POLIS V12 and the Tensional Approach to Major Medical Problems

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Abstract

Classical medicine describes mechanisms at molecular, cellular and systemic levels but rarely provides a unified causal ontology for why a disease appears, progresses or remits. POLIS V12, a fully closed tensional conservation theory, models every system as a polis composed of three meshes (solid, liquid, gaseous). All real systems satisfy the closure condition $\varepsilon = \sum K_m(2+K_m) = 0$, and their state is quantified by the disequilibrium index $\mathrm{IDT}^* \in [0, 1)$. This paper applies the tensional ontology to seven major medical problems: cancer, autoimmune diseases, neurodegeneration, aging, genetic disorders, chronic pain, and organ failure. For each condition, we identify the affected meshes, reinterpret pathological observations in terms of K_m anomalies and phase transitions, and propose therapeutic strategies derived from restoring tensional equilibrium. The RMCE cycle (Reception, Memorisation, Comparison, Execution) is used to model immune discrimination, neural processing and cellular signalling. Explicit falsification criteria are given for each application. No clinical data are fabricated; only the tensional reading of known phenomenology is provided.

Contents

1 Introduction

Classical medicine has accumulated vast empirical knowledge, but a unifying causal framework remains absent. POLIS V12 provides such a framework. In this ontology, a human being is a polis with three meshes:

- **Solid mesh**: bones, fixed organs, DNA, cytoskeleton, extracellular matrix – the structural anchor.
- **Liquid mesh**: blood, lymph, interstitial fluid, synaptic transmitters – the tensional buffer that redistributes tension.
- **Gaseous mesh**: breath, metabolic gases, thermal dissipation, symbolic activity – the expanding boundary.

Any deviation from equilibrium is expressed by the sum of tensional residuals:

$$\varepsilon = \sum_m K_m(2 + K_m) = 0$$

where $K_m = (v_m - T)/(v_{\max} - T) \in [0, 1]$ and $T = K_{\min}$ is the tensional origin. The index:

$$\mathrm{IDT}^* = \frac{\sum x_m}{1 + \sum x_m}, \qquad x_m = K_m(2 + K_m)$$

measures the structural distance from equilibrium. For all real systems, $\mathrm{IDT}^* \approx 0.85$ (Phase 4). A disease is a local or global deviation from this normal tensional pattern, often accompanied by a shift to Phase 3 (saturation) or Phase 4 (explosive rupture without closure).

2 Cancer as an Uncontrolled Phase 4 Explosion

Affected mesh: solid mesh (nuclear and cytoskeletal architecture), secondarily liquid mesh (signalling) and gaseous mesh (metabolic waste).

A normal cell maintains K_m within a range that allows apoptosis when $\sum x_m$ cannot be minimised. Cancer arises when a somatic mutation increases the local K_m of the solid mesh beyond the reversible limit, and the cell loses the ability to execute Phase 5 (reorganisation) or Phase 6 (healing). Instead, it enters a self-sustaining Phase 4 explosion: uncontrolled division, invasion and metastasis.

The closure condition for healthy tissue is $\varepsilon_{\text{tissue}} = \sum_{\text{cells}} K_{\text{cell}}(2 + K_{\text{cell}}) \approx 0$. In cancer, certain cells have $K_{\text{cell}} \gg K_{\text{normal}}$, and the tissue IDT^* increases. Classical therapies (chemotherapy, radiation) add an external tensional load that drives Phase 4 cytotoxicity without restoring Phase 5 reorganisation.

Tensional therapeutic strategy:

- Restore apoptotic pathways (lower K_{nuclear} via epigenetic modulation).
- Re-establish contact inhibition (increase $VT = K - T$ between adjacent cells via cell-adhesion molecules).
- Use low-dose, periodic stressors (metronomic chemotherapy) to induce Phase 5 reorganisation rather than Phase 4 explosion.

Falsification: If a tumour with documented high IDT* (measured from histopathological features) does not show a reduction in IDT* after a tensional intervention designed to lower K_m, the application to that cancer type is falsified.

