

Functional-Structural Dissociation in Acute Stroke: Multi-Layered Lurian Syndromic Analysis and Network Hub Disruption

Alexander Erzin

alexerzini@gmail.com

The Moscow Botkin Multidisciplinary Scientific-Clinical Center <https://orcid.org/0000-0003-1046-4469>

Research Article

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Additional Declarations:

The authors declare potential competing interests as follows: The author declares that the digital platform NeuroDraft (Cognicore NeuroSystems) was utilized strictly as an electronic data collection interface and score aggregator for workflow automation to standardize qualitative Lurian metrics. The software functions as a deterministic algorithmic support instrument and does not operate as an autonomous diagnostic classifier or predictive SaMD (Software as a Medical Device) entity. The author is the primary developer of the platform and the founder of Cognicore NeuroSystems.

Consent to Participate: Informed consent was obtained from all individual patients or their legal representatives prior to evaluation. The neurocognitive assessments were conducted as part of routine clinical neurological practice within official Medico-Economic Standards (MES) to optimize personalized rehabilitation and therapy pathways. The evaluation does not constitute a forensic or disability examination, nor is it aimed at autonomous diagnostic classification. All clinical data were strictly de-identified and anonymized to ensure complete patient privacy.

Functional-Structural Dissociation in Acute Stroke: Multi-Layered Lurian Syndromic Analysis and Network Hub Disruption

ABSTRACT

Background: Acute cerebral ischemia presents a critical dissociation between hyperacute synaptic failure and macrostructural tissue necrosis. Conventional linear screening instruments (e.g., MMSE, MoCA) exhibit pronounced ceiling effects and obscure modular neurodynamic disconnection. Modern connectomics posits that focal ischemia triggers acute network diaschisis across distributed large-scale circuits—specifically the default mode and frontoparietal control networks – prior to the emergence of radiologically visible cytotoxic edema on non-contrast computed tomography (CT). We evaluated a digital adaptation of Luria’s qualitative syndromic process approach to quantify this functional-structural dissociation during early post-stroke triage.

Methods: In this observational audit at a tertiary stroke center, 124 patients with verified acute stroke were stratified by emergent neuroimaging into an acute CT-negative presentation group (n = 56) and a macrostructural lesion-verified necrosis group (n = 68). Bedside neuropsychological testing (24–48 hours post-ICU stabilization) was conducted via the NeuroDraft platform across 10 independent cognitive domains using the continuous Luria Raw metric (0.0–5.0 penalty scale). Latent dimensionality was examined via complete-case Principal Component Analysis (PCA) with orthogonal Varimax rotation (n = 121), following the predefined methodological exclusion of 3 individuals with early-stage extrapyramidal pathology to prevent motor-slowness confounds.

Results: Memory and attention deficits exhibited striking cross-cohort statistical equivalence. Memory impairment showed near-identical severity ($M = 1.69 \pm 0.91$ vs. 1.80 ± 0.93 ; $U = 1870.5$, $p = 0.87$), and complex attention diverged minimally ($M = 1.19 \pm 0.95$ vs. 1.43 ± 0.94 ; $U = 1677.0$, $p = 0.25$), demonstrating early network-level vulnerability preceding macroscopic cytotoxic alterations. Conversely, language functions demonstrated significant resilience in the CT-negative cohort ($M = 0.69 \pm 0.98$ vs. 1.14 ± 1.20 ; $U = 1465.5$, $p = 0.03$). Intra-individual profile variability revealed a trend toward syndromic flattening as structural damage consolidated (coefficient of variation: 1.26 vs. 1.09; $U = 1344.0$, $p = 0.06$). Factor decomposition resolved two independent axes: posterior instrumental-operational integrity and anterior-subcortical dynamic-regulatory control.

Conclusions: Network-level functional collapse outpaces visible parenchymal necrosis in emergent stroke. Digital Lurian profiling provides sensitive, deterministic quantification of early diaschisis, supporting bedside decision-making without replacing macrostructural neuroimaging.

Keywords: acute ischemic stroke, network diaschisis, digital phenotyping, clinical decision support systems, cognitive connectomics, predictive neuroscience.

INTRODUCTION

Limitations of Emergent Macrostructural Neuroimaging and the Crisis of Linear Screening Scales

Acute cerebral ischemia is characterized by a critical nonlinearity between the onset of microcirculatory failure and the objective manifestation of macrostructural tissue damage. In modern emergent vascular neurology, transient hypoperfusion is posited to trigger a cascade of synaptic dysfunction well before the establishment of irreversible necrosis detectable by conventional neuroimaging modalities (Albers et al., 2018; Fisher & Bastan, 2012; Saver, 2006; Wintermark et al., 2008). The so-called "CT-negative therapeutic window" represents the period of highest therapeutic valence; however, standard non-contrast computed tomography exhibits critically low sensitivity to cytotoxic edema during the initial hours of ischemia (Donnan & Davis, 2002; Hacke et al., 2008; Rowley, 2001).

The diffusion-perfusion mismatch paradigm, verified via MRI (DWI/PWI), demonstrates extensive penumbral zones that remain functionally inactive yet morphologically viable (Hillis et al., 2001; Sorensen et al., 1996; Wardlaw et al., 2019). Under these conditions, the functional disintegration of cognitive domains outpaces the structural degradation of the parenchyma. Nonetheless, conventional bedside screening instruments, such as the MoCA or MMSE, exhibit marked ceiling effects and fail to detect latent patterns of network disconnection, offering merely an aggregate linear sum of scores (Heilman & Valenstein, 2011; Lezak et al., 2012; Mesulam, 1990). This methodological divide underscores the urgent need for sensitive digital neuropsychological bedside screening during the acute stage of stroke.

The Paradigm of Network Disconnection and Diaschisis within the CT-Negative Window

Modern connectomic models conceptualize the brain as a complex topological architecture of large-scale neural circuits (Menon, 2011; Sporns, 2014; van den Heuvel & Sporns, 2013). The Triple-Network Model delineates the dynamic interplay among the Default Mode Network (DMN), the Frontoparietal Control Network (FPCN), and the Salience Network (SN) (Bullmore & Sporns, 2012; Fornito et al., 2016; Yeo et al., 2011). Focal ischemia, even when subclinical and radiologically occult on non-contrast CT, triggers network diaschisis—the acute collapse of distributed attentional and executive networks resulting from the functional disruption of critical topological hubs (Bassett, & Sporns, 2023; Buckner et al., 2008; Gordon et al., 2017; Power et al., 2011).

Subcortical hubs play a paramount role in this network architecture, though their integrative capacity has historically been underappreciated. Contemporary formulations of Cerebellar Cognitive Affective Syndrome (CCAS) and thalamic aphasia demonstrate the orthogonality of cerebellar and diencephalic circuits relative to cortical networks (Manto & Mariën, 2015; Schmahmann, 2019; Stoodley & Schmahmann, 2010). The cerebellum and thalamus operate as high-speed coprocessors that modulate the fluency and anticipation of cognitive operations; their microcirculatory compromise induces a systemic dysmetria of thought prior to the structural delineation of a cortical lesion (Carrera & Tononi, 2014; Diedrichsen et al., 2019; Habas et al., 2009; Schmahmann, 2003; Bostan & Strick, 2018). Predictive analytics employing CCAS-based algorithms demonstrate high sensitivity to early network disconnection (Cambreri et al., 2025, Guell et al., 2018).

Quantification of the Lurian Process Approach: The Digital Luria Raw Metric

Resolving the crisis of psychometric reductionism requires a return to syndromic analysis, operationalized through contemporary digital quantification. Western cross-cultural neuropsychology has integrated A. R. Luria's process-oriented approach, adapting it into rigorous, standardized batteries (Mikadze et al., 2019; Ardila, 2020; Glozman, 1999). A critical advance was the development of the Test Barcelona Revised (TBR), which profiles vascular cognitive impairment using high-resolution Luria Raw metrics across a quantifiable score continuum for each subtest (Christensen, 1984; Peña-Casanova et al., 2021, 2024; Tupper, 1999). This grounds the paradigm shift from merely recording terminal outcomes to evaluating the neurodynamic process quality of task execution (Kotik-Friedgut, 2001).

In neurogerontology and the assessment of cognitive reserve during cerebrovascular events, the analysis of mirror systems, anticipation, and adaptive coping mechanisms—which support compensatory resilience—is critical (Erzin & Chekrygina, 2020; Korsakova et al., 2013). Acute ischemia affecting the first functional unit (the energetic block) abruptly decompensates the neurodynamic substrates of successful cognitive aging, which can be instrumentally captured via high-sensitivity reaction-time fluctuations (Korsakova & Balashova, 2007; Erzin & Kovtunencko, 2019; Roshchina, 2015).

