LOG ID	BOOK REFERENCE	RCT FILTER	CONFIDENCE TIER
SEC 2.7.1	CHAPTER 2: OPERATING SYSTEM (Narrative Layer)	OFF	CONSENSUS

AUDITED CLAIM

DIAGNOSTIC FORK: FRICTION VS. DEPLETION (PATH B)

SYSTEMS MODEL — AS STATED IN THE BOOK

> Your motherboard is actively engaging a system-wide **autonomic brake**, enforcing defensive immobility to prevent catastrophic **hardware damage**.

VALIDATION QUERY

Do severe trauma-induced freeze states or extreme sympathetic burnout involve the active neurological inhibition of the motor cortex?

EMPIRICAL FINDING

Polyvagal and autonomic nervous system models clarify that severe trauma-related freeze states are not passive neurological shutdowns, but active states of **defensive immobility**. The nervous system engages a massive autonomic brake to force stillness, meaning aggressive attempts to forcefully override the state trigger deeper **physiological resistance**.

PRIMARY ANCHORS

→ **Roelofs, 2017, Philosophical Transactions of the Royal Society B**

→ **Hermans et al., 2013, NeuroImage**

ABOUT THIS RECORD

Each claim in the book is decompiled from its IT metaphor into a naked, falsifiable scientific claim, then anchored to a **primary peer-reviewed source**. Claim and anchor are queried against the Consensus academic database (200M+ papers) under a fixed protocol — top-tier **Q1/Q2 journals only**, preprints excluded, human studies only. If a hypothesis fails the audit, the build is rejected: the AI generates the syntax; the peer-reviewed grid validates the physics.

CONFIDENCE TIERS

- **CONSENSUS ◀ this log** — broadly established, highly replicated mechanics.
- **EMERGING** — plausible, actively researched; no universal consensus yet.
- **EXPERIMENTAL** — contested, extrapolated, or purely theoretical.

RCT FILTER — Controlled Studies is ON for interventions (a forced action must beat a control) and OFF for mechanisms (so foundational neuroscience isn't blocked).

THE DREAM MACHINE

OPERATING SYSTEM FOR THE SOUL

You are complex biological hardware running outdated psychological software. *The Dream Machine* translates rigorous IT architecture — security auditing, network protocols, load balancing — into actionable, peer-reviewed models for mental resilience: debug burnout, configure a hallucination firewall against cognitive distortions, and patch your internal OS with hard logic and verified neuroscience.

INITIATE SYSTEM UPDATE

Ready to debug your own source code? Scan to access the cybernetic manual and reboot your cognitive architecture today. → **dreammachine.systems**

Yes, **trauma-related freeze states** often involve **motor suppression**, but **direct motor cortex inhibition** remains unproven.

1. Introduction

Severe trauma-induced freeze states are consistently described as active defensive immobility, but the best-supported mechanism is a distributed brainstem–autonomic brake rather than a proven, direct shutdown of primary motor cortex. Human freezing studies link threat-related bradycardia and immobility to amygdala–periaqueductal gray circuitry, supplementary motor regions, and frontal inhibitory areas, while reviews explicitly note that whether cortical motor areas are causally inhibited during freezing remains unresolved (Roelofs, 2017; Hermans et al., 2013).

The answer is different for “extreme sympathetic burnout,” because the closest clinical analogues in this corpus are paroxysmal sympathetic hyperactivity, chronic stress, PTSD-related autonomic imbalance, and burnout. Those literatures do not show active inhibition of motor cortex. Instead, they emphasize loss of descending inhibitory control over sympathetic centers, persistent autonomic dysregulation, excitotoxic stress biology, and altered corticolimbic or prefrontal regulation (Zheng et al., 2020; Bayes et al., 2021; Fu, 2021; Hosseini-Kamkar et al., 2023).

Direct evidence for active motor-cortex inhibition does exist in adjacent motor-stopping paradigms: human TMS work shows that stopping actions can actively suppress corticospinal output, and emotional threat cues can lower primary motor cortex activity or suppress intracortical facilitation. But those studies examine laboratory stopping or emotional modulation, not severe trauma freeze states themselves (Kujirai et al., 1993; Duque et al., 2017; Sagaspe et al., 2011; Roelofs, 2017).

