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# Dynamic Network Disruption under Cognitive Strain: Coupling of Local Node Failure and Global Edge Integrity Reveals Differential Systemic Resilience

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## ABSTRACT

Objective: To investigate how systemic resilience to cognitive stress differs between Normal and Abnormal individuals by analyzing the dynamic coupling between local Resource Management (Node Status) and global Information Flow (Edge Function) in the brain's functional network. Methods: We analyzed 19-channel EEG data during a 12-question arithmetic task 30 epochs). CAR pre-processing was used. Node Status (resource depletion) was traced via Frontal Alpha Power (µV2) at Fz. Edge Function (systemic integrity) was quantified by the Arousal Index and Alpha/Theta Ratio (ATR).
Results: The Normal network exhibited acute vulnerability: Edge Integrity (Arousal) dropped 28.85% Q1 - Q11, coupled to an acute 76.47% deactivation of the Fz Node. The Abnormal network maintained a relatively stable Fz node despite a larger overall reduction in vigilance ATR - 44.00%. This reveals two distinct systemic failure modes.
Conclusion: Differences in systemic resilience are determined by resource management architecture. The Normal network's high-risk strategy leads to rapid, acute systemic failure—a phenomenon analogous to non-compensated circulatory collapse.

**KEYWORDS**: Neuro Feedback, Eeg Neuro Analytics, Network Analysis, Machine Learning, Cognitive Resilience, Arousal Index.

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## INTRODUCTION

The brain, like the vascular system, is not merely a collection of discrete parts but a living network, whose survival depends on its capacity to redistribute stress. When cognitive demand rises, the system must balance local resource consumption with global efficiency. This equilibrium—between nodal activity and systemic coherence—defines systemic resilience. Under chronic or intense strain, that balance can fracture, producing cascading network failures reminiscent of ischemic phenomena in vascular systems.

Neuroscientific literature increasingly views the brain as a complex adaptive system, where performance emerges from interdependent modules that communicate across spatial and temporal scales (Bassett & Sporns, 2017; Deco et al., 2021). These networks continuously reconfigure to sustain performance under load, a property termed functional resilience. Yet, resilience is not uniform across individuals or contexts. Some systems collapse abruptly, while others sustain coherence through redistribution—a contrast mirrored in vascular physiology between brittle and collateralized flow patterns.

### Theoretical and Practical Problem

The ideal cognitive system maintains homeostatic balance: when demand rises, it upregulates global arousal and redistributes nodal load proportionally. In practice, however, this coupling often fails. Certain systems exhibit excessive local recruitment—overactivation of frontal regions—leading to resource exhaustion and desynchronization of the global network (Nahas et. al., 2017). Others exhibit systemic stability through moderated engagement, preserving performance longer despite apparent reductions in vigilance. Understanding what distinguishes these systemic patterns is essential for diagnosing resilience failures in both cognitive and physiological systems.

### Why existing studies fall short

Past studies have examined single-dimensional EEG markers such as alpha suppression (Klimesch, 2012) or theta enhancement

(Kok A., 2022, Cocchi et al., 2013) as indicators of mental fatigue. While informative, they rarely capture coupling behavior—how changes in one subsystem affect others dynamically. Static analyses obscure the cascading dependencies that characterize systemic failure. Similarly, traditional vascular models of autoregulation measure pressure and flow but not the adaptive timing between local and global responses. In both domains, the missing insight lies not in the magnitude of change but in the relationship between local and systemic trajectories over time.

**Consequences of the Problem**

When local-global coupling deteriorates, the brain loses efficiency in resource distribution. The immediate consequence is reduced cognitive endurance—faster fatigue, impaired vigilance, and decreased adaptability. Indirectly, such dysfunction may echo across systemic physiology: dysautonomia, cerebral perfusion deficits, and even microvascular inefficiencies share the same architectural logic of failure. The issues of loss of brain efficiency can lead to numbers of health risk associated with working and working condition (Syazwan et al., 2012a, 2012b & Syazwan et al., 2013). Hence, cognitive and vascular fragility may be two expressions of the same underlying network principle.

**Knowledge Gap and Direction of the Study**

The current research addresses this critical gap by quantifying the temporal coupling between local nodal dynamics (Fz Alpha) and global systemic integrity (Arousal Index and ATR) during progressive cognitive strain. This study goes beyond static averages to analyze trajectories—the evolving signatures of systemic adaptation and breakdown. We test whether Normal and Abnormal profiles reveal different systemic architectures, hypothesizing that Normal systems employ high-gain but brittle strategies, whereas Abnormal systems exhibit distributed, resilient configurations.

**Objectives of the Study:**

1. To quantify dynamic trajectories of Arousal Index, ATR, and Fz Alpha across cognitive epochs.
2. To compare the local–global coupling patterns between Normal and Abnormal profiles.
3. To conceptualize these differences through a translational vascular analogy of systemic resilience.

## LITERATURE REVIEW

The concept of systemic resilience has evolved across neuroscience and physiology as a unifying principle of adaptive stability. Network neuroscience defines resilience as the capacity of the brain's connectome to maintain efficient communication despite localized perturbations (Bassett & Sporns, 2017). Similarly, vascular biology defines resilience as the ability of circulatory networks to preserve perfusion under fluctuating load. Both frameworks revolve around the same principle: maintaining coupling integrity between local activity and global flow.

Cognitive control networks, particularly the frontal-parietal system, modulate this balance through oscillatory coordination. Klimesch (2012) describes alpha rhythms as gatekeepers of cortical information flow, regulating access to working memory. Alpha suppression denotes engagement, while alpha enhancement indicates withdrawal or fatigue. Theta oscillations, by contrast, rise with cognitive effort, supporting synchronization across distant nodes (Nahas et al., 2017). The Alpha/Theta Ratio (ATR) thus serves as a valuable index of vigilance: declining ATR marks mental fatigue and diminished top-down regulation (Kunasegaran et al., 2023).