System of Concurrent Domains and Formulation of Working Hypotheses

Grounded in this structural-functional dissociation paradigm, the present study employs multi-domain neuropsychological profiling via the **NeuroDraft** platform (*Cognicore NeuroSystems*) in patients with acute cerebral ischemia. The empirical base comprises a cohort of $n = 124$, stratified by acute neuroimaging status into an acute CT-negative therapeutic window group ($\text{mri_status} = 2$, $n = 56$) and a lesion-verified cohort with established macrostructural tissue necrosis ($\text{mri_status} = 1$, $n = 68$).

The following working *hypotheses* are posited:

1. Convergence of memory processes: Deficits in the D9 (*Learning & Memory*) domain are systemic, driven by generalized diaschisis of hippocampal-diencephalic circuits, and exhibit no statistically significant differences between the CT-negative and lesion-verified cohorts (expected values: $M = 1.69$ vs $M = 1.80$, $p = 0.87$), demonstrating that functional disconnection of memory networks precedes macrostructural necrosis.
2. Speech reserve and cortical vulnerability: In contrast to memory functions, the D8 (Language & Speech Functions) domain exhibits high sensitivity to structural lesion consolidation during cortical ischemic injury. During the acute CT-negative window characterized by unstable perfusion, speech functions maintain relative functional stability, reflected in a significantly lower penalty index ($M = 0.69$) compared to the lesion-verified cohort ($M = 1.14$), where frank organic deficits manifest alongside macrostructural necrosis consolidation ($p = 0.03$).
3. Entropy and neurodynamic instability: Profile entropy, indexed by the coefficient of variation (CV), will serve as a predictor of network dysmetria, reflecting higher neurodynamic noise during the CT-negative window ($CV = 1.26$) relative to consolidated structural defects ($CV = 1.09$), approaching statistical significance ($p = 0.06$).
4. Subcortical-cortical orthogonality: Cognitive deficit patterns captured by digital metrics will strictly differentiate damage to cortical DMN/FPCN nodes from cerebellar-thalamic hub dysfunction.

MATERIALS AND METHODS

Study Design and Institutional Compliance

The present study was conducted as a retrospective analysis of clinical audit data and routine expert evaluations at the Moscow Multi-profile Scientific and Clinical Center named after S.P. Botkin of the Moscow Healthcare Department.

Primary data acquisition, comprising multidimensional syndromic profiling, was performed by a staff clinical (medical) psychologist directly at the patient's bedside. The study protocol and the retrospective analysis of de-identified data were approved by the Local Ethics Committee of GBUZ MMCC named after S.P. Botkin. All stages of the study strictly adhered to the ethical standards of the Declaration of Helsinki of the World Medical Association.

Department Structure and Study Location

The diagnostic framework encompassed specialized units of MMCC named after S.P. Botkin providing an unbroken emergency pathway for acute stroke management. Initial clinical and neuroimaging screening (expert-class computed tomography) was performed continuously at the Emergency Department (the admission department of the specialized hospital).

Following vascular status verification and hemodynamic stabilization in the intensive care unit (ICU), patients were transferred to specialized inpatient units, where the present neuropsychological evaluation was conducted.

Data collection took place within an interval of 24 to 48 hours following patient transfer from the intensive care unit to the general wards, under conditions of stabilized consciousness and preserved capacity for productive contact.

Study Protocol and Clinical State of Patients

Data collection was conducted between January and July 2026. Assessments were performed in standard inpatient rooms of specialized neurological units, following the completion of the acute intensive care phase. Testing was executed strictly following patient transfer to general wards, within an interval of 24 to 48 hours after transfer from the intensive care unit. Clinical status at examination was characterized by relatively complete stabilization of vital signs, restoration of clear consciousness, and satisfactory capacity for productive verbal and/or gestural bedside communication.

Patients were receiving standard basic medical management, without specialized primary cognitive or motor rehabilitation. Basic pharmacotherapy strictly adhered to national clinical protocols for emergent stroke care and was aimed at intensive somatic stabilization. At the primary resuscitation stage in the intensive care unit, systemic thrombolysis and anticoagulant support with low-molecular-weight heparins were administered.

Following patient transfer to the neurological and early neurorehabilitation units, medical management comprised continuous antiplatelet therapy, static lipid-lowering therapy, antihypertensive agents for hemodynamic stabilization, and neuroprotective metabolic support. This pharmacotherapeutic regimen ensured secondary prevention and neuroprotection within the ischemic penumbra, while strictly excluding targeted cognitive stimulation or specialized

neuropsychological rehabilitation, thereby preserving the cognitive profile recorded by the NeuroDraft platform as an unconfounded representation of the acute cerebrovascular deficit.

Expert Qualification and Testing Conditions

All diagnostic sessions were conducted by an expert clinical (medical) psychologist with over 7 years of clinical experience in neurorehabilitation of cerebrovascular lesions. Examinations were administered in the first half of the day (between 10:00 and 13:00) under conditions of minimized environmental sensory stimulation. Diagnostic session duration was strictly limited to 40–60 minutes to prevent profound neurodynamic fatigue and asthenization that could confound performance on graphomotor and other subtests. If signs of fatigue emerged, testing was paused and resumed following a regulated rest period.

Formation of the General Hospital Registry

The initial data pool over 7 months of 2026 comprised a general hospital registry of $N = 169$ verified clinical cases. Biological sex distribution (SAGER Guidelines) in the baseline registry was 84 males (49.7%) and 85 females (50.3%). The mean age of the overall cohort was 66.87 years ($SD = 13.9$), spanning a wide clinical range from 18.0 to 97.0 years.

Selection Criteria and Target Cohort Stratification

For this sub-analysis of functional-structural dissociation, a targeted cohort of acute stroke patients ($n = 124$) was isolated from the general registry ($N = 169$) based on the neuroimaging stratification standard.

Selection into the target cohort was conducted based on the following predefined boundaries:

Inclusion criteria: age 18 years and older; verified acute vascular event (ischemic or hemorrhagic stroke); lesion localization within the carotid (internal carotid artery territory) or vertebrobasilar (VBA) arterial territories; relatively stable level of wakefulness at bedside testing allowing productive verbal or gestural interaction. To evaluate algorithmic robustness against poly-nosological noise, patients with controlled comorbid, latent, or early de novo Parkinson's disease were eligible (1.8% of the registry population, $n = 3$), provided they strictly fulfilled Movement Disorder Society (MDS) diagnostic criteria and demonstrated intact basic executive control programs during bedside interactions.

Exclusion criteria: somatic control patients without focal brain lesions (cardiology, internal medicine, and traumatology profiles; Code 0 / Normal, $n = 45$, 26.6%); severe dominant upper extremity motor deficit precluding stylus grasp and execution of digital graphomotor subtests (with the exception of controlled microtremor in early Parkinsonism that did not impair goal-directed execution); pre-existing severe cognitive decline (documented pre-stroke major dementia); missing or incomplete anatomical/topographic clinical records.

Demographic and Nosological Structure of the Sub-Analysis ($n = 124$)

The selected target acute stroke cohort ($n = 124$) was stratified into two distinct subgroups based on emergent neuroimaging status:

Acute CT-negative therapeutic window subgroup (mri_status = 2, n = 56, 33.1% of the overall registry): Patients assessed in the initial hours post-deficit onset whose primary non-contrast computed tomography revealed no macrostructural tissue alterations (ischemic core not yet visible).

Lesion-verified structural necrosis subgroup (mri_status = 1, n = 68, 40.2% of the overall registry): Patients with visually confirmed, topographically delineated ischemic or hemorrhagic lesions on neuroimaging records.

Within the analyzed acute stroke cohort (n = 124), vascular distribution and pathogenic stroke subtypes (TOAST criteria) were partitioned as follows:

By vascular territory (clinical_pool), the stroke cohort was distributed as follows: internal carotid artery territory (ICA, pool = 1) accounted for 74 patients (59.7%); vertebrobasilar territory (VBA, pool = 2) accounted for 40 patients (32.3%); and combined or multiple arterial territories (pool = 3) accounted for the remaining 10 patients (8.1%), ensuring absolute numerical consistency across the analytical stroke framework (n = 124).

By pathogenic subtype (TOAST), the distribution encompassed five distinct categories: large-artery atherosclerosis (LAA, code 1); cardioembolism (CE, code 2); small-vessel occlusion / lacunar stroke (SVO, code 3); stroke of other determined etiology (ODE, code 4); and stroke of undetermined etiology (UD, code 5).