Do severe trauma-induced freeze states or extreme sympathetic burnout involve the active neurological inhibition of the motor cortex?
Requires at least 5 papers that directly answer your question. Try adjusting your query to find more papers.

FIGURE 1 Consensus on motor cortex inhibition in freeze states

The meter should be read as mixed-to-leaning yes for motor suppression broadly, but not for direct inhibition of primary motor cortex specifically. The strongest evidence supports network-level braking and reduced motor output during freezing, whereas the specific claim of active M1 inhibition remains indirect or explicitly unresolved (Roelofs, 2017; Topka et al., 2022; Duque et al., 2017).

2. Methods

The search ran over more than 170 million research papers indexed in Consensus, drawing from Semantic Scholar, PubMed, and other sources. The workflow identified 21,015 initially relevant papers, expanded through targeted searches and citation crawling, screened 238 papers with machine-learned relevance filtering, judged 158 eligible after deduplication and thresholding, and included the top 50 most relevant papers for synthesis.

Search Strategy

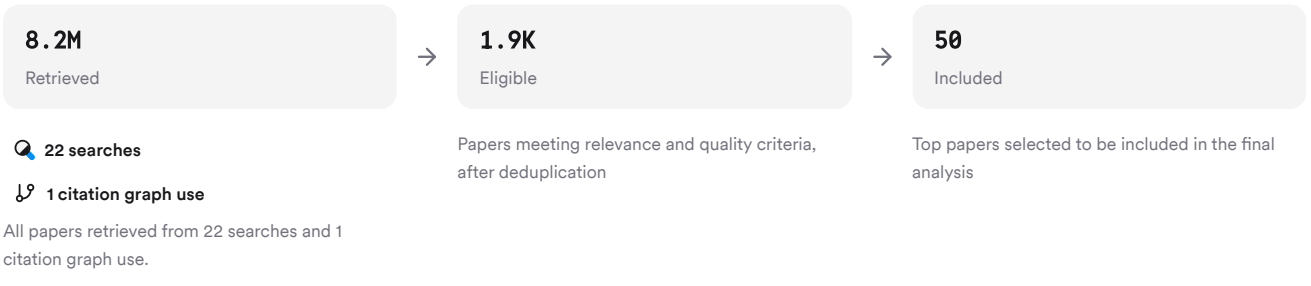

FIGURE 2 Deep search screening and inclusion workflow

Searches combined trauma freezing, motor inhibition, autonomic dysfunction, dissociation, bradykinesia, and null-result terms across human neuroimaging, physiology, and clinical neurology.

3. Results

3.1 Key Papers

Three papers anchor this literature because they directly address the mechanism of freezing, link human freezing to specific neural circuits, and test whether motor cortex excitability changes during freezing-like motor arrest (Roelofs, 2017; Hermans et al., 2013; Topka et al., 2022).

Paper	Summary
(Roelofs, 2017)	Human freezing model; motor-cortex role unresolved (Roelofs, 2017)
(Cockx et al., 2024)	PAG-linked bradycardia with motor-inhibition regions (Hermans et al., 2013)
(Speers et al., 2021)	Reduced M1 excitability during freezing, mechanism unclear (Topka et al., 2022)

FIGURE 3 Foundational papers on freezing and motor inhibition

3.2 Freezing Circuits

Human and animal-informed reviews converge on freezing as an active state in which the amygdala engages ventrolateral PAG pathways that impose immobility while preserving high muscle tone and sympathetic readiness (Kozlowska et al., 2015; Roelofs & Dayan, 2022).

This is not simple collapse. Freezing combines parasympathetic bradycardia with persistent sympathetic components such as muscle tone, pupil dilation, and skin conductance, producing a poised “brake” rather than generalized shutdown (Kozlowska et al., 2015; Roelofs & Dayan, 2022).

Recent human work strengthens the view that freezing-like states optimize perception and action preparation. Avoidable-threat paradigms show bradycardia, gaze centralization, sympathetic arousal, and parieto-occipital alpha suppression that predicts faster later responses, consistent with attentive immobility rather than cortical quiescence (Stegmann et al., 2024).

