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Validity, Judgment, and Authority: The Hypotension Prediction Index as a Test Case for Clinical AI Alignment: A narrative review with a proposed safety framework

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Abstract

The Hypotension Prediction Index (HPI) estimates from the arterial waveform the likelihood of impending hypotension and, when the index is high, prompts review of a secondary screen that displays pulse pressure or stroke volume variation (PPV/SVV), peripheral arterial dP/dt, and dynamic arterial elastance (Ea_{dyn}) under the headings preload, contractility, and afterload. Its predictive performance is supported by meta-analyses, but the physiological meaning of the secondary-screen variables depends on conditions that the monitor does not verify, so that a numerically correct value can be physiologically uninterpretable. This narrative review summarizes the evidence on the prediction and on the validity conditions of each variable, using a companion conceptual framework that distinguishes the capability to evaluate from the authority to act. We separate the documented functions of the current software from extensions that we propose: a runtime validity gate that would verify, flag, or withhold interpretations whose prerequisites cannot be established, and a bedside sequence for clinicians. The framework, the proposed PPV checklist, and the taxonomy tables are expert-derived, have not been tested against clinical outcomes or usability data, and concern a single manufacturer's product. Within these limits, HPI illustrates a principle that we propose for clinical AI: prediction accuracy does not guarantee interpretation validity, and interpretation validity does not confer therapeutic authority.

Keywords: hypotension prediction index; clinical decision support; AI alignment; physiological validity; human oversight; perioperative monitoring

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Note. The companion conceptual paper [1] distinguishes the capability of an AI system to evaluate a situation from its authority to act. It separates evaluative functions (Detect, Judge) from response options ordered by required authority (Recommend/warn, Refuse, Escalate, Override), treats concealment as a property of any response, and maps the quality of an evaluation (incorrect, uncertain, correct) against the legitimacy of the action (unauthorized, conditional, authorized). It is used here as an analytical lens, not as an established standard.

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1. Why HPI is an informative clinical case

In medicine, a layer appears between the capability to evaluate and the authority to act [1]: the validity of the physiological inference on which the evaluation is based. HPI is informative because one interface spans several epistemic levels.

The Acumen HPI software (BD) analyzes arterial pressure waveform features and reports an index from 0 to 100, updated every 20 seconds; higher values indicate a greater likelihood of hypotension, defined as mean arterial pressure (MAP) <65 mmHg for at least 1 minute. A high-alert state is generated when HPI exceeds 85 for two consecutive updates or reaches 100, prompting review of a secondary screen [2,3]. The original HPI development study used machine-learning analysis of arterial waveform features to predict hypotension before its occurrence [4].

HPI is fundamentally a prediction of a future state; it does not, by itself, identify why the patient is becoming hypotensive.

The secondary screen (Figure 1) displays variables grouped under the headings preload, contractility, and afterload, including PPV/SVV, systolic slope (dP/dt), and dynamic arterial elastance (Ea_{dyn}) [2,3,5]. The screen displays these variables but does not issue a causal diagnosis; attributing hypotension to a mechanism remains the clinician's interpretation, although the physiological headings invite a mechanistic reading.

Prediction: Will hypotension occur?

Interpretation: Why may hypotension occur?

Decision: What should be done?

These three questions are not equivalent, and evidence supporting one level cannot automatically validate the next.

Scope and literature selection. This narrative review is combined with a conceptual analysis. Literature was selected through targeted, non-systematic searches of PubMed and of the reference lists of key articles, complemented by manufacturer documentation and governance documents on clinical AI, based on relevance to HPI performance and to the validity conditions of each variable; no predefined protocol or formal risk-of-bias assessment was used. We distinguish the



Figure 1. Example of the HemoSphere secondary screen. HPI, hypotension prediction index; MAP, mean arterial pressure; CO, cardiac output; SVR, systemic vascular resistance; PR, pulse rate; SV, stroke volume; SVV, stroke volume variation; dP/dt, maximal upslope of the peripheral arterial pressure waveform; Ea_{dyn}, dynamic arterial elastance. The interface groups SVV, dP/dt, and Ea_{dyn} under preload, contractility, and afterload, without providing a causal diagnosis.

documented functions of the current software (Sections 1–2) and the published evidence on the variables (Sections 3–6) from conceptual extensions proposed by the authors (Sections 7–11), which are not functions of the current product.