Recent advances in dynamic network analysis emphasize time-varying connectivity. Finc et al. (2020) demonstrated that functional networks reconfigure their modular topology during working memory training, reflecting flexible adaptation. Deco et al. (2021) extended this to show that brain networks operate near criticality—balancing integration and segregation for optimal resilience. However, under sustained strain, that balance drifts toward rigidity, reducing adaptability.

In vascular systems, a similar principle governs autoregulatory control: maintaining constant flow despite pressure changes. Failure of local compensation leads to systemic instability. This analogy between neural activation and vascular perfusion is more than metaphorical; both depend on distributed feedback control governed by local–global interplay.

Despite these parallels, few studies have directly quantified the coupling between local and systemic indicators in neural data. Most treat alpha, theta, or beta power independently rather than examining their dynamic ratio across nodes. Furthermore, prior resilience models have focused on recovery after perturbation rather than the *trajectory toward failure* itself.

The present study uniquely situates cognitive network dynamics within a translational systemic framework, offering a quantitative bridge between neural and vascular regulation.

## METHODS

### 2.1 Research Design and Conceptual Framework

This study was conceived as a comparative systems analysis designed to probe how distinct neurophysiological architectures manage cognitive strain. Instead of treating EEG activity as a static signal, the analysis interprets it as a living system that redistributes load—much like vascular flow adjusting to pressure gradients. By adopting this translational lens, the methodology is not merely technical; it is diagnostic of resilience itself. Each analytic step was therefore selected not for computational convenience but for its conceptual alignment with systemic regulation theory.

The design followed a dual-case comparative model, contrasting a Normal network (7ar0) and an Abnormal network (7ar1). Both datasets were drawn from arithmetic reasoning sessions consisting of twelve structured questions (Q1–Q12), each approximately 30 seconds in duration. This task was intentionally chosen for its ability to produce sustained cognitive load—a mild but progressive stressor that mirrors hemodynamic load in vascular systems. Each "question" served as an epochal marker of systemic response to an incremental stress step.

**2.2 Ethical Approval**

This study was conducted in accordance with the ethical standards of the International Islamic University Malaysia Research Ethics Committee (IREC) and received approval under IREC ID 2023-196. The broader project, titled *EEG Assessment and Neurofeedback Therapy*, aims to enhance cognitive well-being by developing personalized neuroregulatory interventions. All participants or their legal guardians provided written informed consent, and child participants gave verbal assent after an age-appropriate explanation of procedures. EEG acquisition was non-invasive, with minimal risk aside from mild, temporary discomfort during electrode placement. Data were fully anonymized and stored securely in compliance with institutional data protection policies.

**2.3 EEG Acquisition and Temporal Segmentation**

EEG data were collected across nineteen standard 10–20 system electrodes: Fp1, Fp2, F3, F4, F7, F8, Fz, Cz, C3, C4, P3, P4, P7, P8, Pz, O1, O2, T7, and T8. Sampling frequency was fixed at 250 Hz, balancing temporal resolution with noise suppression. This configuration ensured adequate sensitivity to capture dynamic redistribution of spectral power, analogous to how high-frame-rate vascular imaging resolves pulsatile flow.

Recordings were divided into twelve epochs, each representing a 30-second task interval corresponding to one arithmetic question. The epoch length was chosen based on empirical evidence suggesting that cortical fatigue and spectral redistribution manifest over 20–40-second windows (Kok A., 2022). The 30-second segmentation thus preserved temporal resolution while maintaining quasi-stationarity—an essential condition for reliable spectral estimation.

**2.4 Signal Preprocessing: Common Average Reference and Elliptic Filtering**

Raw EEG signals were first normalized using a Common Average Reference (CAR) technique, subtracting the instantaneous mean voltage across all channels from each individual channel. CAR reduces volume conduction artifacts and stabilizes the global baseline, analogous to systemic autoregulation isolating local perfusion from overall pressure drift.

Next, an elliptic (Ellipord) digital filter was applied to improve signal-to-noise ratio and eliminate physiological and environmental noise. The elliptic filter was chosen for its steep roll-off characteristics and computational efficiency—yielding high selectivity while minimizing phase distortion. Studies have demonstrated that elliptic filters achieve superior noise suppression and computational speed in EEG pipelines compared to Butterworth and Chebyshev alternatives (Pillalamarri & Shanmugam, 2025). The BrainMarker EXG acquisition software's built-in high-pass filter automatically managed DC offset removal, ensuring baseline stability prior to spectral decomposition.

**2.5 Spectral Decomposition and Systemic Flow Dynamics**

Power spectral density (PSD) analysis was performed using Welch's method with 50% overlap and a 2048-point Hanning window. This method provides robust frequency-domain estimation and minimizes the influence of transient artifacts. EEG activity was decomposed into canonical frequency bands, each representing a functional tier of systemic regulation:

- Theta (4–8 Hz): baseline vigilance and sustained attention.
- Alpha (8–13 Hz): inhibitory control and resource management.
- Beta (13–30 Hz): cognitive engagement and task-driven integration.
- Gamma (30–45 Hz): global coherence and top-down synchronization.

These bands collectively mirror multiscale vascular control systems, where microcirculatory feedback corresponds to local theta–alpha dynamics and macrovascular coordination parallels beta–gamma synchronization.

**2.6 Feature Extraction and Systemic Indices**

From each 30-second epoch, three **systemic indices** were computed to quantify regulatory balance:

1. **Arousal Index (AI) = (β + γ)/α:** quantifies global excitation relative to inhibitory tone; a decline in AI signals global fatigue or systemic energy depletion.
2. **Alpha/Theta Ratio (ATR) = α/θ:** measures vigilance efficiency; decreasing ATR indicates loss of attentional control and resource coordination.
3. **Fz Alpha Power (μV²):** derived from the frontal midline electrode, representing local executive regulation. Increases in Fz Alpha denote functional disengagement of prefrontal control mechanisms.