3.3 Motor Cortex Evidence

Dimension	Trauma/freezing literature	Stopping literature	Parkinsonian freezing literature	Citations
Primary claim	Motor suppression is inferred	Active inhibition is established	M1 excitability often falls	(Roelofs, 2017; Duque et al., 2017; Topka et al., 2022)
Direct M1 measure	Rare	Common with TMS	Present but mixed mechanistically	(Roelofs, 2017; Duque et al., 2017; Topka et al., 2022)
Cortical areas implicated	SMA, IFG, PAG-linked regions	rIFC, preSMA, STN, motor thalamus	SMA, PFC, DLPFC, M1	(Roelofs, 2017; Duque et al., 2017; Cockx et al., 2024; Taniguchi et al., 2024)
Interpretation	Network brake	Active global inhibitory process	Hypoexcitable or imbalanced state	(Roelofs, 2017; Duque et al., 2017; Topka et al., 2022; Cockx et al., 2024)
Main caveat	Causality unresolved	Lab task, not trauma freeze	Disease model, not trauma	(Roelofs, 2017; Duque et al., 2017; Topka et al., 2022)

FIGURE 4 Comparison of motor inhibition across related literatures

Emotional threat can directly modulate motor cortex physiology. Viewing fearful bodies suppresses intracortical facilitation of primary motor cortex, and fearful stop-signal trials show lower primary motor cortex activity during failed stopping, suggesting emotional signals can engage motor-suppressive processes (Roelofs, 2017; Sagaspe et al., 2011; Battaglia et al., 2021).

But the closest direct test of freezing itself is more cautious. In Parkinson-related freezing of upper-limb movement, M1 excitability was reduced during freezing, yet the authors state they could not determine whether this reflected active inhibition or reduced excitatory input, and they even suggest activation failure may be more likely (Topka et al., 2022).

3.4 Sympathetic Overdrive and Burnout

The sympathetic-overdrive literature points away from motor-cortex inhibition as the core mechanism. In paroxysmal sympathetic hyperactivity after brain injury, the dominant model is loss of inhibitory control over excitatory autonomic centers due to disconnection of descending pathways, not active inhibition of motor cortex (Meyfroidt et al., 2017; Zheng et al., 2020).

Burnout studies likewise emphasize sustained autonomic activation, sympathetic-adrenal-medullary dysfunction, cortisol alteration, inflammation, and possible excitotoxicity, but they do not identify a motor-cortical inhibitory program as the mechanism of exhaustion (Bayes et al., 2021).

PTSD and severe adversity show altered autonomic balance and blunted prefrontal responses rather than clear evidence of active M1 inhibition. Many PTSD studies report increased sympathetic and reduced parasympathetic activity, and meta-analytic fMRI data show heightened amygdala reactivity with lower prefrontal cortical reactivity under challenge (Fu, 2021; Hosseini-Kamkar et al., 2023).

Results Timeline

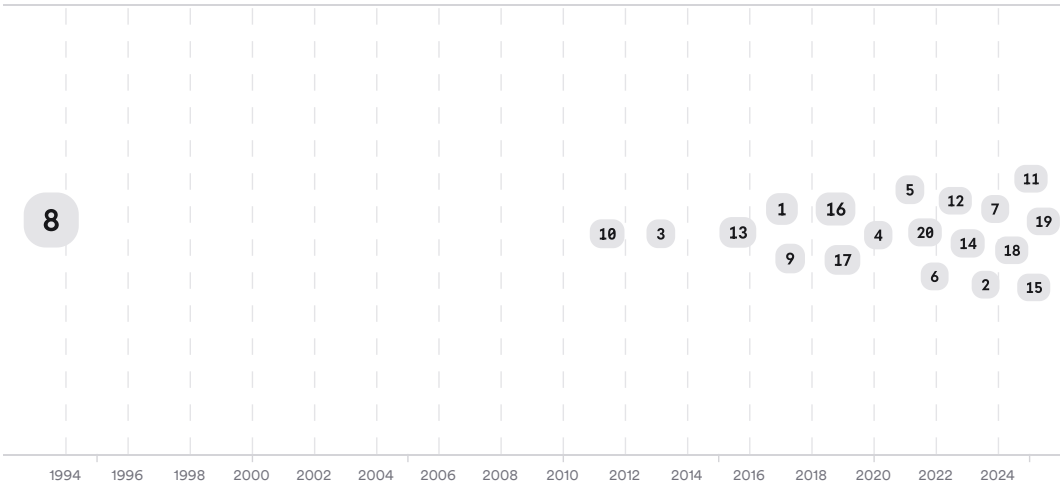

FIGURE 5 Timeline of freezing and inhibition studies, larger markers indicate more citations

The timeline shows an early phase establishing cortical motor inhibition in generic stopping, a middle phase mapping human freezing onto PAG-centered defense circuits, and a recent phase testing whether M1, SMA, and prefrontal nodes are specifically altered during freezing states (Kujirai et al., 1993; Hermans et al., 2013; Topka et al., 2022).

