Vasocomputation as precision weighting

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This post offers an interpretation of Michael Edward Johnson's vasocomputation through the lens of predictive coding. In particular, I'll suggest that *precision weighting*—dynamically adjusting the gain of cortical units—could be implemented by the brain's vasculature.

Information processing in the brain is conventionally attributed to neurons. On this view, the brain's vascular system exists to supply metabolic support. These vessels are encased by vascular smooth muscle cells (VSMCs), which contract or dilate in response to local neural activity. This process, known as functional hyperemia, increases blood flow to active regions and underlies signals detected in fMRI (BOLD) imaging.

Challenging this perspective, Michael Edward Johnson proposes that the vascular system may play an *active* role in computation. His framework, termed **vasocomputation**, includes three interrelated hypotheses:

- **Compressive vasomotion hypothesis (CVH):** The natural rhythmic contractions of VSMCs, called vasomotion, act to compress the dynamic range of nearby neural activity. This process reduces uncertainty by selectively "compressing" the state space, thereby guiding the system toward more distinct, interpretable patterns.

- **Vascular clamp hypothesis (VCH):** Short bursts of VSMC contraction can temporarily "clamp" local neural dynamics, stabilizing activity and allowing transient patterns to persist without requiring synaptic changes. This mechanism may support momentary perceptual stability or the maintenance of sustained predictions.

- **Latched hyperprior hypothesis (LHH):** During prolonged contraction, VSMCs can enter a low-energy latch-bridge mode, sustaining tension for extended periods without additional energy expenditure. This persistent vascular tone "locks in" certain neural states, reducing neural flexibility and responsiveness to new inputs—a process that could underlie intermediate-term memory, habitual patterns, or the persistence of trauma-related neural configurations.

In this post, I outline a computational model in which VSMCs embody *precision weighting* or sensory *gain* in the predictive coding framework. Precision refers to the brain's estimate of how reliable a given prediction is—formally, it is the inverse of the prediction error variance and determines how strongly prediction errors influence inference. In particular, I propose that vessel dilation corresponds to **decreased precision**, while constriction corresponds to **increased precision**.

In predictive coding, precision evolves at a slower time scale than neural activity, it is a function of cortical error units and in turn modulates their influence, forming a bi-directional feedback loop. This same structure could be mirrored in the neurovascular system: fast, local neural activity (errors) drive slower, cumulative changes in vascular tone (precision), which then modulate the gain on those same neural pathways. Because precision gates the effect of prediction errors, vascular tone can selectively amplify or suppress signals—effectively "grasping" or "freezing" particular states of neural activity.

1 Predictive coding

Predictive coding is an influential theory of cortical function which proposes that the brain maintains and updates a generative model of its sensory inputs. This model consists of latent variables $\{\mathbf{v_i}\}$—e.g., neural population activities—connected by directed edges $\mathcal{P} = \{(i \to j)\}$ that specify predictive relationships:

$$p(\{\mathbf{v_i}\}) = \prod_{(i \to j) \in \mathcal{P}} p\Big(\mathbf{v_j} \mid \mathbf{v_i}\Big)$$

Each edge $(i \to j)$ defines a prediction function $\mathbf{f_{ij}}\Big(\mathbf{v_i}; \mathbf{W_{ij}}, \mathbf{b_{ij}}\Big)$, parameterised by weights $\mathbf{W_{ij}}$ and biases $\mathbf{b_{ij}}$, and a precision term $\mathbf{\Pi_{ij}}$, which is the inverse of the variance $\mathbf{\Sigma_{ij}}$ and encodes confidence in the mapping. For the perspective of $\mathbf{v_i}$, this channel represents data, and from the perspective of $\mathbf{v_j}$, it represents prior information.

