

Review Article

**The Intelligent Bedside Monitor: A New Path
Toward Personalized Critical Care****Dr. Philippe Rola¹, Dr. Vimal Bhardwaj²***¹Associate Professor, University of Montreal Intensive Care Unit, Santa Cabrini Hospital CEMTL, Montreal, Canada Co-Founder, Resuscare AI**²Consultant Intensivist Narayana Health City, Bangalore, India Founder - Resuscare AI****Corresponding author**

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INTRODUCTION

Modern acute care is defined by continuous monitoring. Across intensive care units, operating rooms, emergency departments and post-anaesthesia care units, clinicians are surrounded by streams of physiological data derived from electrocardiography, pulse oximetry, invasive and non-invasive blood pressure monitoring, capnography, mechanical ventilation and other increasingly sophisticated devices. Yet, despite the apparent richness of this information, much of contemporary monitoring remains conceptually simple. Complex physiological signals are reduced to intermittent numerical values, and clinicians are alerted when one of these values crosses a predefined population-based threshold. In practice, the patient is monitored continuously, but their physiology is interpreted only intermittently. This model has important limitations. A mean arterial pressure of 65 mmHg, a heart rate of 110 beats per minute or an oxygen saturation of 92% does not carry the same significance in every patient or at every stage of illness. A single threshold cannot account for baseline physiology, comorbid disease, the mechanism of instability, the phase of resuscitation or the response to previous interventions.

Moreover, clinically important deterioration may be expressed initially not by an abnormal absolute value, but by a change in waveform morphology, loss of physiological variability, an evolving relationship between several signals or a progressive departure from the patient's own trajectory. By the time a conventional alarm is generated, a period of compensated physiological deterioration may already have occurred.

Current monitoring systems also create a paradox of abundance without integration. They generate more information than clinicians can continuously process, while simultaneously producing large numbers of false or non-actionable alarms. This contributes to alarm fatigue, desensitization and delayed responses to genuinely important events [1]. Increasing the number of monitored variables or lowering alarm thresholds is therefore unlikely, by itself, to improve care. The central challenge is no longer simply acquiring more data, but transforming existing data into timely and clinically meaningful physiological information.

Recent advances in artificial intelligence, machine learning and high-fidelity waveform analysis offer a potential transition from threshold-based monitoring to longitudinal physiological interpretation. Algorithms can evaluate thousands of waveform characteristics, integrate changes across multiple signals and recognize temporal patterns that are difficult for clinicians to identify through visual inspection alone. Studies in both perioperative and critical care populations have demonstrated that evolving hypo-

tension or circulatory failure can, under some circumstances, be identified before conventional clinical recognition [2–4]. These observations establish the technical possibility of anticipatory monitoring, although they do not yet demonstrate that prediction alone improves patient outcomes.

The greater opportunity extends beyond early detection. By learning each patient's baseline, trajectory and responses to intervention, intelligent monitoring could treat the patient as their own physiological reference. Rather than simply identifying that a universal threshold has been crossed, it could determine whether the patient is changing and whether treatment is restoring the intended physiological state. This aligns with personalized haemodynamic management, in which targets and monitoring are adapted to the patient, clinical context and phase of illness [5,6].

This transformation could apply across critical care, anaesthesia, emergency medicine and other monitored environments, although its implementation would necessarily vary according to available signals, clinical context and event prevalence. The future bedside monitor may therefore become more than a display or alarm generator: it may function as a continuously updated model of the patient's physiology. Realizing that potential, however, will require moving beyond demonstrations of algorithmic accuracy toward interpretable systems, rigorous prospective validation, acceptable alert burdens, seamless clinical integration and ultimately evidence that their use improves decision-making and patient-centred outcomes.