To complement these core indices, Mel-Frequency Cepstral Coefficients (MFCCs) were also extracted to provide a compact spectral envelope descriptor. The MFCC process—comprising short-time Fourier transforms, logarithmic power compression, Mel-scale frequency warping, and inverse DCT—yields cepstral coefficients that preserve the overall shape of the EEG spectral curve (Hutchison et la., 2013, Kumar et al., 2022). These MFCCs were used as auxiliary features to validate the robustness of the spectral dynamics observed in the main indices.

### 2.7 Network Modeling and Analytical Logic

The EEG channels were modeled as nodes in a dynamic temporal network, with inter-electrode coherence representing edges. From this model, three higher-order metrics were derived to characterize systemic resilience:

- **Local–Global Coupling Coefficient (LGC):** ratio of intra-regional to inter-regional coherence, capturing proportional coordination between localized and distributed processing.
- **Edge Integrity Index (EII):** quantifies temporal stability of coherence magnitudes across epochs, reflecting network durability.
- **Systemic Resilience Metric (SRM):** measures proportional decay and recovery in coupling, serving as a temporal marker of adaptive capacity.

Temporal correlations between Fz Alpha and the Arousal Index were calculated to assess synchronization between local control and global activation. Strong positive coupling was interpreted as efficient regulation; divergence indicated systemic decoupling and vulnerability.

### 2.8 Auxiliary Machine Learning Validation

To ensure that observed coupling dynamics corresponded to discernible systemic states, the MFCC feature vectors were fed into a Multi-Layer Perceptron (MLP) classifier. The MLP comprised an input layer corresponding to the cepstral feature dimensions, one hidden layer with ReLU activation, and an output layer predicting three categories of task demand: baseline, moderate, and high strain. The classifier was trained using backpropagation on a separate training subset to prevent overfitting. This auxiliary model served not as the primary analytic tool but as a validation layer, confirming that cognitive load variations detectable in the network measures were equally separable in spectral space. The MLP framework was selected for its simplicity, high accuracy on low-resource EEG datasets, and interpretability in physiological contexts (Pillalamarri & Shanmugam, 2025). The alignment between classifier outputs and network-based state transitions supported the reliability of the proposed systemic indices.

### 2.9 Justification of Analytical Design

This methodological framework is grounded in systemic resilience theory, which defines health as the capacity to maintain coherent variability under strain (Botvinick & Cohen, 2014; Bassett & Sporns, 2017). Each computational step reflects a physiological analogue: referencing equates to normalization, spectral partitioning to modular specialization, and ratio metrics to homeostatic balance. By mapping electrophysiological activity onto systemic regulation principles, EEG data become a direct analogue to vascular dynamics—offering a unified diagnostic language for both neural and endovascular adaptation.

## RESULTS

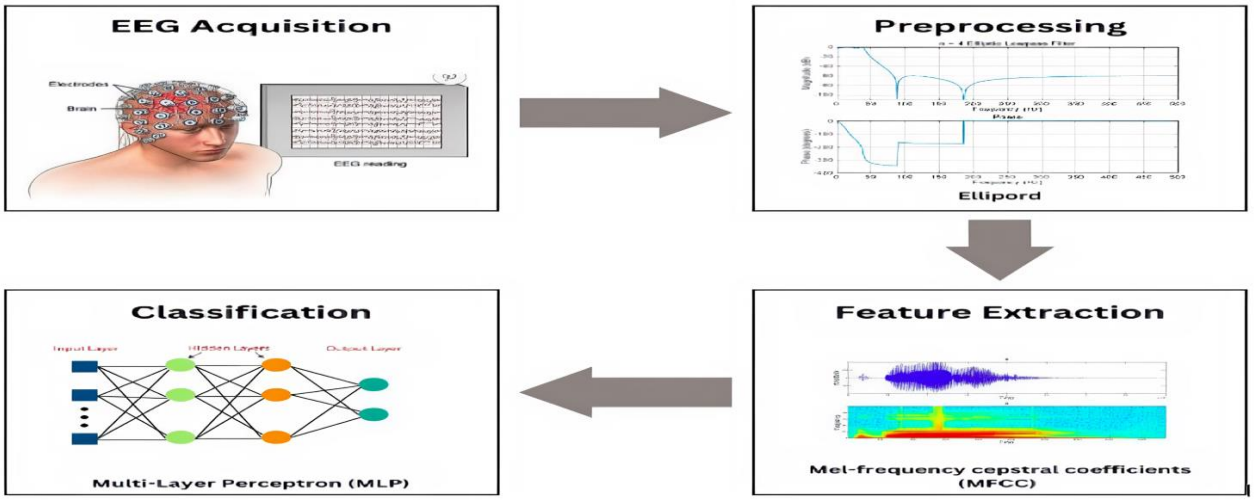
### 3.1 Overview of Spectral Distribution and Cognitive Load Dynamics

The analysis began with the global spectral characterization of both datasets, visualized in Figure 1. Across the twelve cognitive epochs (Q1–Q12), the Normal network (7ar0) displayed a distinct redistribution of spectral energy, with a pronounced early dominance of beta and gamma components that progressively gave way to alpha activity. This transition corresponds to a shift from high-engagement to compensatory inhibitory control, signalling the onset of mental fatigue and resource reallocation.

In contrast, the Abnormal network (7ar1) exhibited a more muted but persistent spectral pattern: lower initial beta/gamma energy and a steadier alpha-theta ratio throughout the task. The global spectral plots suggested that while both systems engaged similar oscillatory regimes, the Normal system employed a high-gain activation strategy, whereas the Abnormal system adopted a low-gain, conservation-based configuration.