Notably, $\mathbf{\Pi_{ij}}$ may be represented as a matrix capturing the covariances between feature dimensions, a vector (per output dimension), or a scalar (global precision). In this way, the system can learn the covariance structure between specific features. These definitions establish the conditional distribution:

$$p\Big(\mathbf{v_j} \mid \mathbf{v_i}\Big) = \mathcal{N}\Big(\mathbf{v_j} \mid \mathbf{f_{ij}}(\mathbf{v_i}), \mathbf{\Pi_{ij}}^{-1}\Big)$$

Free energy. The system infers latent causes, parameters and precisions minimising the variational free energy $\mathcal{F}$—a tractable bound on model evidence. Under Gaussian assumptions, this becomes a sum over precision-weighted prediction errors:

$$\boldsymbol{\varepsilon_{ij}} = \mathbf{v_j} - \mathbf{f_{ij}}(\mathbf{v_i})$$

$$\mathcal{F} = \sum_{(i \to j) \in \mathcal{P}} \frac{1}{2} \boldsymbol{\varepsilon_{ij}}^\top \mathbf{\Pi_{ij}} \boldsymbol{\varepsilon_{ij}}$$

Therefore, the geometry of the energy landscape is governed by the precision values. High precision increases the curvature, while low precision flattens the landscape.

Inference and Learning To minimise $\mathcal{F}$, the system updates, variables $\mathbf{v_i}$, which update rapidly, precisions $\mathbf{\Pi_{ij}}$, which adapt more slowly (capturing, in part, the covariance of error dimensions over time), and weights $\mathbf{W_{ij}}$, which learn over longer timescales. Let $\partial^{\mathbf{v}}_{\mathbf{ij}} = \frac{\partial \mathbf{f_{ij}}}{\partial \mathbf{v_i}}$ and $\partial^{\mathbf{W}}_{\mathbf{ij}} = \frac{\partial \mathbf{f_{ij}}}{\partial \mathbf{W_{ij}}}$ denote the Jacobians of the prediction function. The update rule for each variable $\mathbf{v_i}$ is a function of precisions and error units:

$$\Delta \mathbf{v_i} = \eta \left(\underbrace{\sum_{j \in \mathrm{ch}(i)} (\partial^{\mathbf{v}}_{\mathbf{ij}})^\top \mathbf{\Pi_{ij}}\, \boldsymbol{\varepsilon_{ij}}}_{\text{outgoing predictions}} - \underbrace{\sum_{k \in \mathrm{pa}(i)} (\partial^{\mathbf{v}}_{\mathbf{ki}})^\top \mathbf{\Pi_{ki}}\, \boldsymbol{\varepsilon_{ki}}}_{\text{incoming predictions}} \right)$$

Precision updates are a function occur on a slower timescale and are a function of error units:

$$\Delta \mathbf{\Pi_{ij}}^{-1} = \eta_{\Pi} \left(\boldsymbol{\varepsilon_{ij}} \boldsymbol{\varepsilon_{ij}}^\top - \mathbf{\Pi_{ij}} \right)$$

This rule increases precision when errors are consistently small and decreases it when errors are large or unstable—effectively tracking the reliability (and covariance structure) of each predictive mapping.
Weight updates are also a function of error units.

$$\Delta \mathbf{W_{ij}} = \eta_W\, (\partial^{\mathbf{W}}_{\mathbf{ij}})^\top \mathbf{\Pi_{ij}}\, \boldsymbol{\varepsilon_{ij}}$$

Across all levels, precision acts as a gain on prediction errors. High precision increases the impact of errors on inference and learning; low precision dampens their effect. In predictive coding, the dynamic routing of information throughout the brain is governed by precision weighting: high-precision connections function as preferential routes for error signals, effectively biasing which circuits are active. For instance, if a region exhibits low prediction error, this increases $\mathbf{\Pi}$, "routing" the neural signal preferentially along that pathway. Conversely, if errors are large or persistent, this reduces $\mathbf{\Pi}$, thereby down-weighting those signals.

2 Vasocomputation as precision estimation

In this section, I propose that the vascular system implements a form of precision estimation within the predictive coding framework. Specifically, vascular tone serves as a biophysical correlate of inverse precision, modulating uncertainty on predictive mappings.

We denote vascular tone by the variable $\boldsymbol{\tau}$, representing the contractile state of a vessel: higher $\boldsymbol{\tau}$ indicates vasoconstriction (reduced blood flow), while lower $\boldsymbol{\tau}$ indicates vasodilation (increased blood flow).