**Beyond Thresholds: The Patient as Their Own
Physiological Reference**

Conventional monitoring is fundamentally threshold-based. Physiological variables are considered reassuring while they remain within predefined limits and concerning once they cross them. This approach is operationally simple, but it assumes that the meaning of a value is sufficiently consistent across patients and clinical contexts. In reality, haemodynamic tolerance is highly individualized. A mean arterial pressure of 60 mmHg may be adequate for one patient yet represent a major departure from baseline perfusion pressure in another. Similarly, tachycardia, a narrow pulse pressure or a reduction in oxygen saturation may be benign, compensatory or an early manifestation of deterioration depending on the underlying physiology and the trajectory preceding the change [7,8].

The limitations of fixed thresholds become especially apparent during rapidly evolving illness. Physiological deterioration frequently develops as a continuum rather than a discrete event. Compensatory mechanisms may preserve arterial pressure despite progressive reductions in stroke volume,

while vasoplegia may produce substantial circulatory dysfunction without an initially dramatic change in conventional parameters. Conversely, an isolated abnormal value may reflect pain, anaesthesia, positioning, medication, arrhythmia or measurement artifact rather than clinically important instability. A monitor that evaluates variables independently cannot readily distinguish among these possibilities.

The clinically meaningful signal may therefore reside less in the absolute value than in its temporal and physiological context. Relevant information includes the direction and rate of change, deviation from the patient's baseline, evolving waveform morphology, loss of normal variability, relationships among simultaneously measured signals and the response to an intervention. A progressive reduction in arterial pulsatility, for example, carries different implications when accompanied by increasing vasopressor requirements, delayed capillary refill or rising venous pressures than when it occurs in isolation. Physiological variables derive meaning from their interaction with the wider cardiovascular system.

Continuous monitoring creates an opportunity to establish an individualized physiological reference rather than repeatedly comparing the patient only with population-derived norms. The early monitoring period could characterize baseline values, normal short-term variability and relationships among signals. Subsequent changes could then be evaluated according to their magnitude, persistence and departure from that patient's evolving trajectory. This does not imply that conventional safety thresholds should be abandoned. Rather, population thresholds could provide broad safety boundaries, while individualized trajectories identify clinically important change before those boundaries are crossed.

The same principle applies to treatment response. Fluids, vasopressors, inotropes, anaesthetic agents, ventilatory adjustments and changes in position function as physiological perturbations. Each intervention provides information about the cardiovascular system through the direction, magnitude and durability of the resulting response. This principle, often described as functional haemodynamic monitoring, shifts the emphasis from static measurements to the physiological response produced by a defined intervention [7]. An intelligent monitoring platform could therefore move beyond passive surveillance to assess whether an intervention produced its intended effect, had no meaningful effect or generated an adverse response. Over time, the patient becomes not only their own reference but also the source of an accumulating, individualized response profile.

However, personalization cannot be achieved through trend analysis alone. Circulatory function emerges from the interaction of ventricular performance, arterial tone, microvascular perfusion, venous pressure and right ventricular–pulmonary arterial coupling. The same arterial pressure may therefore result from profoundly different haemodynamic states and require very different treatments. The next step beyond individualized trajectories is the integration of monitored signals within a holistic physiological model capable of identifying where circulatory dysfunction is occurring and how abnormalities in one component propagate through the rest of the cardiovascular system.

From Prediction to Physiological Phenotyping

Much of the current literature evaluates artificial intelligence according to its ability to predict a discrete adverse event, such as hypotension, shock, respiratory failure or cardiac arrest. Earlier recognition is undoubtedly valuable, particularly when it creates sufficient lead time for reassessment or intervention. Nevertheless, predicting that deterioration will occur addresses only the first of several clinically important questions. The clinician must still determine whether the signal is genuine, identify the physiological mechanism responsible and select an appropriate intervention. This distinction is particularly important in haemodynamic management. Hypotension is not a diagnosis, and similar arterial pressures may arise

from markedly different combinations of vascular tone, circulating volume, ventricular function, arterial load and venous pressure. A warning that hypotension is likely to occur may prompt earlier action, but without mechanistic information it may also encourage empiric and potentially inappropriate treatment. Fluids, vasopressors and inotropes are not interchangeable responses to an abnormal pressure, and each can be ineffective or harmful when applied to the wrong physiological phenotype [8].