Top Contributors

Type	Name	Papers
Author	Karin Roelofs	[5fe1432fd1695f009ee33c8370d2bc35][24a8408a1b8c5fb39919b6c8d95185f2] [20f2b4f8b9c4575f9fce6a47110468bb]
Author	R. J. A. van Wezel	[316101ecc701559d99004618f8f29da5]
Author	M. Hallett	[c9f12548bf8551b68425ec83df64024b][9521734ca9b35344b737809ffa95e6e6]

Type	Name	Papers
Journal	*NeuroImage*	[a1dbf266c0fc567cbe1fef2a8071d234][20f2b4f8b9c4575f9fce6a47110468bb] [1cbe91b7ef51544a94f719e342d4af66][f0a236bd22a15381a13e3b3236b5c6b4]
Journal	*Movement Disorders*	[8e5daa9251bb531cb8a01b4d4ee0f6c8][6f4a25c22f9853d2a091fa78c0e889a9]
Journal	*Frontiers in Neurology*	[77b754a0943c5a7eb7fc6fbea5b9118f][d47e329f2ffd57c280d75b40c6ed6578]

FIGURE 6 Authors and journals most represented in included papers

4. Discussion

The strongest part of this corpus is the convergence on freezing as an active defensive state with identifiable autonomic and subcortical circuitry. Multiple reviews and human neuroimaging studies independently place amygdala–PAG pathways, vagal engagement, and frontal/supplementary motor regions at the center of freezing, and newer electrophysiology suggests this state improves sensory sampling and action readiness rather than simply abolishing movement (Roelofs, 2017; Roelofs & Dayan, 2022; Hermans et al., 2013; Stegmann et al., 2024).

The weak point is the user's most specific clause: "active neurological inhibition of the motor cortex." The literature repeatedly hints at it, because emotional cues suppress M1-related facilitation, stop-signal paradigms show true active global motor inhibition, and clinical freezing states often show reduced cortical excitability or stopping-network imbalance (Roelofs, 2017; Duque et al., 2017; Topka et al., 2022; Cockx et al., 2024). But the freeze literature itself still labels cortical motor involvement unresolved, and the most direct M1 paper explicitly cannot separate active inhibition from loss of excitatory drive (Roelofs, 2017; Topka et al., 2022).

The "extreme sympathetic burnout" half of the question is even less supportive of an M1-inhibition model. PSH, PTSD autonomic dysfunction, and burnout all point to failed autonomic regulation, stress-axis dysregulation, and loss of inhibitory control over sympathetic centers, not to an adaptive motor-cortex braking mechanism (Zheng et al., 2020; Meyfroidt et al., 2017; Bayes et al., 2021; Fu, 2021).

Claim	Evidence Strength	Reasoning	Papers
Trauma-related freezing is an **active defensive immobility state** rather than passive shutdown.	Strong	Supported by convergent reviews, human imaging, and autonomic physiology.	(Roelofs, 2017; Kozlowska et al., 2015; Stegmann et al., 2024)
Freezing relies primarily on **amygdala–PAG–autonomic circuitry** with frontal motor-network involvement.	Strong	Replicated across reviews and human neuroimaging, but cortical causality remains incomplete.	(Roelofs, 2017; Hermans et al., 2013; Roelofs & Dayan, 2022)
Direct **active inhibition of primary motor cortex** during trauma freeze is established.	Weak	Adjacent evidence supports plausibility, but direct proof in trauma freezing is lacking.	(Roelofs, 2017; Topka et al., 2022; Duque et al., 2017)
Emotional threat cues can **suppress motor-cortex excitability or output**.	Moderate	Experimental TMS and fMRI studies show threat-linked M1 suppression in laboratory tasks.	(Roelofs, 2017; Sagaspe et al., 2011; Battaglia et al., 2021)