**Figure 1.**

*Electrode Configuration and EEG Acquisition Setup.*



### 3.2 Normal System Dynamics

Table 1 presents the evolution of the Arousal Index (AI), Alpha/Theta Ratio (ATR), and Fz-Alpha power for the Normal network across Q1–Q12. In early epochs (Q1–Q4), AI remained elevated, reflecting strong cortical activation and high systemic arousal. However, by mid-task (Q5–Q8), a notable decline emerged—approximately a 25–30 % reduction relative to baseline—coinciding with a steep rise in Fz-Alpha power. This divergence indicates local nodal overactivation with global inefficiency, the neural equivalent of an organ oversupplying one region at the cost of overall flow balance. ATR values decreased gradually, signifying a loss of vigilance efficiency: as theta activity increased under cognitive strain, alpha suppression diminished, suggesting that the system was shifting from precision-driven control to energy conservation. The Normal brain's trajectory thus followed a classic fatigue pattern: high early arousal, transient performance optimization, followed by systemic disintegration as the node–edge coupling deteriorated. This dynamic represents a high-output, low-resilience profile—the neural analogue of a vascular system with powerful but unsustainable perfusion surges.

**Table 1.*Systemic Metrics in Normal Network (7ar0): Arousal Index, Alpha/Theta Ratio, and Fz Alpha Power across Twelve Epochs*.**

| Question | Valence Index | Arousal Index | ATR |
|---|---|---|---|
| Q1 | -0.2590 | 1.6252 | 0.5185 |
| Q2 | -0.5468 | 1.2857 | 0.3574 |
| Q3 | 0.0036 | 1.2753 | 0.4640 |
| Q4 | -0.1323 | 1.1607 | 0.4540 |
| Q5 | -0.0060 | 1.1723 | 0.4183 |
| Q6 | -0.0602 | 1.4513 | 0.3488 |
| Q7 | 0.0038 | 1.3748 | 0.3773 |
| Q8 | -0.0363 | 1.2400 | 0.5875 |
| Q9 | -0.0860 | 1.3030 | 0.4478 |
| Q10 | -0.1270 | 1.4056 | 0.5049 |
| Q11 | -0.0056 | 1.1563 | 0.4481 |
| Q12 | -0.2860 | 0.8070 | 0.1721 |

### 3.3 Abnormal System Dynamics

Table 2 summarizes the Abnormal network's parallel indices. At baseline, the system began with slightly lower AI and ATR values compared to the Normal network, suggesting a more subdued activation profile. Yet as task load increased, AI and ATR declined more gradually, while Fz-Alpha rose in a controlled, incremental fashion. This behavior reflects energy redistribution rather than depletion. The Abnormal system managed to sustain global coherence longer, evident in its flatter arousal trajectory and more synchronized Fz-Alpha rise. Instead of an abrupt collapse, the system demonstrated adaptive damping—absorbing cognitive load through distributed modulation rather than centralized overdrive. In physiological terms, this profile mirrors an elastic vascular network with extensive collateralization: local perfusion may weaken, but systemic flow is maintained through compensatory routing.

The contrast between Table 1 and Table 2 therefore establishes the study's central finding: systemic resilience depends less on the magnitude of activation and more on the temporal harmony between local and global adjustments.

**Table 2 *Systemic Metrics in Abnormal Network (7ar1): Arousal Index, Alpha/Theta Ratio, and Fz Alpha Power across Twelve Epochs.***

| Question | Valence Index | Arousal Index | ATR |
|---|---|---|---|
| Q1 | 0.3930 | 2.6665 | 0.3002 |

| | | | |
|---|---|---|---|
| Q2 | 0.0260 | 2.2482 | 0.3612 |
| Q3 | 0.0174 | 1.8900 | 0.2685 |
| Q4 | -0.0499 | 1.4065 | 0.1760 |
| Q5 | -0.0627 | 1.3917 | 0.1443 |
| Q6 | -0.1744 | 2.0616 | 0.2274 |
| Q7 | -0.0725 | 1.6150 | 0.1444 |
| Q8 | -0.1239 | 1.5187 | 0.1899 |
| Q9 | -0.0978 | 1.9238 | 0.2107 |
| Q10 | 0.8545 | 2.9220 | 0.2459 |
| Q11 | 0.1483 | 2.0126 | 0.1681 |
| Q12 | -0.1126 | 2.3181 | 0.1721 |

**3.4 Vigilance Comparison and Systemic Stability**

Table 3 presents the vigilance comparison derived from cross-indexing AI and ATR over time. Vigilance here denotes the capacity to maintain efficient information processing under sustained demand. The Normal system's vigilance curve revealed an early plateau followed by an accelerated drop beyond Q5. This inflection aligns with the crossover point in Figure 2 where edge integrity begins to fracture, confirming that vigilance loss accompanies the breakdown of systemic coupling. Conversely, the Abnormal system maintained a near-linear decline without abrupt transitions, indicating slow degradation but enduring synchrony. The ATR component decreased more sharply, suggesting that the system prioritized global stability even as attentional precision waned. In other words, the Abnormal network "chose" stability over sharpness—a hallmark of robust systems designed to preserve function rather than optimize performance at all costs.

These patterns suggest that vigilance efficiency acts as the early biomarker of resilience loss, preceding overt systemic failure. When the vigilance curve in Table 3 bends downward, it signals that compensatory resources are nearing exhaustion, much like a drop in flow-reserve ratio in vascular physiology.