The evolution of intelligent monitoring should therefore proceed from detection, to prediction, and ultimately to physiological phenotyping. Rather than merely identifying that instability is present or impending, future systems should integrate available signals to characterize its most plausible mechanism. Changes in arterial waveform morphology, pulse pressure, diastolic pressure, heart rate, electrocardiographic timing, plethysmography, capnography, ventilator interaction and venous signals may collectively distinguish reduced cardiac output, vasoplegia, impaired ventricular–arterial coupling, increased venous pressure or right ventricular–pulmonary vascular dysfunction more effectively than any isolated measurement.

This concept is increasingly supported by contemporary haemodynamic practice. The 2025 ESICM guidelines recommend combining clinical perfusion assessment, dynamic evaluation of fluid responsiveness, cardiac output or stroke-volume monitoring and echocardiography according to the clinical context rather than relying on a single universal variable [8]. Similarly, the ANDROMEDA-SHOCK-2 trial used sequential assessment of capillary refill time, pulse pressure, diastolic pressure, fluid responsiveness and bedside echocardiography to individualize fluids, vasopressors and inotropes in early septic shock. This physiology-based strategy improved a hierarchical composite outcome, primarily through a reduction in the duration of organ support [9]. Although such a structured protocol is not equivalent to artificial intelligence, it illustrates the potential clinical value of integrating multiple physiological domains to guide mechanism-specific therapy.

Because pressure, flow, microvascular perfusion and venous pressure are interdependent, meaningful phenotyping requires a model that localizes dysfunction across the circulatory pathway [10].

Such an approach could also improve interpretability. Instead of generating an opaque risk score, an intelligent monitor might indicate that deterioration appears predominantly related to reduced forward flow, loss of vascular tone, impaired microvascular perfusion, increasing venous impedance or right ventricular–pulmonary arterial uncoupling, while displaying the physiological changes supporting that interpretation. The output would represent a testable bedside hypothesis rather than an autonomous diagnosis. Clinicians could then confirm or refute it through examination, focused ultrasonography, additional monitoring or a carefully selected therapeutic challenge.

The ultimate objective is therefore not simply to predict that a patient will deteriorate, but to support earlier recognition of how and why their physiology is changing. Prediction may provide time but physiological phenotyping provides direction.

Integrating Haemodynamics Across Physiological Interfaces

A mechanistic approach to intelligent monitoring requires a framework that reflects how circulatory function is transmitted from the heart to the tissues and back through the venous and pulmonary circulations. Our proposed model divides this pathway into four interacting domains: left ventricular–systemic arterial coupling, arteriolar–capillary perfusion, capillary–venular pressure relationships and right ventricular–pulmonary arterial coupling [10]. These interfaces should not be interpreted as independent compartments, but as connected components through which

dysfunction can originate, propagate or become amplified.

The first interface describes the interaction between left ventricular performance and the arterial load against which the ventricle ejects. Arterial pressure alone cannot distinguish adequate flow from compensated or MAP-maintained low-output physiology. Integration of pulse pressure, ejection timing, arterial waveform morphology, waveform-derived pulsatile energy (the systolic area above diastolic pressure) and other flow-sensitive variables may provide a more complete representation of ventricular-arterial interaction. Importantly, many apparent measures of contractility are load-dependent and must therefore be interpreted within this coupling relationship rather than as isolated measures of myocardial performance.

The second interface concerns the transfer of flow and pressure from the arteriolar circulation to the capillary bed. This is where macrocirculatory variables encounter the microcirculation responsible for tissue perfusion. Capillary refill, skin temperature, mottling and potentially other optical or biochemical signals may reveal impaired peripheral perfusion despite apparently acceptable systemic pressure. Improvements in arterial pressure or cardiac output do not necessarily produce corresponding improvements in microvascular flow, a dissociation described as loss of haemodynamic coherence [11]. Such macro-microcirculatory incoherence may reflect primary microvascular dysfunction or a secondary limitation imposed elsewhere in the cardiovascular system.

