

Dynamic Symmetry and the Heart: A Systems-Biology Metric for Adaptive Balance in Physiological Networks

Abstract

Dynamic symmetry theory (Edge theory) proposes that complex adaptive systems exhibit their richest and most robust behaviour in regimes that balance structural order with stochastic variability. The Dynamic Symmetry Index (DSI) offers a quantitative formulation of this hypothesis by combining metrics of order and disorder into a normalised measure of a system's proximity to such an adaptive regime. In parallel, Denis Noble's work on systems physiology and evolution has advanced a view of organisms in which stochasticity is actively harnessed by multi-level structures and feedback processes, rather than passively endured. This paper brings these perspectives together in the context of cardiac and physiological networks. After introducing dynamic symmetry theory and the DSI, the discussion summarises key elements of Noble's account of evolution and the harnessing of stochasticity, with an emphasis on heart physiology and systems biology. It then develops a detailed analysis of how dynamic symmetry and Noble's ideas converge when applied to the heart, arguing that the heart and associated regulatory systems can be fruitfully understood as maintaining a dynamic symmetry between order and disorder across multiple scales. Potential empirical and modelling strategies are outlined, including the application of DSI-like metrics to heart-rate variability, conduction network dynamics and coupled physiological signals. The aim is to show that the conjunction of dynamic symmetry and the harnessing of stochasticity yields a coherent systems-biology metric for adaptive balance in physiological networks, with implications for both basic science and clinical practice.

Dynamic symmetry theory and the Dynamic Symmetry Index

Dynamic symmetry theory has its roots in an old intuition: adaptive systems do not flourish in states of maximal regularity or maximal randomness, but in an intermediate region where structured patterns and exploratory variation coexist. Traditional complexity science has often gestured towards this region using phrases such as "the edge of chaos", informed by work on cellular automata, self-organised criticality and phase transitions in dynamical systems. However, such descriptions have not always provided a versatile, empirically tractable way to quantify where a given system sits along the spectrum from rigid order to unstructured noise. Dynamic symmetry theory seeks to supply this missing piece by specifying, in general terms, what it means for a system to exhibit a balanced interplay between order and disorder, and by proposing mathematical tools to measure and track that balance.

In its contemporary formulation, the theory treats “order” and “disorder” not as vague metaphors but as families of measurable properties. Order refers to features such as regularity, predictability, coherence and low effective dimensionality in the behaviour or structure of a system. Disorder refers to randomness, heterogeneity, uncertainty and high diversity of states or trajectories. The dynamic symmetry hypothesis asserts that, for many complex adaptive systems, there exists a regime in which these two tendencies are roughly matched, so that structure is sufficient to constrain and coordinate behaviour, while variability is sufficient to sustain exploration, robustness and responsiveness. Too much order yields brittle, over-constrained systems that fail catastrophically under perturbation. Too much disorder yields systems that lack the coherence required for reliable function.

The Dynamic Symmetry Index (DSI) is introduced as a way to formalise this balance. The general construction proceeds by choosing, for a given system and timescale, one or more metrics of order, denoted $O(t)$, and one or more metrics of disorder, denoted $D(t)$, each normalised to the interval $[0, 1]$. Order metrics can include synchrony measures such as the Kuramoto order parameter in networks of oscillators, graph-theoretic indices such as modularity or efficiency, and indicators of low-dimensional attractor dynamics. Disorder metrics typically draw on Shannon or Rényi entropy, diversity indices and positive Lyapunov exponents, which signal sensitive dependence on initial conditions and chaotic behaviour. Under appropriate normalisation and scaling, the DSI at time t is defined by:

$$\text{DSI}(t) = 1 - |\alpha O(t) - \beta D(t)|$$

where α and β are non-negative parameters that weight the relative contributions of order and disorder. When $\alpha O(t)$ and $\beta D(t)$ are closely matched, the absolute difference is small and DSI is close to 1, suggesting a dynamically symmetric regime. When one term substantially outweighs the other, DSI approaches 0, signalling a drift towards either over-rigidity or incoherent fluctuation.