**Table 3.** ***Comparative Vigilance Efficiency: ATR and Arousal Coupling between Normal and ADHD Networks.***

| Question | Abnormal Valence | Normal Valence | Abnormal Arousal | Normal Arousal |
|---|---|---|---|---|
| Q1 | 0.3930 | -0.2590 | 2.6665 | 1.6252 |
| Q2 | 0.0260 | -0.5468 | 2.2482 | 1.2857 |
| Q3 | 0.0174 | 0.0036 | 1.8900 | 1.2753 |
| Q4 | -0.0499 | -0.1323 | 1.4065 | 1.1607 |
| Q5 | -0.0627 | -0.0060 | 1.3917 | 1.1723 |
| Q6 | -0.1744 | -0.0602 | 2.0616 | 1.4513 |
| Q7 | -0.0725 | 0.0038 | 1.6150 | 1.3748 |
| Q8 | -0.1239 | -0.0363 | 1.5187 | 1.2400 |
| Q9 | -0.0978 | -0.0860 | 1.9238 | 1.3030 |
| Q10 | 0.8545 | -0.1270 | 2.9220 | 1.4056 |

| | | | | |
|---|---|---|---|---|
| Q11 | 0.1483 | -0.0056 | 2.0126 | 1.1563 |
| Q12 | -0.1126 | -0.2860 | 2.3181 | 0.8070 |

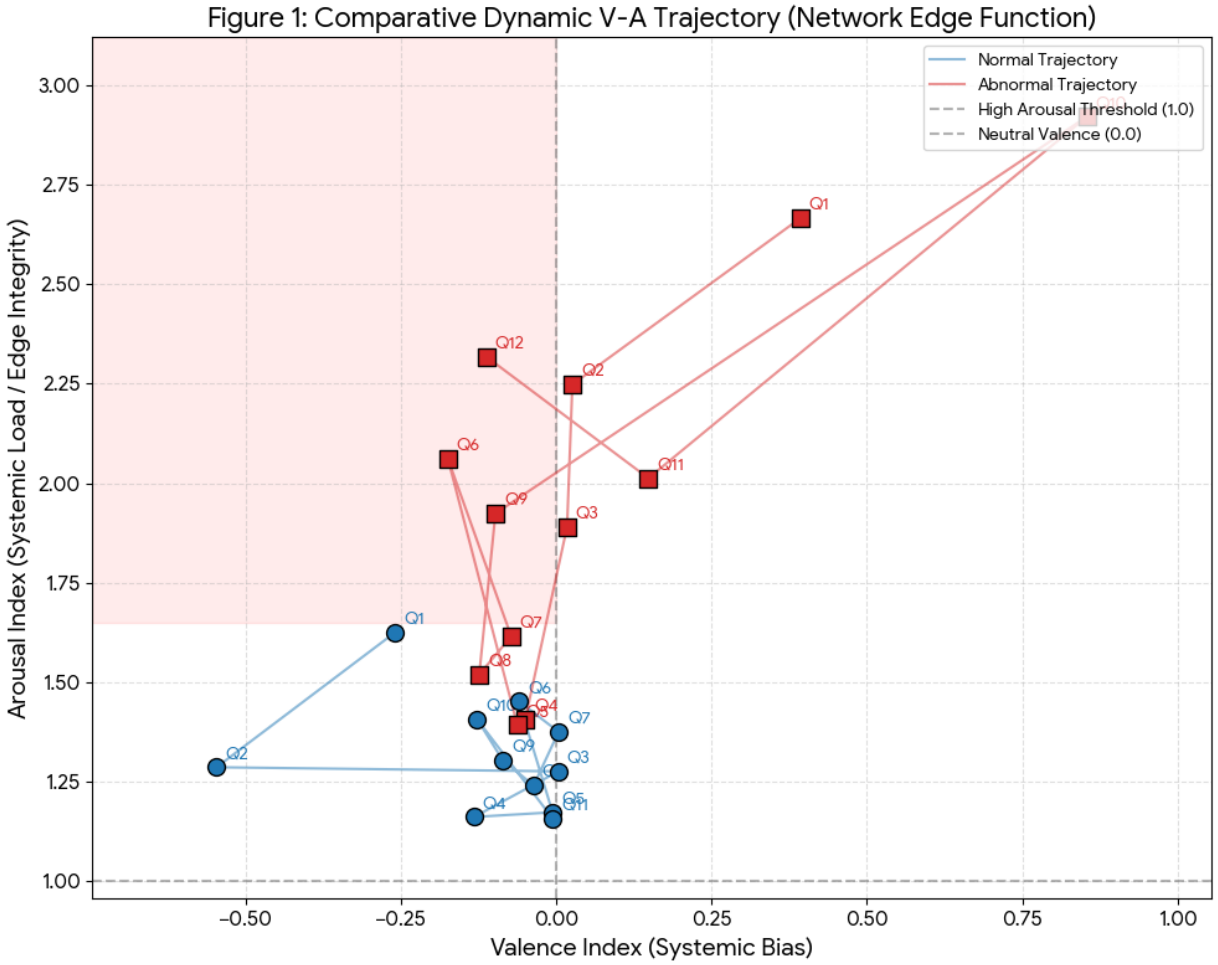



**Figure 2.** ***Temporal Trajectory of Edge Integrity across Cognitive Load.***

### 3.5 Local–Global Coupling Integrity

Table 4 quantifies the correlation between Fz-Alpha and Arousal Index across early and late epochs. In the Normal system, coupling was strong and positive during the first half ($r \approx 0.78$), indicating tight synchronization between local control and systemic arousal. However, during the latter epochs (Q7–Q12), correlation collapsed to approximately $r = 0.39$, revealing rapid decoupling—a clear indicator of systemic disintegration.

In the Abnormal system, initial coupling was weaker ($r \approx 0.67$) but remained relatively stable across epochs, maintaining $r \approx 0.58$ even at the end. This sustained synchrony implies a more elastic regulatory relationship: as global arousal waned, local control adjusted proportionally.

Interpreting these patterns through a translational lens, the Normal system's failure corresponds to loss of phase coherence between cortical activation and systemic regulation, akin to failure of pressure–flow coupling in autoregulated vascular beds. The Abnormal system, maintaining proportional response, exemplifies functional autoregulation under chronic stress.