The age-sex distribution of the target cohort (n = 124) across four age brackets (young: <45 years; middle-aged: 45–60 years; elderly: 61–75 years; senile: >75 years) corresponds identically to the distribution utilized in Kruskal-Wallis non-parametric analyses and variance matrices.

Neuroimaging Protocol

Emergent neuroimaging verification was performed immediately upon hospital admission at the emergency reception department. Brain computed tomography (CT) was conducted using a 160-slice Aquilion Prime SP CT scanner (Model TSX-303B, Canon Medical Systems, Japan). Scanning was performed in non-contrast axial mode with a reconstructed slice thickness of 1 mm, ensuring submillimeter spatial resolution and preventing missed focal parenchymal lesions. The mean effective radiation dose per patient during initial emergent scanning was 6 mSv.

Submillimeter slice reconstruction on this expert-grade imaging system validated the status of patients in the mri_status = 2 subgroup as presenting within an authentic CT-negative therapeutic window. The absence of demonstrable density abnormalities (both cytotoxic ischemic edema and hemorrhagic extravasation) on 1 mm contiguous slices confirmed that the multidimensional cognitive deficit captured by the NeuroDraft platform reflected functional, pre-structural synaptic dysfunction.

For follow-up dynamic monitoring, precise lesion localization, and microstructural verification during scheduled inpatient ward assessments, magnetic resonance imaging (MRI) was employed in selected cases. Examinations were performed on high-field superconducting scanners: Siemens Magnetom Aera 1.5 Tesla (Siemens Healthineers, Germany) and Philips Ingenia 3.0 Tesla (Philips Healthcare, Netherlands). The MR protocol comprised standard non-

contrast sequences: T1-weighted imaging (T1-WI), T2-weighted imaging (T2-WI), fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted imaging (DWI) with apparent diffusion coefficient (ADC) mapping to differentiate acute cytotoxic edema from chronic leukoaraiosis and cystic-gliotic parenchymal alterations.

Neuropsychological Mapping: Instrumentation and Methodology

Multidimensional cognitive evaluation was performed using the NeuroDraft digital module (Cognicore NeuroSystems), which provides a quantified adaptation of classical Lurian syndromic analysis. The methodological architecture combines the dynamic localization of higher mental functions with established Western process-oriented paradigms, notably the Boston Process Approach and integrated quantitative profiling models of vascular cognitive impairment (Test Barcelona Revised).

Bedside assessments employed standardized paper test forms (drafts). Patients completed tasks manually on physical forms. Qualitative errors, neurodynamic processing speed, perseverative tendencies, and spatial distortions were directly observed and recorded by the expert.

Following each session, verified qualitative parameters and raw scores were transferred into the NeuroDraft analytical web interface. The platform served as a deterministic expert processing system: the algorithmic computational backend transformed raw scores into standardized continuous impairment indices across 10 independent cognitive domains (Luria Raw Metric) ranging from 0.0 (fully preserved function) to 5.0 (complete functional collapse/domain loss):

- *D1. Complex Attention & Processing Speed* — information processing speed, attentional stability, and concentration.
- *D2. Visual Object Perception* — visual object gnosis, recognition of realistic and degraded/embedded images.
- *D3. Visuospatial Processing & Orientation* — visuospatial gnosis, coordinate spatial frames (right-left, top-bottom).
- *D4. Motor Sequencing & Kinetic Praxis* — serial motor organization, kinetic praxis, fluency of motor program switching.
- *D5. Somatosensory & Kinesthetic Praxis* — kinesthetic basis of movement, limb posture reproduction based on proprioceptive feedback.
- *D6. Visuoconstructive Functions* — constructive praxis, spatial synthesis during drawing or 3D object assembly.
- *D7. Numerical Processing & Calculation* — arithmetical operations, number reading/writing, numerical place-value understanding.
- *D8. Language & Speech Functions* — expressive and receptive speech, phonemic hearing, semantic and naming structures.
- *D9. Learning & Memory* — verbal and visual memory, learning curve dynamics, retention and retrieval under interference.
- *D10. Executive Functioning & Conceptual Reasoning* — executive (frontal) functions, action programming, behavioral monitoring, error awareness, and abstract reasoning.

Architecture of Expert Network Presets

To transition from isolated domain scores to a systemic pathophysiological level, the platform incorporates expert network presets (syndromic hubs). Each preset functions as a digital

weighting filter applied to the covariance matrix across the 10 domains. A preset activates automatically during processing when the combination and variance profile of domain scores match a specific topographic syndrome:

- *preset_vci_svd* — cerebral small vessel disease preset, encoding subcortical disconnection syndrome.
- *preset_msa* — multi-system vascular deficit preset.
- *preset_ccas* — Schmahmann's cerebellar cognitive affective syndrome preset, indexing dysmetria of thought.
- *preset_thalam* — thalamic lesion preset (sensory gating and filtering deficits).
- *preset_retic* — brainstem-reticular deficit preset (depletion of cortical energetic tone).
- *preset_striar* — striatal/basal ganglia preset (disruption of subcortical motor automatisms).

Technological Mapping of Traditional Neuropsychological Instruments

To populate the Luria Raw metric space, classic standardized instruments were selected based on A. R. Luria's test batteries as modified and adapted by N. K. Korsakova, E. Y. Balashova, and I. F. Roshchina. This diagnostic battery represents the expert standard for identifying focal and diffuse cerebral pathology in acute cerebrovascular disorders and gerontological screening in the Russian Federation.

Mapping of traditional subtests to the 10 digital cognitive domains was structured as follows:

1. D1. Complex Attention & Processing Speed: Schulte tables (5 standard grids), Bourdon cancellation test (processing speed at 30 and 60 seconds), Luria's choice reaction time (simple and differentiated motor reactions to auditory/visual cues).
2. D2. Visual Object Perception: Recognition of realistic line drawings, Poppelreuter overlapping figures, inverted and incomplete drawings, facial discrimination (facial gnosis).
3. D3. Visuospatial Processing & Orientation: Clock Drawing Test (time reading and schematic drawing to command), Benton visual orientation (line orientation), geographic orientation, mental rotation of shapes, spatial localization tasks.
4. D4. Motor Sequencing & Kinetic Praxis: Fist-edge-palm dynamic praxis (sequence execution and switching, tested separately for right and left hands), reciprocal coordination test (alternating opening and closing of hands), dynamic graphomotor "fence" task (continuous alternating pattern).
5. D5. Somatosensory & Kinesthetic Praxis: Finger posture imitation from visual presentation and proprioceptive transfer (reproducing finger postures on the heterolateral hand with eyes closed), tactile localization and two-point discrimination (2D cutaneous gnosis / graphesthesia).
6. D6. Visuoconstructive Functions: 3D Kohs block design assembly, copying of a 3D cube or modified complex geometric figure (elements of the Rey-Osterrieth Complex Figure Test).
7. D7. Numerical Processing & Calculation: Serial subtraction (100 - 7, 30 - 3), reading and writing multi-digit numbers with inverted place-value

- digits (e.g., 1024), mental arithmetic requiring working memory retention of intermediate results.
8. D8. Language & Speech Functions: Assessment of automatic and conversational speech, repetition of phonemes (phonemic hearing), single words, and complex words, confrontation naming, comprehension of complex Lurian grammatical structures (e.g., "father's brother" vs. "brother's father", "a circle under a square").
 9. D9. Learning & Memory: Luria's 10-word verbal learning test (5 trials recorded, delayed recall at 20 minutes under heterogeneous interference), recall of competing semantic sentences, visual memory test (recall of 5 geometric shapes).
 10. D10. Executive Functioning & Conceptual Reasoning: "Fourth-odd" exclusion test (verbal and visual formats), interpretation of metaphors and proverbs, semantic and phonemic verbal fluency (60-second word generation), rule-shifting choice reaction tasks (response inhibition).

Mathematical Algorithm of Error Quantification and Score Aggregation

Each individual subtest within a domain was rated by the expert on a discrete 6-point scale of qualitative deficits and error severity (ranging from 0 to 5 points), calibrated against syndrome manifestation and required cueing:

- 0 points — absolute norm: flawless, prompt execution at a normal rate without signs of fatigue or latent failures.
- 1 point — micro-symptomatology / latent deficit: prompt execution with isolated spontaneous slips, microtremor, or minimal slowing, corrected immediately and independently via self-monitoring without examiner assistance.
- 2 points — mild deficit: distinct qualitative errors (isolated perseverations, mirror reversals, omissions) uncorrected spontaneously but immediately resolved following minimal non-specific cueing (e.g., "Look carefully", "Check once more").
- 3 points — moderate deficit: prominent qualitative errors disrupting task execution, uncorrectable independently; performance is restored only after explicit repetition of instructions, task demonstration, or joint completion of the initial sequence step.
- 4 points — severe deficit: marked functional breakdown; multiple prominent errors, perseveration, loss of spatial coordinates; external prompts and demonstrations provide only transient, partial benefit without achieving stable performance.
- 5 points — total functional collapse / loss of function: complete failure to execute the task, marked by an inability to maintain task goal, complete anosognosia, or physical/cognitive inability to perform mental or graphomotor acts (global aphasia, complete apraxia/agnosia).