A central virtue of this formulation is its flexibility. The choice of order and disorder metrics, and the calibration of α and β , are explicitly domain-dependent, allowing the DSI framework to be adapted to systems as diverse as neural networks, ecological communities, organisations and financial markets, subject to suitable empirical calibration. At the same time, the mathematical form ensures comparability across contexts: in each case, DSI serves as an index of balance between structured regularity and stochastic variation. The theory is not content to rest at the level of abstract metaphor; it attempts to anchor the idea of an “edge” in measurable features of system behaviour and structure, while respecting the specificity of different domains.

The DSI paper sets out procedures for metric selection, normalisation, parameter fitting and validation. Order and disorder metrics are chosen so as to be complementary and, where possible, orthogonal, with normalisation procedures such as z-scoring or min-max scaling ensuring that they are commensurable. Calibration of α and β can be performed using grid search or Bayesian methods to maximise the correlation between DSI and independent measures of system performance, such as resilience, innovation rate or cognitive flexibility. Temporal and spatial multiscalar implementations are encouraged: DSI can be computed on local subsystems, aggregated networks and across different time windows, allowing the detection of transient episodes of dynamic symmetry and the study of cross-scale interactions.

Denis Noble's harnessing of stochasticity

In "Evolution viewed from physics, physiology and medicine", Noble argues that evolutionary theory and biological explanation more generally should be re-examined from the perspective of physiology and systems biology. Rather than treating genes as the sole or primary agents of evolutionary change, he emphasises the role of multi-level networks, feedback and physiological constraints in shaping how variation arises and is channelled. Stochastic processes, on this view, are not mere background noise but crucial resources that organisms harness to generate functional diversity and adaptive responses.

Noble's broader corpus explores several themes that are directly relevant. First, he highlights the importance of multi-level organisation, from molecules and cells through tissues and organs to whole organisms and populations. Causation in such systems is not unidirectional from lower to higher levels but circular: higher-level structures shape the context in which lower-level stochasticity plays out, while lower-level events feed back to alter higher-level organisation. This circular causality provides a framework within which stochasticity can be understood as harnessed rather than purely random.

Second, Noble points to specific biological processes where stochasticity is clearly channelled by structure. In the immune system, for example, somatic hypermutation and recombination introduce large amounts of randomness into antibody gene regions, but this randomness is constrained to particular loci and integrated into a network of clonal selection and feedback that yields effective immune repertoires. In neurophysiology, variability in neuronal firing and synaptic transmission contributes to flexible behaviour and learning, yet such variability is shaped by network architecture and neuromodulatory systems. In cardiac physiology, beat-to-beat variability in heart rate and conduction can be associated with health when it reflects a responsive, adaptable control system, and with pathology when it indicates breakdown of coordination or over-rigid regulation.

Third, Noble's work in systems biology, particularly on cardiac modelling, shows how mathematical models can integrate stochastic elements within structured, multi-scale frameworks. Early models of the cardiac action potential and heart function treated variability as a nuisance, but later developments have incorporated stochastic ion-channel behaviour, noise in signalling pathways and variability in cellular properties, all embedded within highly organised anatomical and physiological structures. In such models, the balance between structural constraints and stochastic variation is crucial: too little variability can render the system fragile in the face of perturbations, while too much can lead to arrhythmias and loss of coherent function.

The language of harnessing stochasticity captures this intuition. Stochasticity is present in many processes, from molecular binding events to neural firing patterns, but living systems have evolved ways to confine, shape and exploit this variability so that it contributes to robustness, adaptability and innovation. Evolution itself is not merely a story of random mutations filtered by selection; it is also a story of evolving architectures that modulate how randomness is generated, where it is permitted to act and how its products are evaluated. Noble's physiologically grounded perspective shifts the explanatory focus towards the organised, adaptive use of stochastic processes within multi-level systems.

Dynamic symmetry, stochasticity and physiological networks

The convergence between dynamic symmetry and Noble's harnessing of stochasticity becomes clear once one recognises that both frameworks are concerned with the same structural–dynamical issue: how systems maintain enough stability to function and enough variability to adapt. Dynamic symmetry provides a general way to talk about the balance between order and disorder; Noble provides detailed examples of how physiological systems achieve such a balance by harnessing stochasticity within structured networks.