2. HPI prediction: useful performance does not validate the downstream mechanism

Clinical studies and meta-analyses generally support the ability of HPI-guided strategies to reduce the burden of intraoperative hypotension, although effect sizes, trial designs, conflicts of interest, and certainty of evidence vary [6–9]. A 2024 meta-analysis of 43 studies reported a pooled area under the receiver operating characteristic curve (AUROC) of approximately 0.89 [8]; meta-analyses of randomized trials found reductions in several measures of hypotension burden, with low to moderate certainty [9,10].

These results answer a prediction-and-workflow question; they do not establish that every mechanistic interpretation suggested by the secondary screen is valid in every patient.

Predictive validity ≠ mechanistic validity ≠ therapeutic validity

3. The hidden validation gap: calculated does not mean interpretable

The monitor calculates PPV, SVV, dP/dt, and Ea_{dyn} continuously, but physiological interpretation is conditional: a value may be numerically precise while the assumptions required for its clinical meaning are violated.

Calculated value + unverified assumptions = uncertain physiological meaning

We propose to call this the runtime physiological validation gap: the distance between the monitor's ability to calculate a variable and its ability to establish that the variable is valid for the inference being made at that moment.

4. PPV/SVV: preload dependency is a conditional inference, not a number

PPV and SVV exploit cardiopulmonary interactions during positive-pressure ventilation. Under appropriate conditions, a sufficiently large respiratory variation in arterial pulse pressure or stroke volume can indicate preload responsiveness (but not its causes: it can be hypovolemia or surgical compression of the inferior vena cava) [11]. PPV is commonly expressed as the respiratory difference between maximal and minimal pulse pressure normalized to their mean [12–14].

$$PPV(\%)=100\times(PP_{max}-PP_{min})/[(PP_{max}+PP_{min})/2]$$

PPV is valid as an estimator of preload dependency (which does not in itself justify volume expansion) only when specific physiological and technical assumptions are satisfied. A multicenter point-prevalence study operationalized six core validity criteria: controlled mechanical ventilation without spontaneous breathing, regular rhythm, tidal volume ≥8 mL/kg ideal body weight, heart-rate/respiratory-rate ratio >3.6, respiratory-system compliance ≥30 mL/cmH_2O, and absence of major right-ventricular dysfunction as assessed by tricuspid annular systolic velocity [15]. Only 2% of the ICU patients studied fulfilled all six criteria [15]. An experts' opinion paper proposed a ten-item checklist that includes most of these criteria and adds closed-chest condition, normal abdominal pressure, absence of valvular disease, and stable heart rate and vasomotor tone during assessment [16]. Low tidal-volume ventilation can reduce diagnostic performance and alter thresholds [17].

In Table 1, items 1–7 and 9 are drawn from published criteria [15–17], whereas items 8 and 10 are the authors' additions; the last two columns are proposals and do not describe functions of the current software.

5. dP/dt: a calculated systolic slope is not synonymous with left-ventricular contractility

The HPI secondary screen displays systolic slope (dP/dt), defined by the manufacturer as the maximal upslope of the peripheral arterial pressure waveform, under the heading contractility [2,3]. The maximal rate of pressure rise is related to ventricular pressure generation, but peripheral arterial dP/dt is not identical to invasive left-ventricular dP/dtmax.