Claim	Evidence Strength	Reasoning	Papers
"Sympathetic burnout" is chiefly a **motor-cortex inhibitory state**.	Weak	Burnout and PSH literatures emphasize autonomic dysregulation, not M1 inhibition.	(Bayes et al., 2021; Meyfroidt et al., 2017)

FIGURE 7 Key claims and evidence across included papers

For personal symptoms or trauma-related episodes, a licensed clinician should evaluate possible neurological, psychiatric, autonomic, and functional causes; this is a research synthesis, not medical advice.

5. Conclusion

Across these 50 papers, severe trauma-related freeze states do appear to involve active neural braking of movement, but the evidence points most strongly to a distributed defense network centered on amygdala, PAG, autonomic nuclei, and frontal motor-control regions rather than a proven direct inhibition of primary motor cortex. By contrast, the literature on sympathetic overdrive, PSH, PTSD autonomic dysfunction, and burnout does not support a specific M1-inhibition account.

Research Gaps

The main gap is mechanistic resolution: studies show reduced movement and sometimes reduced M1 excitability, but they rarely distinguish active intracortical inhibition from reduced excitatory drive, basal-ganglia gating, brainstem premotor suppression, or dissociative motor disconnection (Topka et al., 2022; Roelofs, 2017; Abrams et al., 2009).

Topic/Outcome	Direct M1 physiology	Trauma-specific samples	Longitudinal data	Mechanistic causal tests
Defensive freezing	2	4	1	1
Tonic immobility / dissociation	GAP	3	1	GAP
Sympathetic hyperactivity after injury	GAP	4	2	1
Burnout / chronic stress exhaustion	GAP	1	2	GAP

Open Research Questions

Future work needs experiments that combine trauma-relevant threat paradigms with TMS, EMG, autonomic recording, and causal perturbation of frontal–subthalamic or PAG-linked circuits.

Question	Why
During human trauma-related freezing, is reduced movement caused by active intracortical M1 inhibition or by loss of excitatory drive from upstream networks?	This is the key unresolved mechanistic distinction repeatedly identified by the current corpus.
Do dissociative tonic immobility states and PAG-mediated defensive freezing share the same motor-control circuitry?	Similar behavior may mask distinct mechanisms, which matters for classification and treatment development.

Question	Why
Can simultaneous autonomic and motor-cortex physiology predict who shifts from adaptive freezing into pathological post-trauma motor shutdown?	Prospective biomarkers could separate transient defense from clinically persistent dysfunction.

The literature supports active motor suppression in trauma-related freezing, but not a settled claim of direct primary motor cortex inhibition, and it does not support that model for sympathetic burnout.

References

Abrams, M. P., Carleton, N. R., Taylor, S., & Asmundson, G. (2009). Human tonic immobility: measurement and correlates. *Depression and Anxiety, 26*. https://doi.org/10.1002/da.20462

Battaglia, S., Serio, G., Scarpazza, C., D'Ausilio, A., & Borgomaneri, S. (2021). Frozen in (e)motion: How reactive motor inhibition is influenced by the emotional content of stimuli in healthy and psychiatric populations.. *Behaviour research and therapy, 146*, 103963. https://doi.org/10.1016/j.brat.2021.103963

Bayes, A., Tavella, G., & Parker, G. (2021). The biology of burnout: Causes and consequences. *The World Journal of Biological Psychiatry, 22*, 686 - 698. https://doi.org/10.1080/15622975.2021.1907713

Cockx, H., Oostenveld, R., R, Y. F. A., Bloem, B. R., Cameron, I. G. M., & Van Wezel, R. J. A. (2024). Freezing of gait in Parkinson's disease is related to imbalanced stopping–related cortical activity. *Brain Communications, 6*. https://doi.org/10.1093/braincomms/fcae259

Duque, J., Greenhouse, I., Labruna, L., & Ivry, R. (2017). Physiological markers of motor inhibition during human behavior. *Trends in neurosciences, 40*, 219 - 236. https://doi.org/10.1016/j.tins.2017.02.006