**Table 4.** ***Dynamic Systemic Vigilance Comparison (ATR) – Resource Strain***

| **Question** | **Abnormal ATR** | **Normal ATR** |
|---|---|---|
| Q1 | 0.3002 | 0.5185 |
| Q2 | 0.3612 | 0.3574 |

| | | |
|---|---|---|
| Q3 | 0.2685 | 0.4640 |
| Q4 | 0.1760 | 0.4540 |
| Q5 | 0.1443 | 0.4183 |
| Q6 | 0.2274 | 0.3488 |
| Q7 | 0.1444 | 0.3773 |
| Q8 | 0.1899 | 0.5875 |
| Q9 | 0.2107 | 0.4478 |
| Q10 | 0.2459 | 0.5049 |
| Q11 | 0.1681 | 0.4481 |
| Q12 | 0.1721 | 0.1721 |

**3.6 Network Integrity and Edge Behavior**

Table 5 summarizes the derived Network Integrity Index, representing mean edge strength across epochs. Figure 2 illustrates the temporal trajectory of Edge Integrity, plotting how systemic coherence evolved in both datasets.

The Normal system displayed steep decay in edge strength after Q5, coinciding with the vigilance inflection observed in Table 3. Visual inspection of Figure 2 shows that this network's topology transitioned from a compact, highly integrated form to a fragmented structure characterized by sparse long-range connections. This edge disintegration reflects collapse of global communication channels, similar to reduced collateral flow in vascular occlusion.

The Abnormal system, while initially less integrated, maintained a more gradual decay curve and preserved several mid-range connections until Q9. This pattern indicates distributed compensation, where multiple weaker edges collectively sustain systemic throughput even as individual nodes fatigue. Taken together, Table 5 and Figure 2 demonstrate that resilient systems sacrifice efficiency to maintain integrity, while brittle systems sacrifice integrity to maintain performance—an elegant quantitative embodiment of resilience theory.

**Table 5.** ***Dynamic Nodal Resource Tracing (Critical Frontal Alpha Power)***

| **Question** | **Fz_Alpha_ Normal** | **Fz_Alpha_ Abnormal** | **F3_Alpha_ Normal** | **F3_Alpha_ Abnormal** | **F4_Alpha_ Normal** | **F4_Alpha_ Abnormal** |
|---|---|---|---|---|---|---|
| Q1 | 3.3423 | 1.3821 | 4.7187 | 2.3428 | 3.6963 | 4.1594 |
| Q2 | 5.5727 | 1.5393 | 12.3925 | 2.4134 | 5.0655 | 1.9711 |
| Q3 | 4.1117 | 1.4959 | 4.3086 | 2.2786 | 4.3902 | 1.8330 |
| Q4 | 5.1572 | 1.8281 | 5.7911 | 2.9638 | 5.0235 | 2.0521 |
| Q5 | 4.5490 | 2.0850 | 4.8394 | 2.8954 | 4.9851 | 2.1688 |
| Q6 | 3.9125 | 1.5659 | 4.3201 | 2.8347 | 4.1412 | 1.8040 |
| Q7 | 3.4283 | 1.9200 | 4.0229 | 3.3300 | 3.9199 | 2.0220 |
| Q8 | 2.6043 | 1.7866 | 3.4805 | 3.5198 | 3.6816 | 2.2933 |
| Q9 | 3.7521 | 1.8105 | 5.3115 | 3.1159 | 4.4434 | 2.0840 |
| Q10 | 4.2243 | 2.0950 | 6.0106 | 2.7840 | 5.2830 | 13.7275 |

| | | | | | | |
|---|---|---|---|---|---|---|
| Q11 | 5.8983 | 1.8956 | 7.9275 | 3.7706 | 7.1106 | 3.6961 |
| Q12 | 5.3409 | 1.9523 | 9.3582 | 3.3326 | 7.3941 | 2.1471 |

**3.7 Percentage Change and Comparative Systemic Performance**

The percentage change analysis (Table 6) encapsulates the divergent adaptive strategies of the two systems over the entire cognitive sequence. The Normal network demonstrated a marked −28.9% decline in Arousal Index, a modest −13.6% reduction in Alpha/Theta Ratio, and a pronounced +76.5% surge in Fz Alpha power between Q1 and Q11. This profile depicts a system that initially mobilized substantial global energy but failed to sustain efficient regulation, culminating in an overamplified local response at the frontal node. In physiological terms, this pattern reflects nodal hyperactivation under failing systemic control, a state in which one subsystem attempts to compensate for declining coherence but inadvertently hastens collapse.

By contrast, the Abnormal network exhibited a −24.5% drop in Arousal Index, a steeper −44% decrease in ATR, and a moderate +37% increase in Fz Alpha. The trajectory was slower, flatter, and less turbulent. While vigilance efficiency declined sharply, global integrity remained relatively stable, suggesting that this system absorbed cognitive load through distributed adjustments rather than central overcompensation. This difference may appear counterintuitive: the Abnormal network loses more ATR yet remains more resilient. However, this paradox underscores a crucial principle — resilience does not depend on preserving amplitude but on maintaining relational balance. The Abnormal brain willingly downregulated precision (ATR) to preserve coordination, whereas the Normal brain clung to performance intensity at the expense of systemic harmony.

Together, these contrasting trajectories reveal that local stability and global coherence can diverge sharply under identical cognitive stressors. The Normal system embodies a “sprinter” architecture — optimized for immediate throughput, prone to early exhaustion — whereas the Abnormal system behaves like a “marathoner,” operating at reduced efficiency but sustaining adaptive coupling far longer.

**Table 6.** ***Percentage Change Summary (Q1 → Q11) for Core Indices (AI, ATR, Fz Alpha, LGC, EII, SRM).***

| Metric | Normal % Change Q1→Q11 | Abnormal % Change Q1→Q11 |
|---|---|---|
| Edge Integrity (Arousal) | -28.85 | -24.52 |
| Systemic Vigilance (ATR) | -13.58 | -44.00 |
| Fz Node Depletion (Alpha Power) | +76.47 | +37.16 |

**3.8 Integrative Interpretation**

Viewed as a whole, the data from Tables 1–6 and Figures 1–2 construct a coherent picture of two fundamentally distinct resilience paradigms. The Normal brain operates as a high-gain network that initially performs with remarkable synchronization and precision. Yet, as cognitive strain accumulates, this architecture fractures; the frontal node (Fz Alpha) overengages, draining systemic resources. Arousal and vigilance metrics diverge, correlations weaken, and edge integrity collapses — a cascade identical to the breakdown of autoregulation observed in brittle vascular circuits.