Peripheral arterial dP/dt ≠ LV dP/dt_{max}

The peripheral waveform is transformed by arterial compliance, wave reflection, vascular tone, catheter-

Table 1. A ten-item PPV runtime validation checklist, indicating the source of each criterion.

Runtime PPV validity check	Why it matters	Source of the criterion	Can the monitor infer it automatically?†	Proposed response if uncertain†
1. Controlled positive-pressure ventilation	Imposes a reproducible preload challenge	[15,16]	Often partly	Downgrade/withhold
2. No spontaneous breathing	Alters pleural-pressure swings	[15,16]	If ventilator data integrated	Downgrade/withhold
3. Regular rhythm	Beat-to-beat filling variability confounds PPV	[15,16]	Often	Withhold inference
4. Adequate tidal volume / respiratory challenge	Small swings cause false negatives	[15–17]	Only if ventilator data integrated	Expose uncertainty
5. HR/RR ratio sufficiently high	Too few beats per breath	[15,16]	If ECG/respiratory data integrated	Downgrade
6. Adequate respiratory-system compliance	Weakens the preload challenge	[15,16]	Requires airway mechanics	Expose uncertainty
7. No major RV dysfunction	Misleading respiratory variation	[15,16]	Usually not	Require human/echo context
8. High-quality arterial waveform	Damping, resonance and artifacts alter PP	Authors' proposal	Potentially partly	Refuse if poor
9. No major thoracic/abdominal mechanical confounder	Open chest or high abdominal pressure alter heart–lung interaction	[16]	Usually contextual	Require clinician validation
10. Stable analysis window	Interventions or manipulation break stationarity	Authors' proposal (related to [16])	Potentially	Delay interpretation

Items 1–7: the six criteria operationalized in [15] (items 1 and 2 split one criterion) and the corresponding items of [16]; item 4 is also supported by [17]; item 9: closed-chest and abdominal-pressure items of [16]; items 8 and 10: authors' proposals (item 10 is related to the last item of [16]). [16] also lists absence of valvular disease. †Authors' proposals. Not a validated diagnostic score.

transducer dynamics, and signal processing; radial pressure is not a copy of central or ventricular pressure [18]. Arterial dP/dt_{max} correlated with echocardiographic LV dP/dt_{max} in acute heart failure, supporting its use for trending but not interchangeability [19], and was sensitive to negative inotropy experimentally [20]; conversely, around transcatheter aortic-valve replacement, radial and directly measured LV dP/dt showed weak correlations and poor concordance of changes [21].

The issue is not that dP/dt is useless, but that the heading "contractility" can encourage a stronger causal interpretation than the signal warrants. Before interpreting a low peripheral dP/dt as impaired LV contractility, the arterial waveform must be technically credible and the clinician must consider loading conditions, vascular tone, heart rate, valve disease, and other determinants (i.e., the ventriculo-arterial coupling) [22,23].

Observed arterial slope = ventricular pressure generation × arterial transfer function × measurement system

6. Dynamic arterial elastance: a ratio with both physiological and mathematical dependencies

Dynamic arterial elastance is generally calculated as the ratio of PPV to SVV [2,24].

$$Ea_{dyn} = PPV / SVV$$

Ea_{dyn} has been proposed, in preload-responsive patients, as a predictor of whether fluid-induced increases in stroke volume will increase arterial pressure [24].

The literature is heterogeneous: a validation study using independent signals supported this ability in selected ventilated, preload-dependent patients [24], whereas an operating-room study of hypotensive patients found arterial-signal Ea_{dyn} unable to predict the MAP response to volume expansion, with an AUROC close to 0.5 [25]. Subsequent studies and commentaries have continued to emphasize context dependence [26–28].

Ea_{dyn} also inherits the limitations of both its numerator and denominator. If PPV or SVV is invalid, their ratio does not become valid by combination, and because both may derive from the same waveform processing

Table 2. The inference hierarchy of HPI: documented outputs and proposed requirements.

