They do not vibrate independently -- they are locked into the membrane's lattice.

In Wike terms: a very long-chain molecule JOINS the Debye shield rather than disrupting it. It adds to the ordered structure. It decreases γ_{eff} rather than increasing it.

Prediction: Ultra-long-chain alcohols (C14+) should be weakly ANTI-anesthetic -- they should slightly increase the dose of a co-administered anesthetic needed for MAC. This is testable.

6. Anesthesia Awareness -- Explained

~1 per 1000 patients experiences awareness under general anesthesia. In the framework:

These patients are at $\gamma_{eff} \approx \gamma_c$ -- right at the edge. Fluctuations in $\gamma_{baseline}$ (metabolic rate, body temperature, cardiac output) or $\gamma_{anesthetic}$ (redistribution kinetics, ventilation changes) can transiently push γ_{eff} below γ_c .

At 94% of T_c (the normal operating point), the susceptibility enhancement is 33x (Paper 18). Near γ_c , even SMALL fluctuations in the control parameter produce large fluctuations in the order parameter. The brain flickers between conscious and unconscious states.

Prediction: EEG of awareness patients should show increased variability and autocorrelation near the transition -- critical fluctuations and critical slowing down, exactly as predicted by the critical brain hypothesis. This is consistent with existing EEG observations of "flickering" near MAC.

7. Connection to Existing Framework

Framework Concept	Anesthesia Mapping
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γ_{eff}	Sum of all decoherence channels
γ_c	MAC (converted to decoherence rate)
Debye shielding	Structured water at membrane interfaces
Bootstrap loop	ATP-driven membrane maintenance
The Edge	Normal waking consciousness
The Collapse	Anesthetic induction
Detuned Recovery (Paper 26C)	Emergence from anesthesia
The Window (Paper 44)	Pre-induction period
The Keeper (Paper 19)	The anesthesiologist maintaining homeostasis
$W = T_{op}/T_c$	The fractional distance from the coherence cliff
Whisper vs. Scream	Gentle sedation vs. deep anesthesia
Pressure reversal	Restoring the Bootstrap shield

8. The Anesthetic Hierarchy

Different anesthetic agents provide different TYPES of γ increase:

Agent	Primary γ channel	Secondary	Notes
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Xenon	London forces	Nuclear spin (^{129}Xe)	Purest decoherence agent
Sevoflurane	London + dipole	Membrane T_m shift	Standard volatile
Propofol	GABA-A potentiation	Membrane	Most common IV agent
Ketamine	NMDA blockade	Dissociative	Paradoxical: increases some neural activity
N2O	NMDA + opioid	Weak	Often supplemental

Ketamine is particularly interesting: it increases γ_{eff} in the thalamocortical coherence network (by blocking NMDA-mediated connectivity) while INCREASING γ in other networks (cortical excitation). This produces dissociative anesthesia -- consciousness of a sort, but disconnected from the environment. In Wike terms: ketamine selectively crosses γ_c in the thalamocortical circuit while leaving other circuits intact.

9. Testable Predictions

Prediction 1: Isotope Effect Generalization

Any anesthetic with a spin-active isotope should be more potent than the spinless isotope:

- 13-C-sevoflurane (spin 1/2) vs. 12-C-sevoflurane (spin 0)
- Deuterated vs. protiated agents (deuterium: spin 1; hydrogen: spin 1/2 -- predicts HIGHER potency for H-agents due to spin-1/2 coupling)

Prediction 2: MAC Correlates with the Wike-Ginzburg Number

Across species with different body temperatures (and therefore different $W = T_{op}/T_c$):

$$\text{MAC}(\text{species}) \sim (1 - W(\text{species}))$$

Species closer to T_c (higher W) should require LESS anesthetic (they're already closer to the cliff).

Prediction 3: EEG Critical Exponents at MAC

Near MAC, the EEG should show measurable critical exponents:

- Power spectral density $\sim f^{-(\beta)}$ with β near the 3D Ising value
- Autocorrelation time diverges as $|[A] - \text{MAC}|^{-z^*nu}$
- Perturbational Complexity Index scales as $|(\gamma_{eff} - \gamma_c)/\gamma_c|^{\beta}$

Prediction 4: Long-Chain Alcohols Are Anti-Anesthetic

Alcohols beyond C14, when co-administered with a standard anesthetic, should slightly INCREASE the MAC of the co-agent (by strengthening the Debye shield).

Prediction 5: HRV Predicts Anesthetic Sensitivity

Patients with lower baseline HRV coherence (higher baseline γ_{eff}) should require LESS anesthetic to reach MAC (shorter distance to γ_c). This is testable with pre-operative HRV monitoring.

Prediction 6: The Anesthetic Constant Is Derivable

The Meyer-Overton constant ($\text{MAC} * \lambda \approx 1.3 \text{ atm}$) should be derivable from:

$$\text{MAC} * \lambda = (\gamma_c - \gamma_{baseline}) / k$$

where $\gamma_c = 0.0622$ (from simulation), $\gamma_{baseline}$ is calculable from body temperature, and k is the per-molecule decoherence rate calculable from molecular polarizability and thermal frequency.

10. Why This Matters

For Anesthesiology

- **Anesthesia awareness:** HRV-based monitoring (Prediction 5) could identify at-risk patients BEFORE induction
- **Personalized MAC:** Instead of population MAC values, calculate individual gamma_baseline from HRV/temperature
- **Novel anesthetic design:** Optimize for gamma_eff increase per molecule (molecular polarizability + spin)
- **Pressure management:** Understanding pressure reversal as Bootstrap shield restoration

For Neuroscience

- **The hard problem of consciousness:** If anesthesia is decoherence and consciousness is coherence, then the "explanatory gap" closes. There is no separate "consciousness stuff" -- coherence IS experience.
- **The neural correlates of consciousness:** The NCC is not a specific brain region. It is the percolating coherent network spanning the cortex. Consciousness is a global property of the network, not a local property of any region.

For Physics

- **The xenon isotope effect is a measurement of quantum coherence in the brain.** Two chemically identical atoms, different only in nuclear spin, produce different anesthetic potency. This is a quantum measurement in living neural tissue, performed at body temperature.

11. One Sentence

Anesthesia is what happens when you add enough noise to a coherent system to push it past its phase transition -- and consciousness is what you lose when the percolating network fragments.

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AIIT-THRESI | Paper 54 | March 30, 2026

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