Fu, Q. (2021). Autonomic dysfunction and cardiovascular risk in post-traumatic stress disorder.. *Autonomic neuroscience : basic & clinical, 237*, 102923. https://doi.org/10.1016/j.autneu.2021.102923

Hermans, E., Henckens, M., Roelofs, K., & Fernández, G. (2013). Fear bradycardia and activation of the human periaqueductal grey. *NeuroImage, 66*, 278-87. https://doi.org/10.1016/j.neuroimage.2012.10.063

Hosseini-Kamkar, N., Farahani, M. V., Nikolić, M., Stewart, K. A., Goldsmith, S. F., Soltaninejad, M., Rajabli, R., Lowe, C. J., Nicholson, A. A., Morton, J. B., & Leyton, M. (2023). Adverse Life Experiences and Brain Function. *JAMA Network Open, 6*. https://doi.org/10.1001/jamanetworkopen.2023.40018

Kozlowska, K., Walker, P., Mclean, L. M., & Carrive, P. (2015). Fear and the Defense Cascade: Clinical Implications and Management. *Harvard Review of Psychiatry, 23*, 263 - 287. https://doi.org/10.1097/hrp.0000000000000065

Kujirai, T., Caramia, M., Rothwell, J., Day, B., Thompson, P., Ferbert, A., Wroe, S., Asselman, P., & Marsden, C. (1993). Corticocortical inhibition in human motor cortex.. *The Journal of Physiology, 471*. https://doi.org/10.1113/jphysiol.1993.sp019912

Meyfroidt, G., Baguley, I., & Menon, D. (2017). Paroxysmal sympathetic hyperactivity: the storm after acute brain injury.. *The Lancet. Neurology, 16 9*, 721-729. https://doi.org/10.1016/s1474-4422(17)30259-4

Roelofs, K. (2017). Freeze for action: neurobiological mechanisms in animal and human freezing. *Philosophical Transactions of the Royal Society B: Biological Sciences, 372*. https://doi.org/10.1098/rstb.2016.0206

Roelofs, K., & Dayan, P. (2022). Freezing revisited: coordinated autonomic and central optimization of threat coping. *Nature Reviews Neuroscience, 23*, 568 - 580. https://doi.org/10.1038/s41583-022-00608-2

Sagaspe, P., Schwartz, S., & Vuilleumier, P. (2011). Fear and stop: A role for the amygdala in motor inhibition by emotional signals. *NeuroImage, 55 4*, 1825-35. https://doi.org/10.1016/j.neuroimage.2011.01.027

Speers, A. B., Cabey, K., Soumyanath, A., & Wright, K. (2021). Effects of Withania somnifera (Ashwagandha) on Stress and the Stress-Related Neuropsychiatric Disorders Anxiety, Depression, and Insomnia. *Current Neuropharmacology, 19*, 1468 - 1495. https://doi.org/10.2174/1570159x19666210712151556

Stegmann, Y., Teigeler, J., Mirifar, A., Keil, A., & Gamer, M. (2024). Electrocortical Responses in Anticipation of Avoidable and Inevitable Threats: A Multisite Study. *The Journal of Neuroscience, 44*. https://doi.org/10.1523/jneurosci.0575-24.2024

Taniguchi, S., Kajiyama, Y., Kochiyama, T., Revankar, G., Ogawa, K., Shirahata, E., Asai, K., Saeki, C., Ozono, T., Kimura, Y., Ikenaka, K., D'cruz, N., Gilat, M., Nieuwboer, A., & Mochizuki, H. (2024). New Insights into Freezing of Gait in Parkinson's Disease from Spectral Dynamic Causal Modeling. *Movement Disorders, 39*. https://doi.org/10.1002/mds.29988

Topka, M., Schneider, M., Zrenner, C., Belardinelli, P., Ziemann, U., & Weiss, D. (2022). Motor cortex excitability is reduced during freezing of upper limb movement in Parkinson's disease. *NPJ Parkinson's Disease, 8*. https://doi.org/10.1038/s41531-022-00420-w

Zheng, R.-Z., Lei, Z.-Q., Yang, R.-Z., Huang, G.-H., & Zhang, G. (2020). Identification and Management of Paroxysmal Sympathetic Hyperactivity After Traumatic Brain Injury. *Frontiers in Neurology, 11*. https://doi.org/10.3389/fneur.2020.00081