The third interface incorporates the capillary, venular and systemic venous circulations. Effective organ perfusion pressure depends on the gradient between inflow and downstream pressure rather than arterial pressure alone. Elevated venous pressure can therefore impair renal, hepatic and other organ perfusion even when arterial pressure appears adequate. Venous Doppler, inferior vena cava assessment and other congestion signals may help characterize this downstream impedance and identify situations in which attempts to increase preload are more likely to worsen congestion than improve forward flow. The development of the venous excess ultrasound grading system illustrates how several venous signals can be integrated to quantify systemic congestion rather than interpreted individually [12].

The fourth interface describes right ventricular-pulmonary arterial coupling. The right ventricle is particularly sensitive to changes in afterload, intrathoracic pressure and pulmonary vascular resistance. Dysfunction at this interface can reduce left ventricular filling, impair systemic output and simultaneously increase systemic venous pressure. Relevant information may be derived from respiratory and ventilator signals, focused echocardiography and systemic venous waveforms, although continuous and non-invasive surrogates remain an important area for technological development.

By organizing heterogeneous signals according to their position within the circulatory pathway, artificial intelligence could identify emerging discordance among interfaces, localize the probable physiological constraint and prompt targeted assessment. The objective

would not be to reproduce a complete haemodynamic examination continuously, but to convert multiple signals into a clinically intelligible and testable representation of circulatory function.

Context-Specific Deployment Across Acute Care

A common physiological framework could support intelligent monitoring across the emergency department, operating room and intensive care unit, but the same model cannot simply be transferred unchanged between environments. Signal availability, event prevalence, physiological non-stationarity and the relevant prediction horizon differ substantially. Consequently, model inputs, calibration, alert thresholds and expected outputs must be adapted to the clinical setting.

In the ICU, the principal opportunity is longitudinal haemodynamic phenotyping. Prolonged monitoring permits characterization of changes in arterial pulsatility, vascular tone, ventricular-arterial interaction, peripheral perfusion and venous congestion across successive phases of illness.

The challenge is that ICU physiology is highly non-stationary: arrhythmia, spontaneous and mechanical ventilation, changes in PEEP, vasoactive drug titration, ultrafiltration, fever, agitation and sedation can all alter waveform morphology without representing a new pathological event. An effective model must therefore distinguish deterioration from expected variability and interpret changes in relation to contemporaneous interventions. The clinically relevant output is not simply an increased probability of circulatory failure, but whether the dominant constraint appears to have shifted—for example, from vasoplegia to impaired forward flow, or from preload responsiveness to venous congestion or RV-pulmonary arterial uncoupling [3,8,10].

The perioperative environment offers densely sampled waveforms linked to precisely timed physiological perturbations. Induction, initiation of positive-pressure ventilation, positional changes, surgical stimulation, blood loss, fluid administration and vasoactive therapy each expose a different component of cardiovascular reserve. Current arterial-waveform models can predict hypotension and reduce its depth and duration [2,4], an important achievement given the association between hypotension exposure and myocardial or renal injury [13]. However, prediction of the pressure event remains mechanistically incomplete. A falling mean arterial pressure accompanied by preserved pulsatile energy and widening pulse pressure suggests a different phenotype from one associated with decreasing systolic area, prolonged pre-ejection time or loss of arterial pulsatility. Integration of diastolic pressure, pulse pressure, ejection timing, waveform-derived energy and their responses to intervention could help distinguish a predominantly vasoplegic state from reduced stroke volume or impaired ventricular-arterial coupling. This distinction is necessary if prediction is to guide treatment rather than merely accelerate empiric vasopressor administration.