3 Autoimmune Diseases as Liquid Mesh Confusion

Affected mesh: liquid mesh (immune system, lymphatic flow, cytokine networks).

The immune system is a distributed polis whose function is to distinguish K_{self} from $K_{\text{non-self}}$. This distinction is the Comparison step in the RMCE cycle. Autoimmunity occurs when the Comparison threshold is shifted: K_{self} is misread as $K_{\text{non-self}}$. The liquid mesh accumulates residual tension (high IDT* of the immune network) because the closure condition across the organism includes false mismatches. The result is chronic inflammation and failure to reorganise (Phase 5 stuck in a loop of low-level Phase 4 events).

Tensional therapeutic strategy:

- Restore a stable memory of self-patterns via tolerogenic dendritic cells presenting self-antigens with low VT signals.
- Modulate the sampling window of immune receptors so that small K_m fluctuations are not interpreted as threats.
- Use vagus nerve stimulation to encourage Phase 5 reorganisation of the liquid mesh.

Falsification: If a well-characterised autoimmune disease shows no change in serum cytokine IDT* (computed from a cytokine panel) after a tensional protocol designed to lower immune network tension, the autoimmune branch is falsified.

4 Neurodegeneration as Loss of Solid Mesh Coherence

Affected mesh: solid mesh of neurons (synaptic architecture, protein aggregates) and liquid mesh (cerebrospinal fluid, neurotrophic gradients).

Neurodegenerative diseases (Alzheimer, Parkinson, Huntington) are characterised by misfolded protein accumulation – regions of abnormally high K_m in the solid mesh that resist degradation. The solid mesh can no longer reorganise (Phase 5) because the protein aggregates act as tensional traps. The liquid mesh (CSF flow, glymphatic system) cannot flush them out. Local $x_m = K_m(2 + K_m)$ increases, pushing neurons into Phase 4 (apoptosis) or Phase 7 (degeneration without repair).

Tensional therapeutic strategy:

- Enhance the glymphatic system (liquid mesh) by promoting slow-wave sleep (natural Phase 5 for the brain).
- Use rhythmic sensory stimulation (light, sound) to drive synchronised neuronal firing, reducing protein aggregates by applying uniform VT across the affected area.
- Intermittent fasting changes the gaseous mesh (metabolic substrate), lowering K_m of the solid mesh.

Falsification: If a mouse model of Alzheimer shows no correlation between sleep EEG coherence (surrogate for Phase 5 brain reorganisation) and amyloid-beta clearance, the tensional mechanism is falsified for that model.

5 Aging as Accumulation of Tensional Residuals

Affected mesh: all three meshes.

Aging is not a disease; it is the gradual increase of the global IDT* of the human polis. The solid mesh accumulates cross-links, epigenetic noise and somatic mutations. The liquid mesh loses fluidity. The gaseous mesh becomes less efficient. The closure condition becomes harder to approximate because the global sum slowly drifts upward. Death occurs when the gaseous mesh can no longer sustain VT exchange with the environment – the polis can no longer reorganise and closes permanently.

Tensional therapeutic strategy:

- Exercise and hydration increase liquid mesh flow, temporarily lowering global IDT*.
- Caloric restriction reduces metabolic load on the solid mesh (lower K_m base).

- Senolytic drugs remove cells with excessively high K_m (senescent cells), reducing $\sum x_m$.

Falsification: If a longitudinal study shows no monotonic increase in a tensional ageing biomarker (weighted sum of epigenetic and inflammatory markers normalised as K_m) with chronological age, the tensional model of ageing is falsified.

6 Genetic Disorders as Anomalous Structural K_m Values in DNA

Affected mesh: solid mesh (DNA sequence and chromatin organisation).

A mutation changes the local K_m of the solid mesh. Most mutations are neutral because the tensional mesh can accommodate small K_m variations, buffered by non-coding DNA which functions as a tensional reservoir. Disease arises when the mutation produces a K_m outside the tolerable range. The non-coding DNA is not junk – it is the tensional buffer of the solid mesh (Applications of POLIS, Doc.04).

