From Principle to Function: The Causal Core in the Brain

Introduction

The theory of the causal core describes a centristic-causalistic principle that emerges as a universal structural form in open, dynamic systems. This principle is not a material object, but rather a systemic necessity that, under certain conditions, leads to the formation of a central integration mechanism. In biological evolution, this mechanism has manifested as the cell nucleus and later as the brain. This essay investigates how this causal structural principle is concretely realized in the brain, without reducing it to an anatomical region or a "switch." The analysis connects thermodynamic, neurophysiological, and systems-theoretical perspectives and aims to identify causal collapse as the physiological manifestation of the causal core.

1. The Causal Core as a Thermodynamic-Functional Structural Principle

In complex, open systems, energetic imbalances give rise to structural attractors: centers with maximum reaction density. These "hotspots" bundle causal pathways, coordinate system activity, and form a kind of inner center. The causal core is not an ontological entity, but rather the result of thermodynamic and functional self-organization.

The central conditions for the formation of a causal core are:

- Existing flow gradients (e.g., chemical, electrical, metabolic)
- Nonlinear coupling of reactions (autocatalytic amplification)
- System openness with simultaneous membrane closure
- Persistent feedback and plastic adaptability

These conditions do not lead to a static center, but to a functionally operative locus of relative centralization—a zone of maximum efficacy where various causal streams are coordinated. In evolution, this principle first materialized in the cell nucleus of eukaryotes, forming a functionally condensed control center within the cell.

2. The Brain as an Evolutionary Expression of the Causal Core

In the further course of evolution, the centristic-causal principle has manifested in increasingly complex organizational forms. The brain represents the highest differentiation of this principle to date. It is:

- Thermodynamically active (metabolism, energy consumption)
- Plastic (structurally modifiable through learning and development)
- Functionally integrated (high network connectivity)
- Recursively structured (circular information processing)

The brain is thus **not the origin of the causal core**, but rather its **most complex biological manifestation**. It forms an environment within which secondary functional centers can emerge that recursively continue the principle of the causal core.

3. From Center to Subcenter: The Leap to Functional Networks

The central question now reads: **How does the causal core manifest within the brain itself?**

The answer is crucial: There is no structural location that can be identified as the "heart" of this system. Instead, a **functional core** emerges through dynamic integration:

- e.g., in the **Default Mode Network (DMN)**
- In thalamo-cortical networks
- Through context-dependent oscillation patterns and synchronization

These functional centers emerge—analogous to thermodynamic hotspots—through recursive information flows, connectivity maxima, oscillation coherence, and functional plasticity.

4. No Structure, but Function: The Functional Causal Core in the Brain

Analogous to an automobile engine, whose "heart" cannot be reduced to a single component but lies in the coordination of components, the functional causal core in the brain emerges through:

- Transient synchronization of highly networked nodes (e.g., precuneus, medial PFC)
- Dynamic reorganization during attention shifts
- Global integration of self-referential contents

The centralization occurs **not spatially**, but **systemically**: through emergent patterns of maximum connectivity and functional integration with simultaneous plastic reversibility.

5. Causal Collapse as the Physiological Manifestation of the Principle

The concrete manifestation of the causal core in the brain is **causal collapse**: the moment when divergent activity patterns converge in a coherent, functionally integrated state.

This occurs particularly:

- In states of reflexive attention
- During transitions between external and internal modes
- In decisions, self-modeling, or imagination

Physiologically manifested as:

- Coordinated oscillations (especially gamma and beta waves)

- Dynamic reconfigurations (fMRI-supported network analyses)
- PCI changes during loss or gain of consciousness (Massimini et al.)

6. Collapse as Functional Centralization Rather Than Metaphysical Emergence

The collapse is the moment when the centristic structural principle of the causal core becomes operative—as a transformation from multiplicity to unity, from potential to coherence, from neuronal dispersion to systemic integration. It involves the transitional state in which an open, plastic system temporarily brings together its distributed functions into a centralized, integrating activity pattern. This process marks not maximum causality in an abstract sense, but functional coherence through the bundling of causal pathways within the system itself.