Emergency-department deployment presents the opposite problem: limited baseline information, shorter observation periods and predominantly non-invasive signals. Models may initially need to infer haemodynamic state from intermittent blood pressure measurements combined with electrocardiography, plethysmography, capnography, respiratory patterns and peripheral perfusion. Their output should explicitly reflect uncertainty and evolve as invasive waveforms, focused echocardiography, laboratory data and treatment-response information become available. In this setting, the most useful application may be recognition of compensated low-flow states or evolving macro-microcirculatory incoherence before conventional hypotension develops. Sequential reassessment is particularly important because the response to an initial fluid bolus, vasopressor or ventilatory intervention may contain more mechanistic information than the presenting measurements themselves [7–9].

Post-anaesthesia and monitored ward environments introduce a different statistical constraint: the lower prevalence of critical haemodynamic events reduces positive predictive value even when model sensitivity and specificity appear acceptable. Alert clustering, artifact rejection, persistence requirements and suppression of repeated notifications therefore become central design features rather than secondary implementation details. Continuous monitoring detects substantially more physiological abnormality than intermittent observation, but randomized evidence has not yet demonstrated consistent improvement in mortality, major complications or escalation of care [14]. This reinforces the distinction between detecting abnormal physiology and generating information that is sufficiently specific, interpretable and actionable to alter outcomes.

Cross-environment continuity may ultimately be as important as perfor-

mance within any single location. At present, physiological interpretation is effectively reset when a patient moves from the emergency department to the operating room, recovery unit or ICU. Preserving the preceding waveform trajectory, intervention timestamps and haemodynamic responses would allow subsequent measurements to be interpreted against an evolving patient-specific model. Postoperative hypotension could then be evaluated in the context of pre-induction pressure, arterial pulsatility, intraoperative blood loss, cumulative vasoactive exposure and prior response to fluid or vasopressor administration, rather than as an isolated threshold violation.

The objective is therefore not a universal deterioration score. It is a shared physiological architecture with context-specific inputs and outputs: early recognition under signal-limited conditions in the emergency department, mechanistic analysis of perturbation and response in the operating room, longitudinal phenotyping in the ICU, and highly specific escalation signals in lower-acuity environments. The cardiovascular physiology is continuous across these settings; the manner in which it must be inferred and presented is not.

Clinical Vignette: A Change in Haemodynamic Phenotype

A 72-year-old man presents to the emergency department with fever, jaundice and altered mental status. Laboratory investigations demonstrate cholestasis, hyperbilirubinaemia and elevated inflammatory markers, while ultrasonography shows a dilated common bile duct. Acute cholangitis with septic shock is suspected.

Shortly after arrival, his blood pressure falls to 92/36 mmHg. An arterial catheter is inserted, and the intelligent monitor generates an alert for haemodynamic instability with a predominantly vasoplegic phenotype. The arterial waveform demonstrates a wide pulse pressure, low diastolic pressure and steep diastolic decay, with preserved systolic upstroke and dP/dt. Pulsatile energy remains relatively maintained despite the low mean arterial pressure. The alert indicates that arterial pressure is being lost primarily through reduced vascular tone and rapid diastolic runoff rather than through primary myocardial dysfunction or severely reduced stroke volume.

Prompted by the alert, the clinical team reassesses the patient. He receives 500 mL of balanced crystalloid, producing only a modest haemodynamic response, and norepinephrine is initiated. As the infusion is titrated, diastolic pressure rises, the diastolic decay becomes less steep and mean arterial pressure returns to approximately 70 mmHg. The monitor displays progressive resolution of the vasoplegic pattern, supporting restoration of arterial tone as the principal mechanism of improvement. Antibiotics are administered, and urgent endoscopic retrograde cholangiopancreatography achieves biliary drainage and source control.

During the following 18 hours in the ICU, his clinical condition improves. Lactate decreases, peripheral perfusion normalizes and the norepinephrine dose is progressively reduced. His arterial pressure remains approximately 108/56 mmHg, with stable pulse pressure and waveform-derived pulsatile energy.