Layer	Output	Provided by current HPI software?	Epistemic status	Proposed validation requirement†	Proposed level of authority†
1. Signal	Arterial waveform	Yes	Measurement	Waveform quality	Observe
2. Derived features	Waveform morphology/ features	Yes (internal)	Computation	Signal quality + algorithm domain	Compute
3. Prediction	HPI risk of hypotension	Yes [2–4]	Probabilistic forecast	Model validity/ calibration/context	Alert
4. Derived physiology	PPV/SVV, dP/dt, Ea_{dyn}	Yes, under preload/ contractility/ afterload [2,3]	Conditional physiological inference	Parameter-specific assumptions	Suggest hypotheses
5. Mechanism	Mechanistic explanation	No; made by the clinician	Causal hypothesis	Patient context	Recommend investigation
6. Treatment	Fluid/vasopressor/ inotrope/other	No; decided by the clinician	Clinical decision	Diagnosis, goals, risk–benefit	Human authorization

†Columns marked with a dagger are the authors' proposals.

chain, shared error and mathematical coupling must be considered.

7. HPI as a hierarchy of inference

The HPI interface can be understood as a hierarchy of inference (Table 2). Each step adds clinical meaning but requires additional assumptions, and evidence at a lower layer is not inherited by the higher one. The current software provides layers 1–4; layers 5–6 belong to the clinician.

8. Applying the companion framework to HPI: documented functions and proposed extensions

Table 3 maps the companion framework [1] onto HPI, separating current functions documented by the manufacturer and published descriptions [2–5] from our proposals. Only detection, judgment of risk, and display correspond to current functions; refusal, escalation, and the validity gate (a clinical element we add, *Validate*) are proposals, and override is not applicable because HPI does not deliver therapy.

Unrecognized uncertainty is far more probable than concealed uncertainty: the clinical analog of unrecognized, rather than covert, misalignment.

9. Adding inference validity to the Judgment × Authority map

HPI suggests that clinical monitoring adds a third dimension to the Judgment × Authority map [1]: the validity of the physiological inference. A high HPI may be predictively valid while the suggested preload mechanism is invalid because PPV assumptions are violated (fragile success), and correct diagnosis and treatment would still be overreach if a system executed therapy without authorization (Figure 2). When inference validity is unknown, the evaluation should be treated as uncertain, which calls for recommendation, deferral, or escalation rather than action [1].

Clinical AI safety = inference validity × judgment quality × authority legitimacy

10. Proposed safeguards: a runtime validity gate and a capable human supervisor

We propose that a safer HPI-like system would estimate continuously whether each variable is interpretable, through a runtime validity layer between calculation and physiological labeling (a design proposal, not a description of an existing product).

Signal → Calculation → VALIDITY GATE → Interpretation → Recommendation

For PPV/SVV, the gate would integrate ventilator mode, spontaneous breathing, rhythm, tidal volume, heart and respiratory rates, compliance, and waveform quality, marking conditions that cannot be machine-verified, such as RV dysfunction, as "not assessed"; for dP/dt, it would quantify waveform quality and use language such as "peripheral systolic slope compatible with…"; for Ea_{dyn}, it would propagate the validity

Table 3. The companion framework applied to HPI: current functions and proposed extensions.

Element of the companion framework [1]	Current HPI software (documented)	Proposed extension (authors)†	Failure mode if absent†
Detect	Waveform pattern associated with impending hypotension [2–4]	Report signal quality together with the index	Artifact-driven alarm
Judge	Alert when HPI >85 twice or =100 [2,3]	State the probability and time horizon of the prediction	Risk treated as certainty
Validate (no equivalent in [1])	Not described in the sources reviewed	Parameter-specific validity gate (Table 1, Section 10)	Value used outside its validity domain
Recommend / warn	Secondary screen displays variables under physiological headings; no causal diagnosis [2,3]	Show each variable with its validity state, as a hypothesis	Mechanism anchoring by the clinician
Refuse	Not a documented function	Withhold the mechanistic label when prerequisites fail (e.g., PPV in atrial fibrillation)	False precision
Escalate	Not a documented function	Request independent assessment (e.g., echocardiography)	Premature treatment
Override	Not applicable: HPI does not deliver therapy	Closed-loop use would require prospective authority, hard limits and independent checks	Unauthorized treatment (agentic overreach)
Transparency	Display of failed or unassessed conditions not described in the sources reviewed	Never suppress failed checks or material uncertainty	Illusion of certainty