7. Distinction from Established Theories

In contrast to theories such as the Global Neuronal Workspace (Dehaene, Changeux) or Integrated Information Theory (Tononi), the model developed here understands consciousness **not as a product of information availability** or as a quantifiable measure of integration, but as a **state of functional centralization** in the sense of a **structural transformation principle**.

- IIT speaks of structural complexity (Phi)
- GWT of global access
- This model, however, of a **causal transformation in situ**, conditioned by recursive integration

8. Empirical Perspectives and Distinguishing Features of the Theory of Causal Collapse

The theory of causal collapse claims to capture a fundamental dynamic of consciousness that goes beyond established integration models. It thus raises the claim to empirical relevance and theoretical distinguishability. The following discusses four central touchstones: predictive power, temporal structure, pathological manifestations, and experimental differentiation possibilities.

8.1 Predictive Power

The theory differs from other integration approaches such as Global Workspace Theory (GWT) or Integrated Information Theory (IIT) by not merely describing integration as such, but by assuming a specific transformation point at which an open, plastic system transitions into a state of maximum internal causality. This causal collapse is not continuous, but a nonlinear-recursive transition. This yields the following concrete predictions:

- **Not just integration, but transformation:** The collapse describes a sudden condensation of systemic causality, visible e.g., in transitions between consciousness states (awakening from anesthesia, onset of epileptic seizures, dissociation).
- **Increase in functional density:** The collapse is accompanied by an increase in local reaction density, which should be reflected in entropy or complexity metrics (e.g.,

Perturbational Complexity Index [PCI], synergistic integration according to Luppi et al. 2023).
- **Internal control before representation:** While many models emphasize external representation, causal collapse focuses on internal system coherence. Therefore, it is expected that in pathological states, external world perception remains intact, but self-control breaks down (e.g., in dissociative states).

8.2 Temporal Structure of Collapse

The temporal dimension of causal collapse lies in the range of biological integration processes and should typically be between **100-1000 milliseconds**. The collapse is to be understood as a process-like transformation—analogous to phase transitions in complex systems—and not as a punctual event. Empirical reference points:

- **P300 component** of EEG response as a marker of conscious processing (~300 ms)
- **Synchronization jumps** in EEG or MEG during sudden consciousness changes
- **Slower basal shifts** during transitions between REM sleep and deep sleep

8.3 Pathology: Disorders of the Collapse Principle

Disorders of causal collapse manifest in three typical forms:

- **Missing system coherence:** In dissociative states or severe depression, external world perception remains intact, but the feedback to the system center is missing (ego loss, derealization).
- **Localized or rigid collapse zones:** In obsessive-compulsive disorders or autism, over-stabilized but not plastically networked nodes appear that determine system behavior.
- **Unstable transitions:** Aura phenomena before migraine or epileptic seizures indicate incomplete or excessive collapse processes.

Clinical hypothesis: In patients with depersonalization, there is preserved sensory integration, but disturbed feedback to the systemic center. This could manifest in disturbed re-entrance or weakened medial prefrontal activity.

8.4 Experimental Differentiation Approaches

For empirical testing of the theory, the following approaches can be formulated:

1. **Consciousness dynamics analysis:**
    - Combination of PCI, Integrated Information Decomposition, and re-entrance measurement (e.g., MEG, fMRI) during consciousness transitions (anesthesia, REM sleep, psychedelics).
    - Prognosis: Causal collapse manifests in abrupt, system-wide integration effects with thermodynamic feedback.
2. **TMS+EEG stimulation experiments:**
    - Goal: Differentiation of local versus systemic integration through focal stimulation.
    - Prognosis: Only with systemic reaction does recursive self-amplification of activity appear—this would be an indication of a collapse mechanism.
3. **Analysis of self-perception disorders:**

    - Case studies on anosognosia or hemineglect, where external information is preserved but systemically not re-integrated.
    - Hypothesis: Missing causal collapse prevents feedback to the ego center.

These empirical perspectives open the possibility of establishing causal collapse not only as a theoretical structural principle, but as a differentiable neurobiological phenomenon.

9. Remaining Challenges and Empirical Perspectives

In the further development of the theory of causal collapse, three central challenges arise that are crucial for an empirically robust and distinguishable formulation from competing approaches: the distinction from general phase transitions in complex systems, the question of possible quantification of collapse, and the ontogenetic development of this ability. This chapter is dedicated to the systematic clarification of these open points.