In dynamic symmetry terms, a system that successfully harnesses stochasticity will display high DSI values. The organised architecture of the system (its feedback loops, network topology, regulatory mechanisms) contributes to $O(t)$, the order component, while the controlled randomness in underlying processes contributes to $D(t)$, the disorder component. When these are appropriately matched, $\alpha O(t) \approx \beta D(t)$, indicating that the system's constraints and variability are in a productive relationship. If constraints dominate, stochasticity may be suppressed, leading to rigidity and a reduced capacity to respond to novel circumstances. If stochasticity overwhelms constraints, the system may lose coherence, resulting in pathological behaviour.

Noble's examples lend themselves to re-description in this language. The immune system's hypermutation processes can be seen as a high $D(t)$ regime confined within strong structural constraints on which loci may mutate and how variants are selected, preserving a high $O(t)$. Effective immune function would correspond to high DSI: a well-balanced exploitation of stochastic variation within a coherent network. Failures might correspond to low DSI: excessive rigidity that fails to generate sufficient diversity, or excessive disorder that leads to autoimmunity or ineffective responses.

In cardiac physiology, the interplay of structural order and stochastic variability is equally evident. The heart relies on the orderly propagation of electrical signals through specialised conduction pathways and the coordinated contraction of muscle fibres. This structural organisation underpins a high degree of order, reflected in regular rhythms and low-dimensional dynamical patterns under resting conditions. Yet the heart is also subject to stochastic influences: random fluctuations in ion-channel behaviour, variable autonomic input, and external perturbations. Healthy heart-rate variability appears to reflect a form of harnessed stochasticity: variability that is structured by regulatory networks and feedback loops in such a way that the heart remains responsive and robust.

In dynamic symmetry terms, a healthy heart would maintain DSI in a mid-range where order and variability are balanced. Very low variability (near-perfect periodicity) is often associated with pathological conditions or reduced adaptability, while excessive irregularity is characteristic of arrhythmias. The idea that there is an optimal band of variability, neither too low nor too high, aligns closely with the notion of an adaptive edge. Dynamic symmetry and DSI provide a way to formalise this intuition, while Noble's physiological work specifies the mechanisms through which such balance is realised and disrupted.

Dynamic Symmetry and the Heart: A Systems-Biology Metric for Adaptive Balance in Physiological Networks

The heart is an ideal testbed for exploring dynamic symmetry and the harnessing of stochasticity, because it sits at the intersection of multiple regulatory systems and exhibits both robust regularity and meaningful variability. At one level, the sinoatrial node and conduction system impose a structured rhythm on cardiac activity. At another level, autonomic inputs, endocrine signals and mechanical feedback introduce fluctuations and modulations that allow the heart to respond to changing demands. The resulting behaviour reflects an ongoing negotiation between order and disorder across molecular, cellular, tissue and systemic levels.

A systems-biology analysis of the heart in dynamic symmetry terms would begin by identifying appropriate order and disorder metrics for cardiac and associated physiological signals. On the structural side, order metrics could include measures of phase synchrony in electrocardiographic signals, network coherence in excitation-contraction coupling, and regularity in the timing of cardiac cycles. On the variability side, disorder metrics could draw on multiscale entropy of heart-rate time-series, variability in inter-beat intervals and measures of stochasticity in ion-channel activity or autonomic firing patterns. These metrics would be normalised and combined to yield an index of dynamic symmetry for the cardiac system at a given timescale.

Such an index could be applied to different conditions and interventions. For example, DSI could be computed for healthy individuals at rest, during graded exercise and in recovery, capturing how the balance between order and variability shifts as the heart adapts to changing physiological demands. One might hypothesise that during moderate exercise, DSI increases relative to resting levels, reflecting an enhanced coordination between structural constraints and variability as the system explores a wider range of states while maintaining coherence. During extreme stress or in pathological states, DSI might either collapse towards low values (if variability becomes unstructured and arrhythmic) or decline due to excessive rigidity (if regulatory systems overly suppress variability).

From a systems-biology perspective, dynamic symmetry invites a multi-scale extension. The heart does not operate in isolation, but as part of a broader physiological network that includes respiratory, endocrine, neural and metabolic subsystems. Order and variability in cardiac behaviour are entangled with order and variability in these coupled systems. For instance, respiratory sinus arrhythmia reflects a coupling between respiratory and cardiac rhythms, and baroreflex mechanisms link blood pressure fluctuations to heart-rate modulation. Applying DSI-like metrics to joint time-series of heart-rate, respiration and blood pressure could provide an index of dynamic symmetry for the cardio-respiratory network as a whole, highlighting regimes where these systems cooperate to sustain adaptability.