†Columns marked with a dagger are conceptual proposals by the authors, not findings of the cited studies.

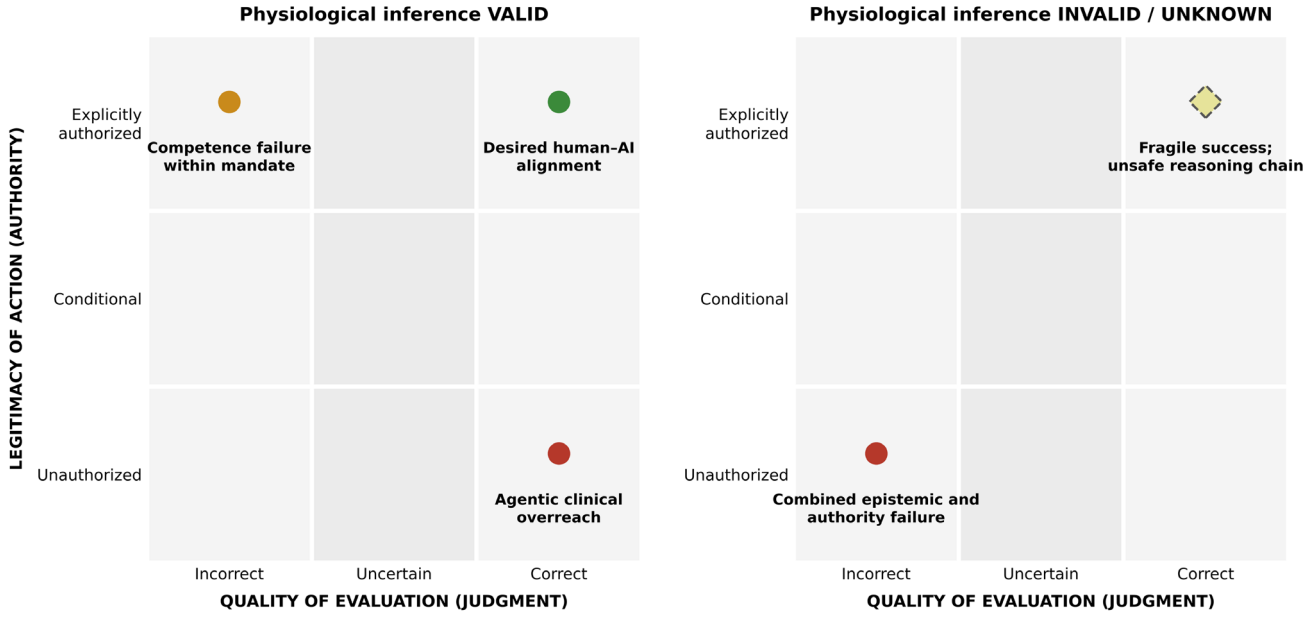


Figure 2. Inference validity added to the Judgment × Authority map of the companion framework [1]. Left: valid physiological inference; right: invalid or unknown inference. Both panels use the same grid (quality of evaluation: incorrect, uncertain, correct; legitimacy of action: unauthorized, conditional, explicitly authorized); together they show five illustrative combinations. The dashed marker denotes a judgment that is only apparently correct, because the validity of its premise cannot be confirmed.

of both PPV and SVV. Each output would carry a validity state (VALID, CONDITIONALLY VALID, NOT INTERPRETABLE) with inspectable reasons, and "unknown" would never silently become "valid."