9.1 Distinction from Criticality

In the literature on complex systems, the concept of *critical states* or *Self-Organized Criticality* (Bak et al.) is frequently used to describe phase transitions between order and chaos. These states are characterized by scale invariance, sensitivity to disturbances, and emergent patterns. Causal collapse differs from this through several features:

- It describes not the transition state, but a *local state of maximum causal condensation* within an open, plastic system.
- While criticality is typically a *global, system-wide phenomenon*, collapse concerns *punctual, functionally selected centers* that dynamically emerge and disappear.
- Collapse is not arbitrary, but *teleonomically* oriented: It serves to stabilize internal control processes through recursive integration.

As a heuristic distinguishing criterion: **Criticality** denotes the *open state of maximum possibility density*, while **causal collapse** describes the *closed state of maximum reality density*.

9.2 Quantification of Causal Collapse

A decisive step toward empirical testability of the theory consists in developing metrics with which collapse can be operationalized and numerically captured. The following approaches are promising:

- **Synergetic information measures** (e.g., "O-Information" or Integrated Information Decomposition according to Luppi et al., 2023): These allow the distinction of redundant, synergistic, and transferred information between subsystems and could map collapse-like condensations.
- **Perturbational Complexity Index (PCI)**: As developed by Massimini et al., PCI describes the systemic complexity of response to stimulation. A high PCI correlates with conscious state and could serve as an indicator for functional nodes.
- **Effective connectivity / Transfer entropy**: Methods like Granger causality or model-based connectivity analyses allow statements about directed information flows and their recursivity.

- **Topological markers in functional networks**: Centrality measures, hubs, cluster coefficients, and recursive cycles could provide information about collapse centers.

The central assumption reads: Collapse manifests in a state of **local maximum directed causal networking**, characterized by high *recursion depth*, *functionality*, and *coherence*.

9.3 Ontogenesis of Collapse

The emergence of the ability for causal collapse in the course of individual development (ontogenesis) is another decisive point for empirical foundation of the theory.

- In early childhood, only *insular, peripheral collapse processes* can be observed, such as in sensorimotor coordination centers.
- With the maturation of thalamo-cortical cycles and interoceptive networks (including Default Mode Network), increasingly recursive loops emerge that enable self-modeling.
- Cognitive leaps (e.g., Piaget's transition to the symbolic phase) correlate with the ability to stabilize *complex internal causal nodes*.

Ontogenetically, a transition emerges from reactive survival processes to *intentional, internally controlled dynamics* that are systemically centered and selectively focused through causal collapse.

9.4 Outlook

Answering these three challenges is not only crucial for the plausibility of the theory, but also enables a clear empirical dividing line from existing integration theories. The concept of causal collapse thereby gains both theoretical depth and experimental operationalizability—and can thus be established as a new, distinct structural principle in consciousness research.

10. Conclusion: From Principle to Manifestation

The causal core is a universal structural principle that has manifested in evolution as the brain. Within this brain, it appears not as a localizable place, but as a recurring tendency toward functional centralization. This tendency is realized in causal collapse:

The collapse is the moment when the principle of the causal core becomes operative—as a temporary state of structural coherence that enables causal integration within the system.

The search for a "heart of the heart" thus leads not to a structure, but to an understanding:

- That centralization is a systemic dynamic
- That consciousness represents a form of functional self-coherence
- And that causal collapse is the physiological expression of a much deeper lying principle that first enables life, cognition, and self-modeling

It is not a material center, but a dynamic principle of systemic self-centralization: a *centralistic-causal mechanism* that emerges in open, plastic systems through recursive integration. Its manifestation in the brain allows biological organisms to generate internally

coordinated states of maximum causality—in the form of a temporary "collapse" of divergent activity into a coherent action node.

This ability for functional centralization establishes a form of **relative autonomy** that is neither deterministically closed nor metaphysically free. The so-called *free will* is, from this perspective, not a transcendent faculty, but the result of recursive, system-internal self-focusing—enabled by the causal core as a structural principle of emergent control. Freedom of will is then nothing other than the *ability of a system to causally focus itself in the moment of decision*, independent of external control.

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