Tensional therapeutic strategy:

- Modulate the epigenetic environment (liquid mesh) to compensate for the anomalous K_m. In sickle cell disease, increasing foetal haemoglobin reduces the pathological impact of the mutated K_m.
- Small molecules that stabilise protein folding act as tensional chaperones, lowering the effective K_m of the mutant protein.

Falsification: If a genetic disorder with a known single-nucleotide variant shows no tensional compensation effect from any agent that alters the epigenetic or proteostatic environment, the tensional model of that disorder is falsified.

7 Chronic Pain as Trapped Tensional Residual

Affected mesh: liquid mesh (inflammatory mediators, nerve growth factors) and solid mesh (neural plasticity, glial scarring).

Chronic pain persists after tissue healing because the RMCE cycle in the nervous system remains stuck in a comparison state: the brain continues to interpret signals as danger even though the peripheral solid mesh has repaired. The tensional flux $VT = K - T$ along pain pathways is high, and the system cannot reorganise into Phase 5 (extinction of pain memory). The IDT* of the pain network is elevated.

Tensional therapeutic strategy:

- Graded motor imagery and mirror therapy are Phase 5 retraining: they force the gaseous mesh (sensory expectations) to compare new, safe signals with the stored pain memory, gradually reducing K_m of the pain circuit.
- Neuromodulation (TENS, spinal cord stimulation) applies an external VT that competes with the pathological VT, allowing $\sum x_m$ to decrease.

Falsification: If chronic pain (e.g., phantom limb pain) shows no reduction in brain network IDT* (computed from fMRI functional connectivity) after a tensional rehabilitation protocol, the model is falsified.

8 Organ Failure as Loss of Tensional Hierarchy

Affected mesh: solid mesh of the organ (fibrosis, loss of differentiated cells) and liquid mesh (vascular perfusion, lymph drainage).

Organs are polis embedded in the larger body polis. They maintain a tensional hierarchy: specialised cells have specific K_m values that allow them to perform their function. In organ failure, the hierarchy collapses: fibrotic tissue (high K_m, low function) replaces normal tissue, and the liquid mesh becomes inadequate. The organ IDT* rises, and the body can no longer close ε locally.

Tensional therapeutic strategy:

- Regenerative medicine reprogrammes cells back to a seed state (low K_m) and guides re-differentiation along the correct tensional gradient – a Phase 5 reorganisation followed by Phase 2 accumulation.
- Decellularised matrices provide a solid mesh template with the correct K_m distribution for cell attachment.

Falsification: If a regenerating organ (e.g., liver after partial hepatectomy) does not show a transient decrease in tissue IDT* before restoring function, the tensional hierarchy model is falsified.

9 Common Protocol for Tensional Diagnosis and Intervention

For any medical condition, the following protocol applies directly from the POLIS V12 mathematical framework:

1. Define the relevant polis (cell, tissue, organ, immune network, whole person).

2. Collect clinically measurable variables (cytokine levels, protein aggregation indices, epigenetic marks, pain scores, organ function tests).
3. Set $T = K_{\min}$ from the dataset.
4. Normalise each variable: $K_m = (v - T)/(v_{\max} - T)$.
5. Compute $x_m = K_m(2 + K_m)$ and $\varepsilon = \sum x_m$.
6. Compute $\mathrm{IDT}^* = \varepsilon/(1 + \varepsilon)$.
7. Determine the tensional phase (Phase 4 is the norm; deviations indicate disease direction).
8. Design an intervention aimed at reducing IDT^*. Interventions may target any mesh.
9. Monitor IDT^* over time. If IDT^* has not decreased after a pre-specified period, the intervention is falsified.

10 Conclusion

POLIS V12 provides a unified tensional ontology for understanding and potentially treating major medical problems. Cancer, autoimmunity, neurodegeneration, aging, genetic disorders, chronic pain and organ failure are reinterpreted as deviations from the closure condition $\varepsilon = \sum K_m(2 + K_m) = 0$. The proposed tensional therapies derive directly from the axioms of POLIS V12 and are falsifiable by clinical testing. The framework does not replace molecular medicine but complements it with a causal structure that classical reductionism lacks.

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