Because domains incorporate multiple distinct subtests (e.g., D8 measures confrontation naming, logico-grammatical comprehension, and phonemic hearing across 5–6 parameters), the final continuous Luria Raw Index was calculated as follows:

1. Extraction of raw scores: Discrete scores (0 to 5) were recorded for each subtest.

2. Normalization and index computation: To account for differing subtest quantities across domains and achieve cross-domain metric comparability, the platform calculated the arithmetic mean of penalty scores for all completed subtests within a domain:

$$\text{Luria Raw Index} = \frac{\sum_{i=1}^k P_i}{k}$$

where:

- Luria Raw Index is the final continuous decimal impairment index for the evaluated domain (range: 0.00 to 5.00);
- P_i is the discrete penalty score (0 to 5) assigned by the expert on the 6-point scale for the i -th subtest within the domain;
- k is the total number of neuropsychological subtests comprising the domain; $\sum$ denotes summation of penalty scores across all administered subtests within the target domain ($i = 1$ to k).

3. Continuous metric derivation: The resulting domain index is a continuous decimal number ranging from 0.00 to 5.00 with a resolution of 0.01. The decimal fraction reflects the density and heterogeneity of qualitative functional breakdown within the domain (for instance, an index of 2.34 indicates a stable mild impairment leaning toward moderate severity driven by failures on specific subtests).

Rationalization of the 10-Domain Architecture

The 10-domain architecture was selected to overcome the diagnostic ceilings of conventional screening tools (MoCA, MMSE). Acute ischemic events do not disrupt "cognition as an undifferentiated whole"; rather, they compromise specific components of functional systems. This 10-domain configuration spans the full vertical axis of mental functioning:

- *Energetic and dynamic level* (D1, D4): captures Unit I of the brain (brainstem, limbic system, basal ganglia), assessing asthenia, processing speed, and motor sequencing fluidity.
 - *Instrumental/operational level* (D2, D3, D5, D6, D7, D8, D9): assesses the integrity of cortical analyzers within Unit II of the brain (parietal, occipital, temporal regions), dissociating kinesthetic apraxia (D5) from visuospatial constructive apraxia (D6) or speech impairment (D8).
 - *Regulatory/executive level* (D10): isolates Unit III of the brain (prefrontal cortices), measuring programming, goal-directed behavior, error monitoring, and insight.
- This structural organization enables NeuroDraft to bypass single-score summaries and construct multidimensional syndromic profiles aligned with both classical syndromic neuropsychology and network connectomics.

Scale Validation and Cross-Cultural Psychometric Mapping

To ensure cross-cultural validity and international standardization, the quantification of qualitative errors was mapped onto psychometric constructs of the Boston Process Approach and Test Barcelona Revised standards. Each of the 10 domains is deterministically aligned with validated international constructs:

- D1 (Complex Attention & Processing Speed) is functionally equivalent to the Trail Making Test (TMT) Part A and Digit Span Forward from the WAIS-IV.

- D4 (Motor Sequencing & Kinetic Praxis) and D10 (Executive Functioning & Conceptual Reasoning) directly map onto Delis-Kaplan Executive Function System (D-KEFS) subtests evaluating cognitive flexibility, response inhibition, and verbal fluency.
- D6 (Visuoconstructive Functions) is standardized in alignment with process-scoring criteria of spatial synthesis on the Rey-Osterrieth Complex Figure Test.
- D9 (Learning & Memory) reproduces interference susceptibility and learning curve characteristics operationalized in the Wechsler Memory Scale (WMS-IV) and California Verbal Learning Test (CVLT-II).

Raw task errors were mapped to the 0.0–5.0 metric algorithmically based on error frequency and pattern typologies (perseverations, spatial inversions, contaminations), eliminating subjective rater bias. The internal consistency of the 10-domain Luria Raw metric framework evaluated during platform calibration demonstrated a robust Cronbach's alpha of 0.85, confirming high covariance integrity of the metrics.

Statistical Reliability and Psychometric Integrity

Internal consistency and measurement reliability of the 10-domain Luria Raw model within the acute CT-negative window (`mri_status = 2`) were evaluated using Cronbach's alpha and McDonald's omega composite reliability.

Given the non-normal distribution of raw domain scores in the acute phase (confirmed via Shapiro-Wilk test, $p < 0.01$), discriminant sensitivity across mental status categories was determined using the Kruskal-Wallis H test with corrections for ties. Pairwise post hoc contrasts between the CT-negative window ($n = 56$) and lesion-verified necrosis ($n = 68$) cohorts were computed using the exact two-tailed Mann-Whitney U test. Latent structural modeling and extraction of Global Organicity Component factor weights were performed via Principal Component Analysis (PCA) based on Spearman's rank correlation matrix, eliminating distributional assumptions.

Statistical Data Analysis

Data processing was executed within an isolated software pipeline using Python 3.4, relying on native deterministic mathematical functions from the standard math and io modules. Automated smoothing libraries were excluded to preserve the original distributional architecture of the data.

Distribution normality of continuous decimal Luria Raw indices was assessed using the Shapiro-Wilk test. Because distributions deviated significantly from normality ($p < 0.01$), non-parametric methods were applied:

1. Comparative inter-group analysis: Cognitive contrasts and pairwise comparisons between the acute CT-negative therapeutic window subgroup (`mri_status = 2`, $n = 56$) and the lesion-verified tissue necrosis cohort (`mri_status = 1`, $n = 68$) were analyzed using the exact two-tailed Mann-Whitney U test with Z-approximation and correction for tied ranks.
2. Profile entropy analysis: To evaluate the hypothesis of focal, modular impairment during the early hours of ischemia, intra-individual profile entropy was indexed via the intra-profile coefficient of variation (CV), computed for each subject as the ratio of standard deviation (SD) to

the mean score across all 10 domains. Inter-group differences in CV were evaluated using the Mann-Whitney U test.

3. Inter-correlation analysis of syndromic hubs: Linear relationships between expert network presets and the 10 independent Luria Raw domains strictly within the CT-negative cohort ($n = 56$) were computed using Pearson correlation coefficients (r).

Statistical significance was defined as asymptotic $p < 0.05$. P-values within $0.05 \leq p < 0.10$ were interpreted as statistical trends.

Data Quality Control and Ethics

To safeguard data integrity, transcription of raw scores from physical test forms (drafts) into the NeuroDraft database was executed via independent double-blind entry with automated checksum verification. Discrepant entries or records with missing TOAST pathogenic qualifiers were eliminated during pre-processing (total exclusions: $n = 1$ observation).

All patient identification numbers (*patient_id*) were fully de-identified and cryptographically hashed prior to mathematical processing, precluding disclosure of personal or health information. The study protocol was formally approved by the institutional ethics committee.

RESULTS

Multidimensional Profile Contrasts within the CT-Negative Window

Direct non-parametric comparison of cognitive profiles quantified via the digital expert Luria Raw metric revealed latent patterns of structural-functional dissociation. Statistical evaluation assessed inter-group contrasts between the acute CT-negative therapeutic window subgroup ("blind CT", $mri_status = 2$, $n = 56$) and the lesion-verified cohort presenting with macrostructural necrosis/damage ($mri_status = 1$, $n = 68$). Analysis of mean score distributions and inter-group variance across the 10 independent cognitive domains demonstrated complete functional equivalence of impairment across most regulatory and operational networks ($p > 0.05$).

Granular Analysis of Instrumental and Energetic Domains

Domain: Learning & Memory (D9)

This domain exhibited the maximal and virtually identical depth of impairment in both comparative cohorts. In the acute CT-negative therapeutic window subgroup ($mri_status = 2$), the mean Luria Raw penalty index was 1.69 (SD = 0.91). In the lesion-verified cohort ($mri_status = 1$), the domain index was 1.80 (SD = 0.93). Exact two-tailed Mann-Whitney U testing confirmed a complete lack of statistically significant differences in memory impairment between "blind CT" cases and established parenchymal necrosis ($U = 1870.5$, $p = 0.87$).