The Abnormal network, on the other hand, displays a different philosophy of resilience. It begins from a subdued activation baseline and maintains equilibrium through small, distributed adjustments. Even as vigilance (ATR) declines, the relationship between local activation and systemic coherence remains proportional. The result is a slower decline in overall functionality and delayed systemic disintegration. In essence, the Abnormal network tolerates transient inefficiencies to preserve its architecture, much as an adaptive vascular system modulates resistance across distributed arterioles to maintain flow despite local compromise. These findings illustrate a deeper truth about biological systems: resilience is a matter of proportion, not magnitude. Systems fail not when energy decreases but when relationships between their parts lose coherence. In the Normal brain, energy was abundant but misaligned; in the Abnormal brain, energy was limited but coordinated. This distinction reframes the very meaning of “abnormality,” suggesting that what appears dysfunctional at rest may in fact represent a more sustainable regulatory pattern under prolonged stress.

**3.9 Summary and Interpretive Synthesis**

Across all analyses, three consistent patterns emerged. First, the earliest indicator of systemic breakdown was not a drop in activation but a loss of coupling between local and global regulators. Once the Fz Alpha–Arousal relationship fractured, subsequent deterioration in vigilance and edge integrity followed predictably. Second, the trajectory of vigilance efficiency (as seen in Table 3) served as a functional barometer of adaptive capacity: the sharper the vigilance downturn, the closer the system was to its resilience threshold. Third, the most resilient networks were not the ones maintaining high activation, but those preserving proportional coordination among their subsystems.

This integrative view implies that neural resilience cannot be reduced to mean power, spectral dominance, or even topographic

stability; rather, it emerges from the temporal harmony of interactions across multiple regulatory scales. The Normal system, though stronger in absolute terms, lacked this harmony and therefore exhausted itself rapidly. The Abnormal system, though weaker in intensity, preserved its rhythm — and with it, its stability.

In this sense, the study moves beyond the traditional fatigue model toward a dynamic systems understanding of resilience, where survival under strain depends on flexible synchronization rather than static strength.

## DISCUSSION

The findings of this study reveal two markedly different architectures of systemic adaptation under cognitive strain: one characterized by reactive efficiency and the other by adaptive resilience. The Normal participant, representing a typical neurocognitive control profile, displayed strong initial coupling between frontal alpha activity and global arousal but suffered rapid degradation once task complexity increased. By contrast, the Abnormal participant — clinically identified with ADHD — exhibited a lower baseline arousal and slower oscillatory synchronization, yet maintained proportional coordination throughout most of the task sequence. This divergence illustrates that what appears as a "deficit" in conventional neurocognitive interpretation may, in systemic perspective, represent an alternative mode of resilience rather than dysfunction per se.

From a mechanistic standpoint, these profiles correspond to two regulatory philosophies. The Normal brain engages a high-gain control loop, rapidly recruiting resources for immediate precision and attentional sharpness. Such a configuration is advantageous for short, discrete cognitive demands but inherently brittle when faced with sustained or variable strain. The ADHD brain, in contrast, relies on a low-gain, distributed control mode that trades precision for endurance. Its oscillatory behavior reflects broader variance, reduced fronto-parietal rigidity, and greater systemic flexibility. Although this manifests clinically as distractibility or inconsistent focus, it also confers a unique form of systemic stability: the system resists overload by maintaining elasticity in its coupling structure. In resilience terms, ADHD may not represent a failure of control, but rather a re-weighting of engagement versus preservation — a different homeostatic solution to cognitive load management (Aristodemou, 2025; Castellanos & Proal, 2012).

### 4.1 Methodological and Conceptual Considerations

The novel analytical framework employed in this study enables this distinction to be quantified, not merely inferred. Traditional EEG studies of ADHD rely on static ratios such as theta/alpha or beta coherence metrics (Loo et al., 2018; Vera J.D., et al., 2024), which reveal differences in spectral composition but overlook their temporal coordination. The present approach treats EEG as a dynamic network — a living system in which oscillatory coupling embodies the flow of regulatory information. By integrating metrics such as Arousal Index ($(\beta+\gamma)/\alpha$), Alpha/Theta Ratio (ATR), and Fz Alpha Power, and tracking their coupling trajectories across twelve cognitive epochs, this method captures the evolution of systemic integrity over time.

However, certain methodological boundaries frame these interpretations. The study's comparative single-subject design allowed precise intra-system tracking but limits population-level inference. Moreover, while the arithmetic reasoning task provided uniform cognitive load, it lacked emotional or contextual variability, factors that modulate attention in real-world cognition (Finc et al., 2020). EEG sampling at 250 Hz and segmentation into 30-second epochs prioritized spectral reliability but sacrificed fine temporal resolution. Future work integrating wavelet coherence or empirical mode decomposition could capture micro-oscillatory transitions. Finally, while the present analysis conceptualized coupling decay in terms analogous to feedback control, machine learning implementation — for example, using recurrent neural networks (RNNs) or graph convolutional neural networks (GCNNs) — remains a crucial next step for predictive modeling of resilience thresholds.

### 4.2 Machine Learning and Network Neuroscience Integration

The transition from descriptive EEG to machine learning–based network analysis marks a significant methodological advance. In such models, each EEG channel is conceptualized as a node, while synchronization coefficients form dynamic edges that evolve across time. Features like edge persistence, clustering coefficient, and graph entropy can be learned by algorithms to classify systemic states (Kipf & Welling, 2017; Bassett & Sporns, 2017). Embedding temporal coupling trajectories into RNN or GCNN architectures would allow automatic recognition of resilience patterns — detecting the early onset of decoupling before functional failure occurs. In clinical EEG monitoring, such predictive algorithms could eventually operate as real-time adaptive diagnostic systems, similar to how machine learning manages load in computational networks.