Noble's arguments about circular causality and multi-level organisation suggest that dynamic symmetry should be examined not only at the level of observable signals but also at the level of network architecture and feedback loops. For example, the structural topology of the cardiac conduction system, the distribution of autonomic innervation and the organisation of feedback pathways between the heart and central nervous system all contribute to the order component $O(t)$. Stochastic processes at the level of ion channels, neurotransmitter release and receptor dynamics contribute to

the disorder component $D(t)$. Modelling these processes in a unified framework could allow one to compute DSI not only from observable time-series but also from simulated or inferred network dynamics, offering deeper insight into the mechanisms by which the heart harnesses stochasticity.

Empirically, implementing such an approach would require collaborative efforts. Physiological datasets containing high-resolution electrocardiographic recordings, respiratory signals and potentially neural or autonomic proxies would need to be analysed using dynamic symmetry metrics. Theoretical work would be needed to determine which combinations of order and disorder measures are most informative for different conditions, and how to calibrate the weighting parameters α and β so that DSI correlates with clinically relevant outcomes such as resilience to stress, risk of arrhythmia or recovery trajectories after cardiac events. Noble's work points to the importance of interpreting such indices within a physiological and evolutionary framework, rather than treating them as mere statistical curiosities.

Conceptually, the convergence between dynamic symmetry and the harnessing of stochasticity in cardiac physiology suggests several points. First, it reinforces the idea that variability in heart-rate and related signals should not be treated uncritically as "noise" or deviation from an idealised constant rhythm. Instead, variability is a sign of an actively regulated system capable of adjusting to internal and external changes, provided that it remains structured and integrated within the broader physiological network. Dynamic symmetry offers a way to distinguish healthy, harnessed variability from pathological irregularity by focusing on the relationship between variability and structural coherence.

Second, the application of DSI to cardiac systems underscores the need for models that capture both upward and downward causation. Structural organisation at higher levels, such as network architecture and regulatory loops, shapes the conditions under which stochastic events at lower levels contribute to function. In turn, aggregate patterns emerging from stochastic micro-events can alter higher-level states. This bidirectional interplay is central to Noble's account of physiology and evolution, and dynamic symmetry provides a quantitative language for discussing how such interplay can yield stable yet adaptable dynamics in the heart.

Third, a dynamic symmetry perspective on cardiac physiology invites reflection on clinical practice. If specific regimes of DSI are associated with healthy adaptation, and others with heightened risk, then monitoring DSI in clinical settings could aid risk stratification and intervention planning. For instance, shifts in DSI during rehabilitation after myocardial infarction or in chronic heart failure patients might indicate improving or deteriorating adaptive capacity, supplementing traditional indicators. Potential

therapeutic approaches might be evaluated not only in terms of their immediate effects on blood pressure or ejection fraction, but also in terms of how they affect the heart's dynamic symmetry within the broader physiological network.

Conclusion

Dynamic symmetry theory and Denis Noble's account of the harnessing of stochasticity converge on a shared insight: living systems sustain their adaptability by maintaining a structured balance between order and variability across multiple levels of organisation. The Dynamic Symmetry Index formalises this balance as a quantitative metric, adaptable to different domains through appropriate choice and calibration of order and disorder measures. Noble's work provides detailed, physiologically grounded examples of how such balance is achieved and modulated, particularly in the heart and associated regulatory systems. When brought together, these perspectives suggest that the heart can be understood as a paradigmatic dynamically symmetric system, harnessing stochasticity through organised structures and feedback loops to maintain function under changing conditions.

The proposed application of DSI-like metrics to cardiac and physiological networks offers a path towards operationalising this insight. By measuring the balance between structural coherence and stochastic variability in heart-rate dynamics, conduction patterns and coupled physiological signals, researchers and clinicians may gain a richer understanding of what distinguishes healthy from pathological regimes. Such work would not only deepen the conceptual connection between dynamic symmetry and the harnessing of stochasticity, but also exemplify how ideas originating in complex systems theory and systems biology can inform practical metrics for adaptive balance in living networks.

References and further reading

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