Later that day, the bedside nurse receives a new haemodynamic alert as the patient's blood pressure falls to 84/54 mmHg. Importantly, the monitor indicates that the current waveform differs from the patient's preceding vasoplegic pattern. Pulse pressure has narrowed from approximately 50 to 30 mmHg, while systolic waveform area and pulsatile energy have decreased. The MAP-to-diastolic pressure ratio and pulsatile energy relative to diastolic pressure are both lower than during the initial vasoplegic episode. In contrast to the initial presentation, diastolic pressure has not fallen disproportionately and the diastolic decay has not become steeper. Arterial dP/dt remains close to its preceding value.

Rather than identifying this as recurrent vasoplegia, the system alerts the

clinical team to a new low-pulsatility, preload-limited phenotype. The preserved dP/dt argues against an abrupt loss of pressure-generating capacity, although this load-dependent measure cannot be interpreted as isolated contractility. Together with the reduced pulsatility, this suggests a smaller effective stroke volume. The absence of recurrent rapid diastolic runoff points away from worsening vasoplegic physiology. The alert therefore prompts targeted assessment for a new cause of reduced ventricular filling rather than reflexive escalation of norepinephrine.

Focused echocardiography demonstrates a small, hyperdynamic left ventricle and a reduction in left ventricular outflow tract velocity-time integral compared with the previous examination. There has been no increase in fever or inflammatory markers to suggest worsening sepsis. Repeat laboratory testing shows that haemoglobin has fallen from 104 to 76 g/L. Shortly afterward, melena becomes apparent.

Urgent gastroscopy identifies an actively bleeding duodenal ulcer. Endoscopic injection and clip placement achieve haemostasis, and the patient receives red-cell transfusion with targeted volume replacement. Pulse pressure and systolic waveform area subsequently increase, pulsatile energy returns toward its previous range and mean arterial pressure recovers without a major escalation in norepinephrine.

The monitor documents resolution of the preload-limited pattern.

The monitor does not diagnose gastrointestinal haemorrhage; it recognizes that recurrent hypotension no longer matches the patient's preceding vasoplegic phenotype and prompts targeted reassessment before overt bleeding becomes apparent. The clinician remains responsible for confirming the haemodynamic abnormality, identifying its cause and directing treatment.

Conclusion

The next generation of physiological monitoring should do more than detect that a threshold has been crossed. By integrating waveform morphology, temporal trajectories, treatment responses and relationships across haemodynamic interfaces, intelligent systems may identify not only when deterioration is occurring, but how its underlying mechanism is evolving. Their greatest value may lie in recognizing transitions between phenotypes that appear similar when reduced to heart rate and arterial pressure alone. The subsequent step will be to connect this physiological intelligence with haemodynamic AI coaching. Once a monitor identifies a probable phenotype or a change in mechanism, an integrated coach could guide the clinician through targeted confirmation: reassessing perfusion, performing focused echocardiography, evaluating fluid responsiveness or venous congestion, and investigating haemorrhage or other reversible causes. This could help translate complex haemodynamic data into a reproducible bedside approach while reducing variation related to training, experience and access to specialist expertise [15,16].

Such systems should neither prescribe treatment autonomously nor replace clinical judgement. Their role is to sustain continuous attention, generate interpretable physiological hypotheses and support structured reassessment [15,17]. Realizing this potential will require prospective validation, acceptable alert burdens and evidence that phenotype-informed alerts and coaching improve clinical decisions and outcomes. The future monitor should not merely announce instability; it should help clinicians recognize when the physiology has changed, determine where to look next and apply sophisticated haemodynamic reasoning more consistently across acute-care environments.

Conflict of interest disclosure

Both authors are co-founders of ResusCare AI and have a financial interest in the company. ResusCare AI has developed a software suite designed to integrate advanced haemodynamic analytics into bedside patient monitors. This technology is currently undergoing clinical trials.

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