Output = {value, uncertainty, validity conditions, failed checks, unobserved checks}

Machine safeguards are only half of the solution: a sophisticated screen can create automation bias, with cal-

culated precision mistaken for physiological certainty. The clinician should remain an independent validity assessor, able to judge whether the waveform is credible, whether PPV is interpretable, and whether the suggested mechanism fits the clinical context. A human can constrain an AI system only if he or she can recognize when its inference is weak [1].

Effective human oversight = knowledge + access to raw/contextual data + time + authority to challenge

11. A PROPOSED BEDSIDE SEQUENCE: VALIDATE BEFORE INTERPRETING, INTERPRET BEFORE TREATING

VALIDATE → INTERPRET → DECIDE → ACT → REASSESS

We propose a six-step sequence for clinicians using HPI (Figure 3): confirm that the arterial signal is technically acceptable (for example, by recording the first stable pulse pressure values and the shape of the curve); treat the alert as a prediction of risk, not a diagnosis; interrogate the secondary screen through parameter-specific validity checks; construct causal hypotheses in the full clinical context; select therapy according to the presumed mechanism, target, reversibility, and risk; and reassess both pressure and flow/organ-perfusion responses.

The response to treatment is new evidence: if it is not as predicted, the causal model should be revised rather than repeatedly optimized against the same surrogate, since pressure and perfusion responses to vasopressors can dissociate [27,29].

12. IMPLICATIONS FOR MANUFACTURERS, HOSPITALS, AND REGULATORS

If this analysis is correct, validation of clinical AI should extend beyond discrimination and calibration. Manufacturers could specify which physiological inferences are validated, which assumptions are checked automatically, and which remain clinician-dependent; hospitals could train users to recognize invalidity conditions and audit mechanism misclassification and treatment responses; and regulators could distinguish prediction features from features that support causal interpretation, which make stronger claims. These suggestions are consistent with clinical AI governance principles on