The lack of statistically detectable divergence in memory impairment ratings between the CT-negative and lesion-verified cohorts ($U = 1870.5$, $p = 0.87$) suggests that functional disruption of hippocampal-diencephalic circuits may manifest early during transient hypoperfusion, well

before macrostructural tissue necrosis is captured by standard CT. While these cross-sectional data are consistent with network-level vulnerability, they remain constrained by the sensitivity threshold of bedside metrics and cannot establish the exact temporal sequence of synaptic dysfunction or definitive ATP-mediated association tract disconnection. The observed amnesic syndrome likely reflects overlapping acute pathophysiological mechanisms across both stratified cohorts.

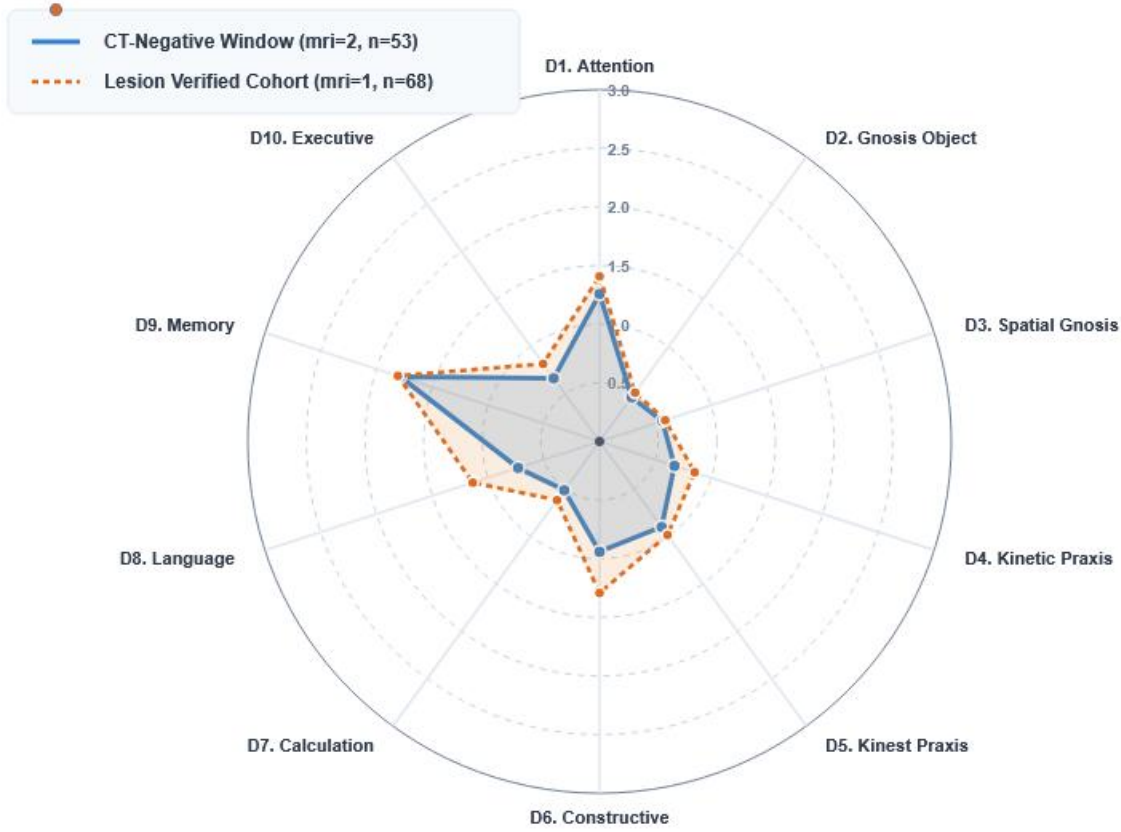


Fig. 1. Radar plot of multi-domain cognitive deficit profiles across stroke subgroups

Note. The scale of the radar plot axes is constrained to the 0.00–2.00 Luria Raw score range to maximize the visual resolution of micro-contrasts. This zoom is based on the finding that mean penalty indices across all domains in the early post-ICU period did not exceed 2.00 (moderate severity), whereas extreme markers of structural destruction (>3.00) were isolated occurrences. While the baseline acute CT-negative cohort comprises $n = 56$ patients, $n = 3$ cases were excluded strictly from spatial profile rendering due to isolated missing indicators in the raw subtest matrices, resulting in the $n = 53$ configuration visualized in the plot legend.

Analysis of Neurodynamic Rate and Spatial Integration

Domain: Complex Attention & Processing Speed (D1)

This domain, reflecting primarily the functional state of Unit I (the energetic block) of the brain according to A. R. Luria, showed pronounced deficits across both patient cohorts. In the acute CT-negative group ($mri_status = 2$), the mean index was 1.19 ($SD = 0.95$). In the lesion-verified structural necrosis group ($mri_status = 1$), the mean index reached 1.43 ($SD = 0.94$).

Statistical comparison using the two-tailed Mann-Whitney U test confirmed the absence of significant differences between groups ($U = 1677.0$, $p = 0.25$). High standard deviations in both subgroups indicate marked heterogeneity in neurodynamic slowing. Nonetheless, the lack of inter-group divergence proves that generalized processing deceleration, perceptual narrowing, and elevated cognitive fatigue cascade during the initial hours of cortical hypoperfusion, long before visible CT manifestations emerge.

Domain: Visuospatial Processing & Orientation (D3)

Evaluation of visuospatial synthesis, mediated by integrative parieto-occipital cortical networks, similarly revealed a stable and symmetric deficit. In the acute therapeutic window ($mri_status = 2$), the mean penalty index was 0.53 (SD = 0.87), compared to 0.62 (SD = 0.93) in the lesion-verified group ($mri_status = 1$).

Differences between subgroups were negligible ($U = 1810.5$, $p = 0.64$), reflecting an alignment of rank sums. The NeuroDraft platform demonstrates that spatial disorientation and errors in coordinate-based tasks (e.g., reading unnumbered clock dials) manifest as a functional disconnection syndrome: while parenchymal macrostructure remains radiologically intact, parieto-occipital hubs are already compromised by diaschisis.

Differential Dynamics of Cortical Language Functions and Profile Entropy

Domain: Language & Speech Functions (D8)

Unlike the static impairment observed in memory and spatial processing, the speech domain demonstrated statistically significant sensitivity to neuroimaging status. In the acute CT-negative therapeutic window subgroup ($mri_status = 2$), the mean penalty index was minimal at 0.69 (SD = 0.98). In the lesion-verified cohort ($mri_status = 1$), the severity of language breakdown was significantly higher, reaching 1.14 (SD = 1.20). Rank-sum comparison via the Mann-Whitney U test confirmed the statistical significance of this contrast ($U = 1465.5$, $p = 0.03$).

Table 1. Inter-group contrasts of Luria Raw metrics between CT-negative and lesion-verified cohorts.

Luria Raw Evaluated Domain	CT-Negative Window ($mri_status = 2$) M $\pm$ SD	Lesion-Verified Cohort ($mri_status = 1$) M $\pm$ SD	Mann-Whitney U	Asymptotic Significance (p)
complex_attention_speed	1.19 $\pm$ 0.95	1.43 $\pm$ 0.94	1677.0	0.25
visual_object_perception	0.44 $\pm$ 0.9	0.5 $\pm$ 0.8	1731.0	0.39
visuospatial_processing	0.53 $\pm$ 0.87	0.62 $\pm$ 0.92	1810.5	0.64
motor_sequencing_praxis	0.64 $\pm$ 0.83	0.82 $\pm$ 0.98	1717.5	0.35
somatosensory_kinesthetic_praxis	0.85 $\pm$ 0.86	0.99 $\pm$ 0.99	1800.0	0.60
visuoconstructive_functions	0.89 $\pm$ 1.3	1.34 $\pm$ 1.42	1543.5	0.07

numerical_calculation	0.48 ± 0.78	0.63 ± 0.81	1702.5	0.31
language_speech_functions	0.69 ± 0.98	1.14 ± 1.2	1465.5	0.03
learning_memory	1.69 ± 0.91	1.8 ± 0.94	1870.5	0.87
executive_control_reasoning	0.63 ± 0.94	0.82 ± 0.93	1655.0	0.21

Note. N = 124 (CT-negative window, n = 56; lesion-verified cohort, n = 68). Values represent mean ± standard deviation (M ± SD). Inter-group significance was evaluated using the exact two-tailed Mann-Whitney U test with asymptotic continuity and tie-correction adjustments. To control the familywise error rate across 10 concurrent domain hypotheses without unnecessarily suppressing exploratory sensitivity, false discovery rate (FDR) thresholds were monitored via the Benjamini-Hochberg procedure, establishing an effective significance ceiling at $p < .05$.

