

THE ARCHITECTURE OF FOCAL CIRCUITS: Local Routing Divergences, Cross-Talk, and Output Impedance

Camila Leão de Matos Brezolin

Independent Researcher, Brasília-DF, Brazil. Specialist in Neuroscience, Data Science and Artificial Intelligence, Data Analysis and Applied Mathematics, and Materials Science and Engineering. Bachelor of Science (B.S.) in Logistics.

ORCID: <https://orcid.org/0009-0009-1009-1157>

Contact E-mail: cmladelfi@gmail.com

Sidnei Brezolin de Freitas

Independent Researcher, Brasília-DF, Brazil. Specialist in Neuroscience, Human Behavior, Data Analysis and Applied Mathematics, and Competitive Intelligence. Software Engineer.

ORCID: <https://orcid.org/0009-0000-1124-4711>

Contact E-mail: brezolin10.pardal@gmail.com

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ABSTRACT

While global neurocomputational phenotypes alter the thermodynamic baseline of the entire brain, specific cognitive variations leave the primary P-FIT macro-bus intact, manifesting instead as localized architectural constraints. This paper isolates and formalizes the Architecture of Focal Circuits under the HEPOE framework, defining Dyslexia, Dysgraphia, and Synesthesia through micro-topological hardware variations. Rather than systemic energy failures or global resistance, these phenotypes represent isolated routing deviations, localized output impedance, or regional insulation failures. By integrating these focal constraints into the Predictive Solvency framework, we establish a mechanistic taxonomy that distinguishes global systemic variants from circumscribed circuit anomalies, providing a precise biophysical model for localized information-processing divergence.

Keywords: HEPOE Framework. Focal Circuits. Dyslexia. Dysgraphia. Apraxia. Synesthesia. Output Impedance. Routing Divergence. Local Cross-Talk.

1. INTRODUCTION: MACRO-SYSTEMIC SOLVENCY VS. MICRO-TOPOLOGICAL CONSTRAINTS

A comprehensive biophysical taxonomy of human cognition requires a strict structural distinction between global and local hardware configurations. Global phenotypes, such as the high-fidelity wide-band processing of the Sentinel Phenotype or the systemic impedance of Autism, uniquely adjust the thermodynamic baseline across the entire neural network. Conversely, focal circuit phenotypes possess standard macro-systemic parameters for global resistance (Ω) and metabolic resynthesis (Φ), yet experience severe localized information-processing constraints. This paper maps these circumscribed anomalies, establishing that localized routing errors, isolated output barriers, and regional cross-talk represent specific hardware traits within dedicated sub-networks.

2. DYSLEXIA: LOCAL ROUTING DIVERGENCE AND TRANSMISSION DELAY

In current clinical descriptions, Dyslexia is categorized as a phonological processing deficit. Under the HEPOE lens, this is formalized as a localized routing divergence within the left perisylvian language bus. Micro-structural variations in neurodevelopment, such as focal cortical ectopias, alter the physical topology of the phonological decoding pathway. Instead of utilizing the standard low-impedance direct route, the electrical signal carrying symbolic data is forced to traverse longer, more complex alternative pathways. This structural detour introduces a significant transmission delay, altering the temporal alignment required for real-time grapheme-to-phoneme conversion. The macro-system remains highly solvent, but the local channel demands increased ATP expenditure to overcome the extended transmission distance, transforming symbolic decoding into a high-friction operation.

3. DYSGRAPHIA AND APRAXIA: LOCAL OUTPUT IMPEDANCE (Ω_{out})

Dysgraphia and motor apraxias present a profound dissociation between central predictive planning and peripheral execution. In these states, the central cognitive model generates flawless predictive blueprints; the internal fidelity of the desired output is fully preserved. The computational breakdown occurs exclusively at the efferent boundary, modeled as Local Output Impedance (Ω_{out}).

The localized projection tracts connecting motor planning nodes to the fine-motor execution pathways possess elevated resistance. As the efferent command signal hits this focal barrier, a massive portion of its electrical energy dissipates as localized Joule heating before reaching the periphery. To overcome Ω_{out} and execute fine-motor movements, the system must dramatically amplify signal voltage, resulting in rapid regional metabolic depletion and severe localized muscle and cognitive fatigue despite intact central solvency.

4. SYNESTHESIA: INSULATION FAILURE AND LOCAL CROSS-TALK

Synesthesia represents a unique micro-structural variation where distinct sensory modalities are physically linked. Sensorimotor development typically relies on localized synaptic pruning to isolate adjacent processing zones, such as the visual and auditory cortices. In the synesthetic hardware, this focal pruning is incomplete, preserving low-impedance structural bridges between independent sensory nodes.

When an incoming data stream activates a primary sensory node (e.g., an auditory frequency), the current leaks directly through these uninsulated structural bridges into the adjacent sensory map (e.g., the visual cortex). This local cross-talk causes simultaneous activation across separate modalities. Within the HEPOE framework, this is not a cognitive association or a metaphor, but a literal physical redirection of data traffic across unpruned, low-resistance local circuits.

Table 1. The HEPOE Clean Topology of Focal Circuitry

Focal Phenotype	Micro-Topological Mechanism	HEPOE Equation Local Modifier
Dyslexia	Left perisylvian ectopias driving signal routing detours.	Increased path length multiplier on local symbolic processing tracts.
Dysgraphia / Apraxia	Elevated physical resistance in efferent motor projection tracts.	Introduction of local output impedance (Ω_{out}) at the motor bus boundary.
Synesthesia	Incomplete synaptic pruning creating low-resistance inter-cortical bridges.	Cross-talk current coefficient leakage across adjacent sensory nodes.

Source: Authors (2026)

5. CONCLUSION: THE CLEAN TOPOLOGY OF FOCAL CIRCUITRY

Isolating focal circuit anomalies from global systemic conditions preserves the purity of the HEPOE taxonomy. By identifying Dyslexia, Dysgraphia, and Synesthesia as localized structural traits, the framework clarifies that an individual can possess a globally high-fidelity, highly solvent macro-architecture while simultaneously navigating specific, high-friction bottlenecks within circumscribed local tracts. This micro-topological approach removes behavioral ambiguity, anchoring clinical variation directly to localized biological circuits.

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