2.1 Activity modules precision (vascular tone)

Prediction errors $\boldsymbol{\varepsilon}$—reflected in cortical firing—drive changes in vascular tone via neurovascular coupling (functional hyperaemia):

$$\Delta\boldsymbol{\tau}_{ij} \propto -\int \boldsymbol{\varepsilon}_{ij}(t)^2\,dt$$

High sustained errors cause vasodilation (lower $\boldsymbol{\tau}$), while low errors promote vasoconstriction (higher $\boldsymbol{\tau}$). Recall that precision updates follow:

$$\Delta\boldsymbol{\Pi}_{ij}^{-1} = \eta_{\Pi}\left(\boldsymbol{\varepsilon}_{ij}\boldsymbol{\varepsilon_{ij}}^{\top} - \boldsymbol{\Pi}_{ij}\right)$$

This motivates associating $\boldsymbol{\tau}_{ij}$ with precision $\boldsymbol{\Pi}_{ij}$: both are are low-pass filters on error activity over time.

2.2 Neurons and synapses are modulated by precision (vascular tone)

A general model of neural updates assumes that activity depends on two factors: (a) prediction error, and (b) local blood flow. Neural states can only update if there is both information (error) and sufficient metabolic support—delivered via cerebral blood flow—to sustain excitability and plasticity.

A generic biophysical update for neural activity $\mathbf{v_i}$ is:

$$\Delta\mathbf{v_i} = \eta' \sum_j g(\boldsymbol{\tau}_{ij})\,\boldsymbol{\varepsilon}_{ij}$$

where $\boldsymbol{\varepsilon}_{ij}$ is the prediction error on edge $i \to j$ and $g(\boldsymbol{\tau}_{ij})$ is some function that maps vascular tone to activity modulation. This mirrors the predictive coding update:

$$\Delta\mathbf{v_i} \approx \eta\left(\sum_{j\in\mathrm{ch}(i)} \boldsymbol{\Pi_{ij}}\,\boldsymbol{\varepsilon}_{ij} - \sum_{k\in\mathrm{pa}(i)} \boldsymbol{\Pi_{ki}}\,\boldsymbol{\varepsilon}_{ki}\right)$$

In both forms, the impact of an error depends not only on its magnitude, but on how much confidence the system assigns to it—via either $\boldsymbol{\Pi}$ or $\boldsymbol{\tau}$. Synaptic weights are likewise modulated by precision:

$$\Delta\mathbf{W_{ij}} = \eta_W\,(\partial_{\mathbf{ij}}^{\mathbf{W}})^{\top}\boldsymbol{\Pi_{ij}}\,\boldsymbol{\varepsilon}_{ij}$$

When a connection has high precision, it strongly constrains inference. Other sources must generate large enough mismatches to override its predictions. Thus, in high precision regimes, errors are suppressed—but when they occur, they produce significant synaptic plasticity.

2.3 Vasoconstriction: small, stable errors increase precision

When prediction errors remain low over time, vessels constrict, leading to an increase in vascular tone $\boldsymbol{\tau}$ and a corresponding rise in precision $\boldsymbol{\Pi_{ij}}$. This amplifies the gain on local error signals, effectively "gripping" or stabilizing the associated prediction. The system becomes more confident in that mapping and suppresses competing signals unless their errors become sufficiently large.

2.4 Vasodilation: large, persistent errors reduce precision

Conversely, sustained mismatches between predictions and sensory inputs trigger vasodilation. Vascular tone $\boldsymbol{\tau}$ decreases, leading to a reduction in precision $\boldsymbol{\Pi_{ij}}$. This down-weights the influence of the corresponding prediction error, reducing its impact on belief updates. In this low-precision regime, the system becomes more responsive to alternative inputs, allowing a broader range of signals to shape inference. This facilitates exploration, representational flexibility, and the potential reconfiguration of model structure.

Vasodilation also increases energetic flow—enhancing the delivery of oxygen, glucose, and neuromodulators to local tissue. This metabolic up regulation supports heightened plasticity. Sustained dilation thus creates conditions favorable for synaptogenesis and structural learning, enabling the emergence of new mappings when prior predictions fail to account for the data.