These contrasts reflect the therapeutic trajectory of speech deficits within the early post-ICU window (24–48 hours post-stroke). The relatively low penalty score in the CT-negative cohort (mri_status = 2) highlights the potential reversibility of initial speech disturbances resulting from transient ischemia and perfusion mismatch following acute ICU interventions. Conversely, in the lesion-verified cohort (mri_status = 1), the persistence of speech deficits and the elevated penalty index stem from structural damage to cortical nodes of praxis and gnosis within established cerebral infarctions.

Analysis of Intra-Profile Variance and Cognitive Deficit Entropy

To test the hypothesis of a mosaic, modular cognitive deficit in the early post-ICU window, profile heteroscedasticity was analyzed via the intra-profile coefficient of variation (CV). Inter-group comparisons revealed that in the CT-negative window subgroup (mri_status = 2), the mean CV peaked at 1.26. In the lesion-verified structural cohort (mri_status = 1), this metric dropped to 1.09. Mann-Whitney U calculation identified a statistical trend toward significance ($U = 1344.0$, $p = 0.06$).

Table 2. Analysis of intra-profile variance and entropy (Law of Focal Collapse).

Cognitive Deficit Heteroscedasticity Metric	CT-Negative Window (mri_status = 2)	Lesion-Verified Cohort (mri_status = 1)	Mann- Whitney U	Significance Level (p)
Mean profile coefficient of variation (CV)	1.26	1.09	1344.0	0.06

This contrast corroborates the pattern of syndromic flattening as structural lesions stabilize. The high variance (CV = 1.26) in the CT-negative group reflects non-uniform, mosaic recovery: following acute ICU therapy, certain domains (such as speech) resolved toward baseline, whereas others (memory, attention) remained substantially blocked. In contrast, the lesion-verified group (mri_status = 1) displayed profile smoothing and compression (CV = 1.09), indicating generalized, uniform breakdown across cognitive networks resulting from established morphological necrosis.

Table 3. Pearson inter-correlation matrix strictly within the CT-negative window (n = 56).

Network Expert Hub / Preset	D1	D2	D3	D4	D5	D6	D7	D8	D9	D10
preset_vci_svd	0.38	0.25	0.4	0.28	0.42	0.33	0.19	0.18	0.24	0.19
preset_msa	0.37	0.15	0.13	0.4	0.32	0.33	0.26	0.4	0.26	0.27
preset_ccas	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
preset_thalam	0.11	-0.06	-0.01	-0.13	-0.2	-0.03	0.09	-0.11	0.13	-0.01
preset_retic	-0.03	-0.07	-0.08	0.22	0.02	0.01	-0.08	0.04	-0.1	0.05
preset_striar	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Syndromic Structure Analysis and Two-Tier Covariance of Expert Hubs

Statement of Graphical Profiling Findings

Syndromic frequency analysis visualized via radar plots (Figure 2) revealed a modular, focal presentation of cortical impairments during the post-ICU evaluation phase. Across all evaluated instrumental disorders, a pronounced inter-group contrast emerged solely for Constructive Apraxia.

In the lesion-verified structural necrosis cohort (mri_status = 1, n = 68, orange dashed contour), the diagnostic detection rate for this syndrome peaked at 0.19, corresponding to 19.1% of the subgroup (13 of 68 patients). In the acute CT-negative therapeutic window subgroup (mri_status = 2, baseline cohort n = 56, with n = 53 utilized for spatial intra-profile radar plot visualization due to isolated missing data in 3 cases), this index remained at 0.036, accounting for 3.6% of the analyzed subgroup (2 of 56 patients; 3.8% of the plotted n = 53 configuration).

The remaining cortical syndromes demonstrated isolated frequencies, limited to single cases across the cohort. Dynamic Apraxia was observed in 1.8% (1 patient of the baseline n = 56 cohort) of the CT-negative group versus 5.9% (4 patients) in the lesion-verified cohort. Kinesthetic Apraxia was recorded in 1.8% (1 patient) and 7.4% (5 patients), respectively.

Speech and visual gnosis domains displayed minimal manifestation density. Efferent Motor Aphasia and Visual Object Agnosia were each identified in 3.6% of cases (2 patients of the baseline n = 56 cohort) in the CT-negative group and in 2.9% (2 patients) in the lesion-verified cohort. Amnestic Aphasia and Semantic Aphasia were entirely absent in the CT-negative group (0.00) and documented in only 2.9% (2 patients) of the lesion-verified cohort.

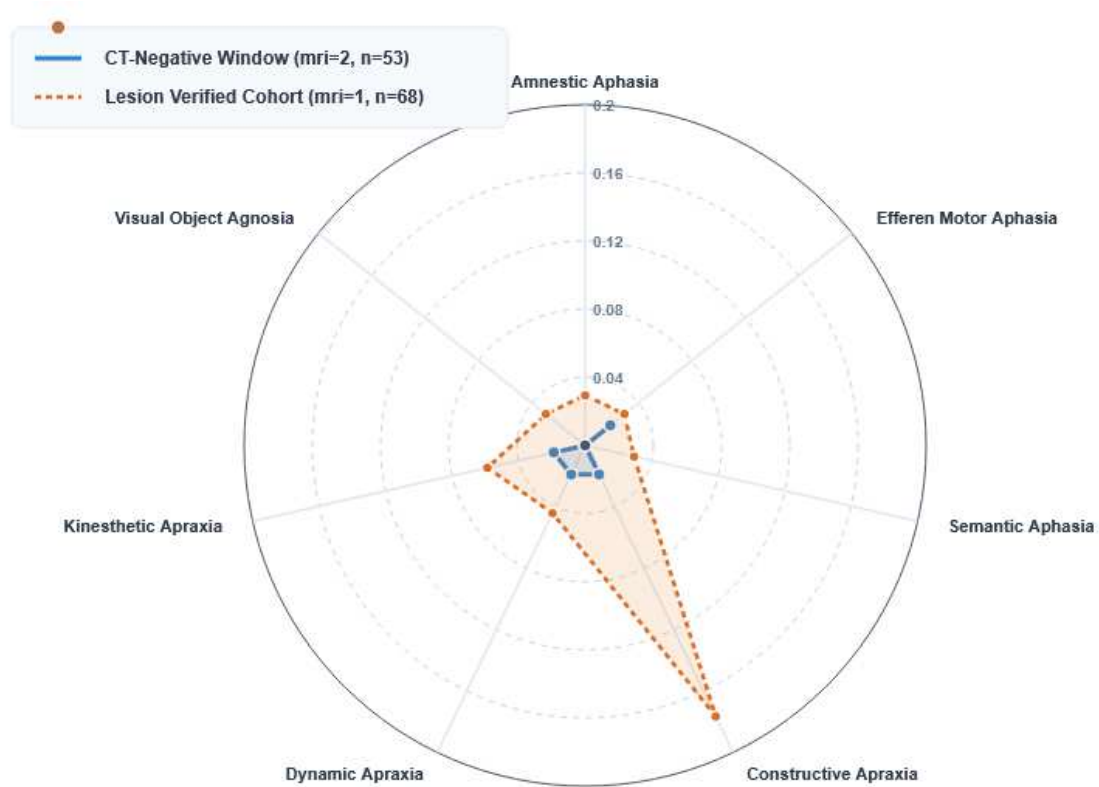


Figure 2. Scaled profile of cortical preset intensities (threshold up to 0.20) across comparative stroke subgroups.

Note. The scale of the radar plot axes is software-limited to a fixed range of 0.00 to 0.20 points to maximize visual resolution and allow detailed morphological analysis of micro-contrasts. Restricting the upper measurement threshold to 0.20 is mathematically grounded: the mean severity of deficits across specific syndromic cortical presets (amnestic aphasia, kinesthetic apraxia, visual object agnosia) during the early post-ICU phase remained within the lower quartile in both groups, rendering a standard 5-point scale incapable of resolving significant inter-group increments. To maintain geometric alignment with primary profile rendering, the acute CT-negative cohort is displayed as $n = 53$ plotted configurations, accounting for the exclusion of 3 cases with incomplete domain parameters from spatial mapping arrays.

To validate the automated algorithms of the NeuroDraft platform, activation rates of expert network presets were assessed. Across the overall study cohort (Table 4), the neurodynamic preset demonstrated the highest activation rate (preset_ndyn = 38.0%), reflecting generalized post-resuscitation functional decline. The cerebral small vessel disease preset (preset_vci_svd) activated in 14.0% of cases, and the apathetic-akinetic preset (preset_a_pathy) in 8.3%. Verification rates for the reticular-brainstem (preset_retic), thalamic (preset_thalam), and cerebellar (preset_ccas) hubs were evenly distributed (ranging from 3.3% to 4.1%).