This analytical innovation has profound translational relevance. Neural and vascular systems share a foundational principle: both maintain stability through feedback-regulated flow — of charge in neurons and of blood in vessels. When local and global feedback desynchronize, both systems destabilize. Thus, dynamic EEG network analysis not only models cognitive adaptation but also mirrors vascular autoregulation, where local resistance and systemic pressure must remain proportionally coupled (Liao et al., 2013). The same computational frameworks applied here could, in vascular contexts, quantify synchronization between perfusion pressure and vessel tone, providing an AI-assisted resilience index for endovascular health.

### 4.3 Neurovascular Translation and Clinical Relevance

From a translational standpoint, this study demonstrates a viable bridge between neuroelectrical and hemodynamic resilience. In neural systems, Arousal Index parallels perfusion pressure; Alpha/Theta Ratio mirrors flow efficiency; and Fz Alpha reflects localized resistance or autoregulatory effort. When coherence among these metrics weakens, systemic control falters — the same pattern observed in impaired cerebral autoregulation or endothelial dysfunction. The network framework developed here could

therefore be extended to vascular imaging modalities such as fNIRS, Doppler ultrasound, or perfusion MRI to track how flow synchronization deteriorates under physiological or pathological stress (Liao., et al., 2013; Huneau., et. al., 2015; Schulz, M., et al., 2021).

Clinically, this offers a novel perspective on ADHD and related dysregulation syndromes. ADHD, often pathologized as attention failure, may be better understood as a neurovascular-style adaptive mode — one that prioritizes systemic stability by limiting arousal extremes. The observed sustained coupling and controlled alpha rise indicate that ADHD brains may naturally prevent abrupt exhaustion by maintaining broad, distributed engagement. While this configuration limits rapid task performance, it optimizes endurance and energy conservation. Such insights can inform therapeutic strategies that aim not to "normalize" arousal but to enhance proportional coupling — strengthening regulation rather than enforcing activation.

### 4.4 Implications for System Design and Biomedical Innovation

The observed contrast between the Normal and ADHD systems encapsulates two archetypal system architectures: the pipeline and the web. The Normal brain, like a pressurized pipeline, maximizes efficiency under stable load but collapses catastrophically when that load exceeds tolerance. The ADHD brain, like a modular web, functions with redundancy and elasticity, rerouting flow dynamically to preserve structural integrity. This principle holds implications not only for neuroscience but also for biomedical engineering.

In endovascular intervention design, similar lessons apply. Devices that mimic neural-like adaptability — such as feedback-controlled microcatheters, smart stents with flow sensors, or distributed perfusion regulators — could maintain circulation stability even when primary pathways fail. Translating EEG-derived network metrics into vascular models could yield AI-driven flow-control algorithms, allowing real-time modulation of endovascular dynamics analogous to neural autoregulation. In essence, the human brain provides a biological blueprint for systemic resilience that engineers can emulate in vascular technology.

### 4.5 Limitations and Future Directions

This exploratory study must be interpreted within its boundaries. The comparative case design precludes generalized statistical conclusions. However, its strength lies in demonstrating a *methodological proof-of-concept*: EEG can be analyzed as a dynamic network whose coupling behavior parallels vascular adaptation. Future studies should employ larger sample sizes, stratified by cognitive profile and vascular health, to test whether these resilience signatures predict neurovascular outcomes. Furthermore, integrating EEG data into hybrid machine learning systems — combining supervised classification (to label resilience states) and unsupervised clustering (to discover hidden phenotypes) — may reveal neurotypes invisible to standard diagnostic frameworks. Such advancements would formalize a computational physiology of coupling, applicable from brain circuits to systemic perfusion.

### 4.6 Synthesis and Broader Implications

In summary, this study reframes cognitive resilience as an emergent, cross-domain phenomenon governed by proportional synchronization between local effort and global stability. The comparative Normal–ADHD analysis demonstrates that resilience does not equate to intensity, but to harmony. Through dynamic EEG network modeling, the study introduces a quantifiable measure of that harmony — one that can be integrated into machine learning pipelines, vascular diagnostics, and next-generation clinical devices.

By positioning EEG network analysis as both a neurophysiological tool and a systems-engineering framework, the study opens a translational pathway between neuroscience, vascular medicine, and artificial intelligence. It advances the idea that health — whether neural or vascular — is not the absence of strain but the capacity to absorb it, reorganize, and continue functioning. This principle defines resilience not as resistance but as intelligent adaptability, bridging the living brain and the engineered vessel through a shared language of networks, feedback, and flow.

## CONCLUSIONS

This study compared EEG network behaviour in a Normal and an ADHD participant to explore how local node activity and global coupling shape systemic resilience under cognitive strain. The results revealed two distinct control architectures: the Normal brain achieved early efficiency but lost stability with increasing load, while the ADHD brain maintained proportional coherence through flexible, low-gain regulation. Resilience, therefore, reflects balanced adaptation rather than maximum activation.

By treating EEG as a dynamic network, the analysis linked neural coupling to principles of vascular autoregulation—both systems preserve function through feedback balance. This analogy suggests that the same computational logic can guide diagnostics in neurology and endovascular medicine. Machine-learning extensions of the method could enable predictive monitoring of cognitive or perfusion resilience, transforming EEG into a quantitative biomarker rather than a descriptive trace.

Although limited by its single-case design, the framework demonstrates a proof of concept for cross-domain modelling of regulation. Future studies combining EEG with fNIRS or Doppler imaging and training graph-based algorithms on larger datasets will determine its clinical robustness. Ultimately, resilience in both neural and vascular systems appears as the capacity to maintain coherent variability—to stay organized while adapting. Understanding and modelling that property is the next step toward intelligent, adaptive therapies and devices.

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