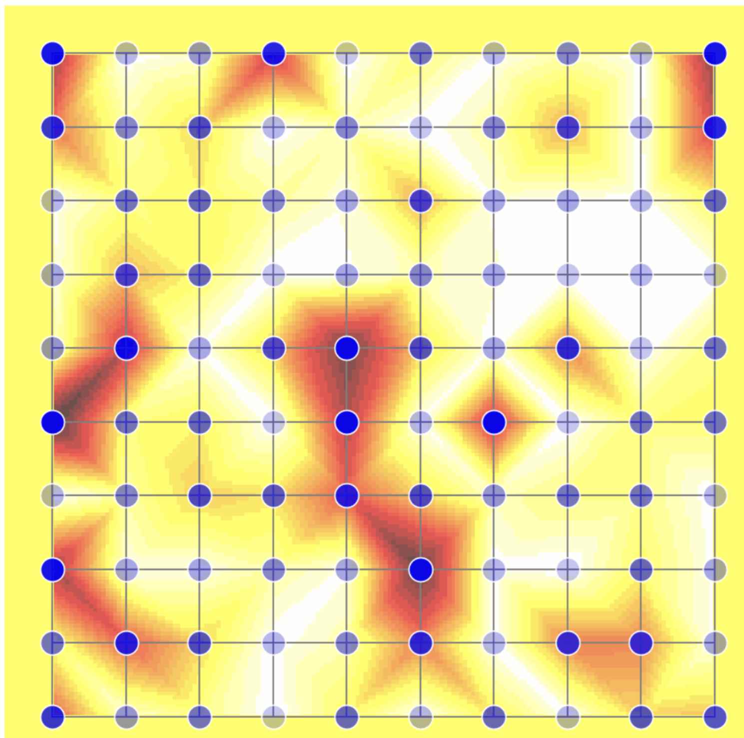

Figure 1: *Islands of uncertainty encoded by blood flow. Blue nodes correspond to error units.*

To illustrate how vascular tone shapes inference, consider a belief $\mathbf{x}$ influenced by three variables: $\mathbf{z}$, $\mathbf{y}$, and $\mathbf{w}$. Each sends a prediction to $\mathbf{x}$ via a corresponding mapping—$\mathbf{f_{zx}}(z)$, $\mathbf{f_{yx}}(y)$, and $\mathbf{f_{wx}}(w)$—with associated precisions $\boldsymbol{\pi}_{zx}$, $\boldsymbol{\pi}_{yx}$, and $\boldsymbol{\pi}_{wx}$. The predictive coding update for $\mathbf{x}$ is:

$$x \leftarrow x + \eta \left(\boldsymbol{\pi}_{zx}\, \varepsilon_{zx} + \boldsymbol{\pi}_{yx}\, \varepsilon_{yx} + \boldsymbol{\pi}_{wx}\, \varepsilon_{wx} \right)$$

Each term reflects how much the corresponding prediction error ε influences the update of $\mathbf{x}$, scaled by its precision. If the $\mathbf{z} \to \mathbf{x}$ vessel constricts (i.e., $\boldsymbol{\tau}_{zx}$ increases), this raises the precision $\boldsymbol{\pi}_{zx}$. As a result, $\mathbf{z}$ exerts a stronger influence on $\mathbf{x}$, biasing inference toward $\mathbf{z}$'s prediction—even if $\mathbf{y}$ or $\mathbf{w}$ suggest otherwise. If the $\mathbf{y} \to \mathbf{x}$ or $\mathbf{w} \to \mathbf{x}$ vessels dilate (i.e., $\boldsymbol{\tau}_{yx}$ or $\boldsymbol{\tau}_{wx}$ decrease), their precisions $\boldsymbol{\pi}_{yx}$ and $\boldsymbol{\pi}_{wx}$ drop. These signals are now treated as less reliable, and their contribution to $\mathbf{x}$'s update diminishes. In this way, vascular tone dynamically gates the influence of different inputs based on their recent reliability. Constriction sharpens and stabilises prediction channels; dilation loosens their grip, allowing the system to discount noisy or uncertain inputs.

Temporal averaging: In predictive coding, estimating precision requires averaging over time to assess the stability of prediction errors. Vascular tone, which updates slower than spiking activity, is well-suited for this role. By integrating over error fluctuations, it provides a smoothed, time-averaged estimate of precision—tracking the long-term reliability of predictive relationships and their covariances.
Spatial coordination: Vascular modulation operates at a broader spatial scale—such as cortical micro-columns—enabling precision to be coordinated across local neural populations. This spatial diffusion may bind neurons into functional assemblies that share statistical structure, extending beyond what is captured by direct synaptic connectivity alone.
Metabolic vs computational gain: Vasodilation increases metabolic gain (e.g., oxygen and glucose delivery, excitability), while reducing computational gain (precision). This dissociation reflects a functional division of labour: when large or uncertain prediction errors arise, the system lowers its confidence in current beliefs while simultaneously allocating resources to support learning, exploration, or flexible updating of representations.