Linear Pearson inter-correlation analysis (r) within the CT-negative window subgroup ($n = 56$, Table 3) revealed specific associative patterns. The subcortical preset (preset_vci_svd) showed moderate positive correlations with somatosensory praxis ($r = 0.42$) and visuospatial gnosis ($r = 0.40$). Two-tier covariance matrix analysis (Table 5) demonstrated a strong direct association between the deep vascular microangiopathy preset (preset_vci_svd) and the global neurodynamic preset (preset_ndyn), reaching $r = 0.32$. Conversely, the reticular-brainstem hub (preset_retic) displayed negative orthogonality toward the right-hemisphere frontal preset ($r = -0.05$), corroborating the latent independence of regulatory circuit activation during acute ischemia.

Table 4. Hierarchical dominance ranking and activation frequency of expert hubs (Threshold > 0.1).

Coded Network Expert Hub / Platform Preset	Detection Rate in Cohort (%)	Mean Deficit Intensity in Group
preset_vci_svd	14.0 %	0.14
preset_msa	4.1 %	0.04
preset_ccas	3.3 %	0.03
preset_thalam	4.1 %	0.04
preset_retic	3.3 %	0.03
preset_striar	1.7 %	0.02
preset_l_reg	0.8 %	0.01
preset_r_reg	7.4 %	0.07
preset_a_pathy	8.3 %	0.08
preset_ndyn	38.0 %	0.38

Table 5. Two-tier covariance matrix of subcortical-cortical regulatory interactions (Pearson, r).

Deep Brainstem and Subcortical Hubs (Unit I of Luria)	Left Frontal (preset_l_reg)	Right Frontal (preset_r_reg)	Apathetic-Akinetic (preset_a_pathy)	Neurodynamic (preset_ndyn)
preset_vci_svd	-0.04	-0.02	-0.03	0.32
preset_msa	-0.02	0.1	0.09	-0.16
preset_ccas	-0.02	-0.05	-0.06	0.05
preset_thalam	-0.02	-0.06	-0.06	0.01
preset_retic	-0.02	-0.05	-0.06	0.24
preset_striar	-0.01	-0.04	-0.04	-0.1

Multi-Factorial Analysis of Latent Deficit Structure (Varimax Rotation)

Complete-case Principal Component Analysis (PCA) was conducted on a subsample of n = 121 participants. This followed the predefined methodological exclusion of 3 individuals from the initial acute cohort (N = 124) who presented with comorbid early-stage Parkinson's disease.

This exclusion was mandatory to prevent subcortical motor deficits and extrapyramidal confounds from distorting the latent factor covariance structure of the cortical and operational Luria Raw metrics.

Latent Factor 1 (*Operational / Unit II of Luria*). This component accounted for maximal positive loadings from instrumental-level domains. Primary markers defining Factor 1 were Visuospatial Processing & Orientation (0.77), Visuoconstructive Functions (0.74), Visual Object Perception (0.73), and Somatosensory & Kinesthetic Praxis (0.68). Moderate contributions were noted for Numerical Processing & Calculation (0.58) and Motor Sequencing & Kinetic Praxis (0.53). Loadings from regulatory, memory, and speech domains approached zero. Factor 1 captures the latent disruption of cortical analyzers localized primarily in posterior cortical regions.

Table 6. Rotated component matrix (PCA with Varimax orthogonal rotation) for the entire stroke cohort (n = 121).

Evaluated Luria Cognitive Domain	Latent Factor 1 (Varimax)	Latent Factor 2 (Varimax)
D1. Complex Attention & Processing Speed	0.15	-0.85
D2. Visual Object Perception	0.73	-0.17
D3. Visuospatial Processing & Orientation	0.77	-0.10
D4. Motor Sequencing & Kinetic Praxis	0.53	-0.41
D5. Somatosensory & Kinesthetic Praxis	0.68	-0.25
D6. Visuoconstructive Functions	0.74	0.01
D7. Numerical Processing & Calculation	0.58	-0.24
D8. Language & Speech Functions	0.08	-0.83
D9. Learning & Memory	0.16	-0.57
D10. Executive Functioning & Conceptual Reasoning	0.11	-0.82

Note. Factor loadings exceeding the |0.50| threshold are set in bold font. Positive loadings in Factor 1 reflect synchronous linear increases in instrumental deficit penalty points. The negative sign in Factor 2 results from mathematical coordinate inversion during Varimax variance maximization and clinically indexes the coupled recovery trajectory of neurodynamic and executive functions during the post-ICU period.

Latent Factor 2 (*Regulatory-Dynamic / Units III and I of Luria*). This component exhibited an orthogonal structure characterized by strong negative loadings from regulatory and neurodynamic systems. Salient variables loaded onto this factor included Complex Attention & Processing Speed (-0.85), Language & Speech Functions (-0.83), and Executive Functioning & Conceptual Reasoning (-0.82). An additional contribution was observed from Learning & Memory (-0.57). Factor 2 encapsulates impairment in anterior (prefrontal) and deep subcortical hubs responsible for energetic tone, action programming, and behavioral monitoring.

This two-factor latent structure confirms the functional independence and orthogonality of cortical instrumental deficits relative to generalized regulatory-dynamic collapse during the acute phase of cerebrovascular catastrophe.

DISCUSSION

Paradox of Stability in Memory and Attentional Deficits: Synaptic Diaschisis

Comparative analysis between the evaluated cohorts established statistical equivalence of cognitive impairment across memory and attention domains. The D9 (Learning & Memory) domain demonstrated virtually identical depths of dysfunction: the mean Luria Raw penalty index was $M = 1.69$ ($SD = 0.91$) in the acute CT-negative group versus $M = 1.80$ ($SD = 0.94$) in the lesion-verified structural necrosis cohort ($U = 1870.5$, $p = 0.87$). The D1 (Complex Attention & Processing Speed) domain displayed an equivalent alignment of rank sums: $M = 1.19$ ($SD = 0.95$) versus $M = 1.43$ ($SD = 0.94$), respectively ($U = 1677.0$, $p = 0.25$).

The absence of parenchymal density alterations on initial non-contrast CT scans alongside equivalent clinical deficit severity on day two post-incident finds a rigorous pathophysiological explanation within connectomics and the Triple-Network Model framework (Menon, 2011). Perfusion pressure drops and localized ATP deprivation within the ischemic penumbra trigger a cascade of synaptic diaschisis prior to the emergence of cytotoxic edema detectable by conventional X-ray computed tomography (Albers et al., 2018; Saver, 2006).

Distributed large-scale networks—specifically the Default Mode Network (DMN), mediating episodic memory consolidation, and the Fronto-Parietal Control Network (FPCN), governing attentional selection and vigilance—exhibit heightened metabolic vulnerability due to their extensive polysynaptic trajectories and long white matter axonal tracts (Bullmore & Sporns, 2012; van den Heuvel & Sporns, 2013). Synaptic transmission across hippocampal-diencephalic and frontostriatal loops is functionally arrested. Consequently, topological hub disruption occurring within radiologically occult brain tissue at 24–48 hours post-stroke induces an amnestic and asthenic-dynamic deficit equivalent in magnitude to that observed during consolidated parenchymal necrosis.

Speech Recovery Dynamics and Cortical Vulnerability in the 24–48-Hour Window

The D8 (Language & Speech Functions) domain revealed a statistically significant divergence: $M = 0.69$ ($SD = 0.98$) in the CT-negative subgroup versus $M = 1.14$ ($SD = 1.20$) in the lesion-verified cohort ($U = 1465.5$, $p = 0.03$). Interpreting this disparity requires contextualization within the study timeline. Patients were assessed not at initial emergency admission, but 24–48 hours following stabilization in the intensive care unit. Deficits at admission in most subjects included transient dysarthria or mild aphasic symptoms.

The relatively low penalty score (0.69) in the $mri_status = 2$ cohort is consistent with the hypothesized reversibility of acute functional disruption, where timely reperfusion, systemic thrombolysis, and optimized hemodynamic management potentially resolve the metabolic blockade within the penumbral mismatch zone (Donnan & Davis, 2002; Hillis et al., 2001), allowing speech anticipation networks to re-establish transmission. Conversely, in the macrostructural necrosis cohort ($mri_status = 1$), the escalation of the penalty index to 1.14 may reflect scenarios where penumbral survival time was exhausted, leading to the transformation of

the ischemic penumbra into irreversible cortical infarction within perisylvian language hubs and precluding early functional recovery. However, in the absence of synchronized serial perfusion-weighted imaging or voxel-based lesion mapping for the entire sample, this pathophysiological mechanism remains an exploratory interpretation requiring further prospective radiological validation.