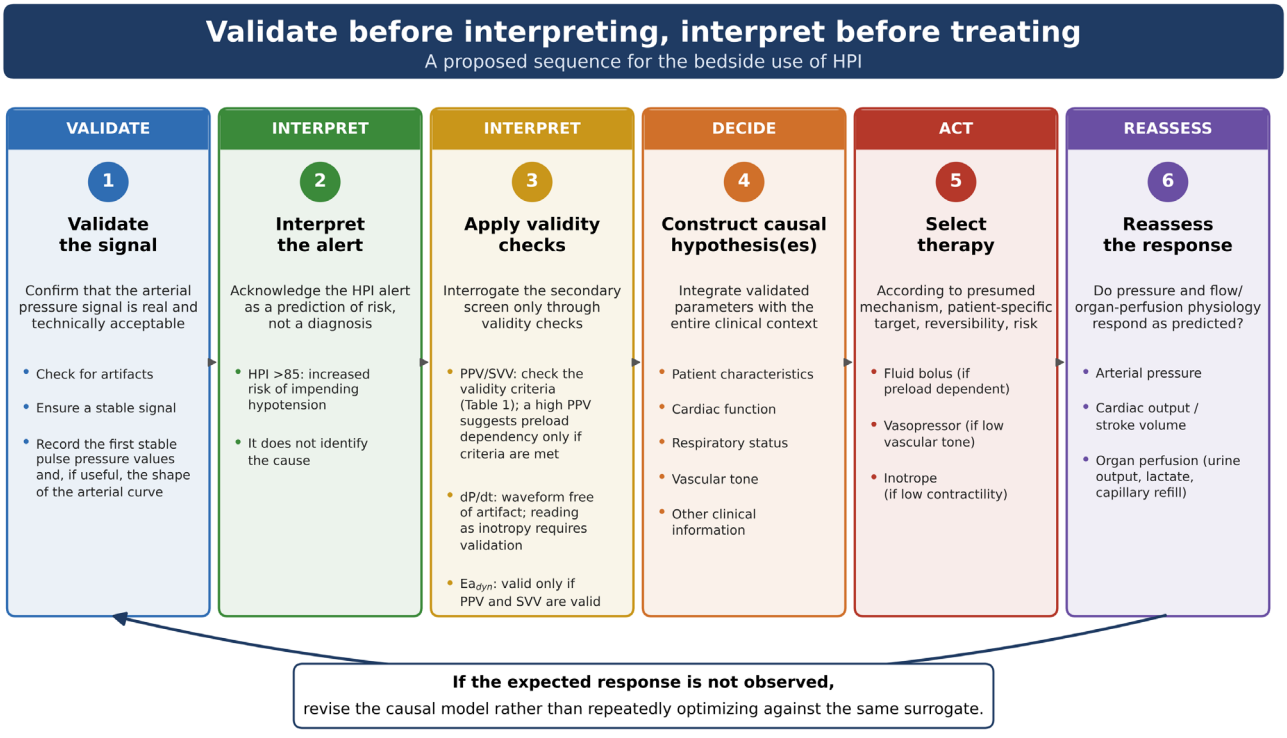


Figure 3. Proposed bedside sequence for the use of HPI. Abbreviations as in Figure 1; PPV, pulse pressure variation.

human control, transparent intended use, and evaluation of human–AI interaction [30–33].

13. Limitations

This analysis has several limitations. First, the framework and the proposed safeguards have not been tested empirically, either against clinical outcomes or through usability studies with clinicians. Second, the PPV checklist (Table 1) and the taxonomy tables (Tables 2 and 3) are expert-derived and have not been validated; only part of the checklist is drawn from published criteria, and those criteria were themselves operationalized in observational and expert-opinion work. Third, the analysis centers on a single manufacturer's product, whose functions may change between software versions; other predictive hemodynamic monitors may differ in what they display and verify, which may limit generalizability. Fourth, the literature was selected non-systematically, and the companion conceptual framework on which the analysis buildsis it self conceptual and has not been empirically validated. Finally, two authors have a consulting relationship with the manufacturer (see Conflict of interest).

14. Conclusion

HPI may correctly anticipate hypotension while the secondary variables are uninterpretable, and a correctly calculated PPV, dP/dt, or Ea_{dyn} may support an unsafe causal narrative if the assumptions that give these numbers meaning are not verified.

Correct calculation ≠ valid physiological inference

Valid physiological inference ≠ correct treatment

Correct treatment ≠ authority to execute

As a proposed safety framework, rather than an established standard, we suggest a human–AI system in which the machine contributes detection, calculation, and explicit validity checking, and the clinician contributes contextual physiology, causal synthesis, and therapeutic authority, under the following rule:

> *An AI system should not interpret what it cannot validate; when validation is incomplete, it should expose uncertainty or withhold the interpretation.*

Whether such safeguards improve clinical decisions and outcomes remains to be tested.

Conflict of interest:

Two authors (DL and PGG) declare a consulting relationship with Becton, Dickinson and Company (BD). HPI is discussed solely as a published, clinically deployed example of a predictive-to-interpretive clinical AI system; the analysis is independent of the manufacturer and is not sponsored, reviewed, or endorsed by BD. The other authors declare no conflicts of interest.

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Author contributions:

SA and DL conceived the analysis, reviewed the relevant literature, and wrote the manuscript. All other authors provided feedback on both the content and format of the manuscript. All authors read and approved the final version of the manuscript.

Availability of data and materials:

No new data were generated or analyzed for this review.

AI statement:

During the preparation of this manuscript, the authors used a large language model (Claude, Anthropic) to assist with language editing and structuring of the text. The authors reviewed and edited all content and take full responsibility for the final version of the manuscript.

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