In summary, neural activity (error signals) and vascular responses (precision estimation) form a bi-directional, time-scale–dependent loop: fast spiking modulates slower vascular adjustments, which in turn regulate the gain on subsequent neural processing.

2.4.1 Compressive Vasomotion Hypothesis (CVH)

Claim: Vasomotion consists of rhythmic oscillations in arteriole diameter (approximately 0.1 Hz) that produce periodic changes in blood flow. Vasocomputation proposes that these oscillations function as a compression mechanism: by cyclically modulating precision, they collapse uncertain or diffuse neural representations into more stable, distinct configurations.

Interpretation: During the dilation phase of each vasomotor cycle, vascular tone $\boldsymbol{\tau}$ decreases, reducing precision $\mathbf{\Pi_{ij}}$ and flattening the free energy landscape. This allows multiple competing predictions to coexist without any particular channel dominating. As the cycle shifts toward constriction (i.e., increased $\boldsymbol{\tau}$), precision rises and selectively reinforces the pathway with the lowest cumulative error. Over time, this rhythmic fluctuation sharpens representational certainty by pruning unstable states and stabilising the most reliable inferences. This process enables the system to resolve ambiguity and consolidate coherent interpretations without relying on immediate synaptic change.

$$\begin{aligned} \text{During dilation:} \quad & \boldsymbol{\tau} \downarrow \Rightarrow \mathbf{\Pi_{ij}} \downarrow \quad \Rightarrow \quad \Delta \mathbf{v_i} \propto \text{lower gain on } \boldsymbol{\varepsilon_{ij}} \\ \text{During constriction:} \quad & \boldsymbol{\tau} \uparrow \Rightarrow \mathbf{\Pi_{ij}} \uparrow \quad \Rightarrow \quad \Delta \mathbf{v_i} \propto \text{higher gain on } \boldsymbol{\varepsilon_{ij}} \end{aligned}$$

2.4.2 Vascular Clamp Hypothesis (VCH)

Claim: Short-term vasoconstriction can act as a "clamp" on local neural dynamics. By maintaining elevated vascular tone $\boldsymbol{\tau}$ for seconds to minutes, the system temporarily increases precision $\mathbf{\Pi_{ij}}$, stabilizing a particular predictive relationship in a high-confidence state.

Interpretation: Sustained high $\boldsymbol{\tau}$ locks the system into a high-precision regime, amplifying small errors and reinforcing existing relationship. This effectively "holds open" a particular inference pathway, allowing it to persist across time even as sensory evidence fluctuates. In predictive coding terms, the clamp strengthens the influence of that edge on downstream variables:

$$\Delta \mathbf{v_i} \propto \text{High } \mathbf{\Pi_{ij}} \times \boldsymbol{\varepsilon_{ij}} \ldots$$

Rather than clamping neural activity per se, the vasculature secures a relationship within the generative model, ensuring that it continues to dominate inference unless definitively contradicted. This allows the brain to uphold interpretations or contextual priors without immediate plasticity.

2.4.3 Latched Hyperprior Hypothesis (LHH)

Claim: In a latch state, vascular smooth muscle maintains prolonged contraction with minimal energetic cost via the latch-bridge mechanism. This corresponds to a long-term elevation in vascular tone $\boldsymbol{\tau}$, which enforces persistently high precision $\mathbf{\Pi_{ij}}$ on a given predictive relationship.

Interpretation: Prolonged high $\boldsymbol{\tau}$ encodes structural confidence in a particular mapping. Even if errors occur, they are treated as noise unless sustained and significant. The resulting entrenched precision biases inference toward explanations that conform to the latched relationship:

$$\Delta \mathbf{v_i} \propto \text{Persistent high } \mathbf{\Pi_{ij}} \times \boldsymbol{\varepsilon_{ij}}...$$

This effectively locks in portions of the generative model, creating hyperpriors that shape future inference. Biologically, vascular latching enables the system to impose rigid constraints—such as ingrained beliefs, habits, or trauma-associated threat models—without continuous metabolic expenditure. The latch-bridge mechanism allows tone to be maintained efficiently, encoding long-term priors in vascular structure and providing a durable substrate for shaping cognition and behavior over extended timescales.