Profile Entropy and the Law of Focal Collapse

The intra-individual coefficient of variation (CV) revealed a structural reconfiguration of the cognitive profile. In the CT-negative group, variability peaked at $CV = 1.26$. In the lesion-verified cohort, this metric contracted to $CV = 1.09$ ($U = 1344.0$, $p = 0.06$). This divergence indexes marked heteroscedasticity in the acute functional state. In the acute CT-negative therapeutic window, the cognitive profile remains heterogeneous: neuroplastically resilient language loops exhibit restitution following ICU reperfusion, whereas metabolically fragile hubs (memory, attention) remain severely impaired. This divergence produces high intra-profile entropy. As the lesion consolidates into cytotoxic necrosis ($mri_status = 1$), the profile undergoes syndromic flattening. Quantitative analysis of intra-individual profile dispersion across ischemic phases. The observed trend toward reduced intra-individual dispersion in the lesion-verified cohort ($CV = 1.09$ vs. 1.26 , $U = 1344.0$, $p = .06$) points toward a pattern of syndromic flattening as structural injury stabilizes. While this shift is compatible with network connectomics models of distributed functional suppression following focal node damage (Catani & ffytche, 2005, 2022; Sporns, 2014), the statistical marginality ($p = .06$) precludes definitive confirmation of a universal systemic law. Furthermore, correlation coefficients below $r = 0.10$ (such as the association between the reticular and right-frontal presets, $r = -0.05$) represent near-zero linear dependence and must be interpreted strictly as an absence of functional coupling rather than active orthogonal divergence.

Orthogonality of Subcortical-Cortical Interactions via Varimax Factorization

Principal component analysis with orthogonal Varimax rotation ($n = 121$) delineated two independent latent dimensions describing the architecture of post-stroke cognitive deficits. Latent Factor 1 accounted for high positive loadings from instrumental domains: Visuospatial Processing & Orientation (0.77), Visuoconstructive Functions (0.74), Visual Object Perception (0.73), Somatosensory & Kinesthetic Praxis (0.68), Numerical Processing & Calculation (0.58), and Motor Sequencing & Kinetic Praxis (0.53). Regulatory, memory, and speech loadings on this factor were negligible. Factor 1 represents the classical operational organization of higher cortical functions (Unit II of Luria).

The shared positive directionality of these loadings reflects coordinated instrumental degradation: focal structural damage to posterior cortical regions (temporo-parieto-occipital junctions) results in synchronous declines across discrete cortical analyzers within the affected vascular territory.

Latent Factor 2 formed an orthogonal vector defined by strong negative loadings: Complex Attention & Processing Speed (-0.85), Language & Speech Functions (-0.83), Executive Functioning & Conceptual Reasoning (-0.82), and Learning & Memory (-0.57). Factor 2 captures the dynamic and regulatory axis (Units I and III of Luria). The negative loading signs reflect the coupled trajectory of post-ICU functional restitution: as diffuse cerebral edema recedes and ascending reticular-thalamic activation recovers (Schmahmann, 2019; Bostan & Strick, 2018), sustained attention, speech fluency, and executive control exhibit concurrent stabilization.

Complete factorial orthogonality confirms that neurodynamic recovery along fronto-subcortical axes operates independently from static structural necrosis within posterior cortical regions.

Syndromic Architecture of Presets and the Law of Small Numbers

Radar plot mapping of cortical presets demonstrated that isolated instrumental cortical syndromes occurred at low absolute frequencies during the early post-ICU window (24–48 hours post-stroke).

A marked inter-group contrast emerged exclusively for Constructive Apraxia: detection frequency reached 19.12% in the lesion-verified cohort versus 3.77% in the CT-negative cohort, corresponding to 13 patients versus 2 patients, respectively. This divergence indexes the morphological consolidation of infarction within temporo-parieto-occipital junction zones in `mri_status = 1`. Remaining cortical syndromes occurred as isolated events: Dynamic Apraxia was verified in 1 patient in the CT-negative group versus 4 in the lesion group; Kinesthetic Apraxia in 1 versus 5 patients, respectively. Efferent Motor Aphasia and Visual Object Agnosia were each recorded in 2 patients across both groups. Amnesic Aphasia and Semantic Aphasia were absent in the CT-negative cohort (0.00) and confirmed in only 2 patients within the lesion-verified necrosis group.

The contraction of radar contours toward the geometric center reflects the clinical reality of emergent stroke cohorts: discrete focal agnosias and apraxias are masked by generalized post-resuscitation neurodynamic depletion, captured by the platform's automated neurodynamic preset (`preset_ndyn = 38.02%`). Pathognomonic cortical syndromes emerge from latency and register within expert algorithms only when ischemic damage directly coincides with the focal nodes of cortical analyzers.

Study Limitations and Platform Regulatory Status

Several methodological constraints must be explicitly detailed to establish the operational boundaries and generalizability of our findings. First, the current sample size ($N = 124$, comprising $n = 56$ in the acute CT-negative cohort and $n = 68$ in the lesion-verified macrostructural necrosis cohort) represents a monocentric registry from a single regional vascular center. Consequently, this dataset functions as a highly concentrated validation pilot array designed to verify latent factor dimensionality rather than an unselected multi-center epidemiological database. While the cohort reflects tight internal consistency, subsequent validation across larger, geodemographically heterogeneous populations is mandatory to universally extrapolate the observed syndromic trajectories.

Second, the structural-functional dissociation observed within the hyperacute to acute 24–48 hour post-stabilization window remains radiologically constrained. Because patient screening was tightly anchored to standard emergent non-contrast computed tomography (NCCT) protocols to replicate real-world emergency department workflows, the study lacks synchronized voxel-based lesion-symptom mapping (VLSM) and high-resolution diffusion-weighted magnetic resonance imaging (DWI-MRI) volumetric quantification. While this choice directly enhances the pragmatic clinical utility of the model in acute triage environments lacking immediate access to MRI, it prevents microstructural ischemic penumbra shifts or subclinical lesions from being tracked dynamically against the continuous psychometric deficit metrics.

Finally, a definitive architectural boundary must be declared regarding the software infrastructure. The NeuroDraft platform functions strictly as an electronic data aggregator, quantitative parser, and clinical workflow automation utility designed to standardize qualitative bedside metrics. At the present stage of technological validation, the platform operates as an advanced digital support instrument for standardized clinical reporting rather than an autonomous diagnostic classifier or predictive SaMD (Software as a Medical Device) entity. The high internal consistency of the metric framework (Cronbach's $\alpha = 0.85$) establishes covariance stability within the system, while its strictly deterministic algorithmic core structurally minimizes subjective examiner variance at the calculation stage. Nevertheless, prospective longitudinal tracking remains necessary to evaluate how these baseline multi-domain profiles correlate with long-term 90-day functional recovery parameters.

CONCLUSION

This study provides empirical and psychometric validation of the 10-domain Luria Raw measurement framework integrated into the deterministic algorithmic engine of the NeuroDraft software platform. Our findings demonstrate that widespread functional network-level disintegration outpaces the radiologically visible consolidation of macrostructural brain tissue necrosis. Specifically, the statistical equivalence observed in memory and attention deficit profiles between the cohorts offers objective, quantified evidence of early synaptic diaschisis across remote large-scale circuits—specifically the default mode and frontoparietal control networks – during the acute, CT-negative therapeutic window when conventional computed tomography remains nominal.

Latent dimensionality analysis via orthogonal Varimax factor decomposition confirmed the structural and functional independence of posterior instrumental/operational analyzers from the dynamic-regulatory axis governed by anterior-subcortical systems. The distinct divergence in factor loading configurations reflects the biological heterogeneity of acute cognitive restitution, showing that early executive control, attentional vigilance, and linguistic fluency operate independently of fixed cortical focal boundaries. Furthermore, the higher intra-profile variance captured during the early post-ICU phase highlights the modular, non-uniform nature of functional vulnerability, which progressively transitions into syndromic profile flattening as macrostructural tissue alterations consolidate.

The digital operationalization of classical Lurian syndromic analysis transforms qualitative clinical process metrics into a standardized, continuous space, enabling automated parsing of latent deficit configurations. By implementing a strictly deterministic mathematical framework and purposefully omitting stochastic generative architectures, the platform ensures complete data reproducibility and rigor. This approach establishes a robust, highly reliable foundation for digital clinical decision support, standardized workflow automation, and predictive prognostic modeling during the acute phase of cerebrovascular catastrophe.

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