3 Related ideas

Hemo-neural hypothesis Compared to muscle tissue, brain regions routinely receive an *excess* of blood flow—implying a computational function beyond mere metabolic support. Moreover, there is evidence of anticipatory hemodynamic signals: for instance, studies have shown that blood flow in the visual cortex increases prior to stimulus onset, suggesting a predictive tuning of vascular responses. Additional research demonstrates rhythmic vessel oscillations, known as vasomotion, that synchronize across broad cortical regions. Sasaki et al. (2024) found that rhythmic visual stimuli at 0.25 Hz induced widespread vasomotor synchronisation, extending beyond the visual cortex into the cerebellum (Plastic vasomotion entrainment — eLife). This synchronised vasomotion exhibited plasticity, with its magnitude in specific cerebellar regions correlating with performance improvements in visual learning tasks.

Embodiment and evolutionary context From an evolutionary standpoint, modulating variance via vascular smooth muscle offers an energy-efficient strategy for resource allocation. By constricting vessels around well-predicted inputs (i.e. low variance), the brain can conserve energy, while diverting blood to uncertain areas (high variance) where exploration and synaptic reconfiguration are more likely. This efficiency is further enhanced by the latch-bridge mechanism in smooth muscle cells, which allows sustained contraction with minimal energy expenditure—effectively "locking in" high-precision states without ongoing metabolic cost. Such mechanisms are ancient and widespread: simpler organisms like plants and slime molds coordinate behaviour through fluid dynamics and chemical gradients, suggesting that vascular-style computation is embedded in biological evolution. In mammals, this layered architecture persists: fast neural spikes detect and transmit immediate changes, while slower vasomotor rhythms help consolidate patterns and regulate long-term uncertainty.

Attention and global states Attention can be understood as targeted precision control—modulated locally by vascular tone. Focusing on a particular stimulus may involve vasoconstriction, which increases local precision and enhances the influence of relevant prediction errors. Conversely, vasodilation may lower precision, allowing for greater flexibility and sensitivity to subtle or ambiguous inputs. A compelling illustration is heartbeat-linked sensory gating, where arterial pressure surges during systole induce transient vasoconstriction ("micro-clamps"), momentarily reducing cortical responsiveness. This mechanism may help the brain suppress irrelevant inputs during predictable internal events. Indeed, faint sensory stimuli are less likely to be perceived during these peaks, and subjective time perception appears to expand or compress depending on the cardiac cycle. On broader timescales, slow vascular oscillations—such as ~ 0.1 Hz Mayer waves, entrained by respiration and cardiac rhythms—can synchronise across cortical regions, creating fluctuating global precision states. These rhythms may play a role in orchestrating emotional tone, attentional readiness, and inter-regional communication.

Psychiatric and neurological implications Variations in vascular precision weighting offer a framework for understanding psychiatric and neurological disorders. For instance, schizophrenia may reflect dysregulated vascular modulation—either over-dilation (excessive uncertainty) or under-constriction (inadequate filtering)—leading to hallucinations, delusions, or unstable belief updating. Autism spectrum conditions have been linked to chronic under-constriction in early sensory circuits, potentially resulting in overwhelming perceptual detail and difficulties in generalisation. Trauma-related disorders, including PTSD, may involve persistent vascular "latching," where chronic vasoconstriction reinforces high-precision threat predictions and resists updating even in safe environments. Similarly, chronic anxiety and stress-related conditions may reflect prolonged global vasoconstriction, maintaining a hyper-precise threat-monitoring state that fuels both psychological and somatic tension. Importantly, several therapeutic strategies—including meditation, breath work, and psychedelics—may act by modulating vascular tone and, consequently, recalibrating precision. Psychedelics, in particular, promote vasodilation, relaxing entrenched vascular latches and enhancing cognitive flexibility. Changes in vascular tone across sleep stages—such as reduced precision during REM sleep—may also support synaptic remodeling and memory consolidation, underscoring the broader computational role of vascular dynamics.

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