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Perspective

Beyond Mean Arterial Pressure: The Pulse Energy Ratio as a Potential Framework for Perioperative Hemodynamic Interpretation

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Abstract

Continuous arterial pressure monitoring is common during anesthesia, yet interpretation remains largely centred on systolic, diastolic, and mean arterial pressure (MAP). We have recently proposed the Pulse Energy Ratio (PER) as a dimensionless descriptor of the pulsatile contribution to arterial pressure. While validation studies are underway, PER may have particular relevance to anesthesia, where rapid changes in loading conditions, cardiac performance, vascular tone, ventilation, and surgical conditions create an accelerated microcosm of hemodynamic disturbance. We hypothesize that directional changes in PER could identify moments at which structured physiological reassessment is warranted. A decreasing PER may precede overt hypotension, although this temporal relationship has not yet been demonstrated. During hypotension, a concomitant decrease in PER could prompt assessment of ventricular ejection and venous return, whereas a preserved or increasing PER could raise the possibility of predominant vasodilation. PER is hypothesized to provide a continuous signal that may prompt physiological reassessment before conventional hypotension thresholds are crossed. These hypothesized patterns are proposed as prompts for focused physiological assessment rather than diagnostic phenotypes or treatment directives and require prospective validation.

Keywords: pulse energy ratio; arterial waveform; perioperative hemodynamics; intraoperative hypotension; vasoplegia; hemodynamic monitoring

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1. Introduction

Arterial pressure monitoring is a cornerstone of anesthetic practice. Current perioperative consensus recommendations emphasize avoiding clinically important hypotension and directing treatment toward its presumed cause [1]. This reflects a substantial observational literature associating the depth and duration of intraoperative hypotension with myocardial injury, acute kidney injury, and mortality [1,2]. In practice, however, the continuous signal provided by an arterial catheter is commonly reduced to systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial pressure (MAP).

MAP is an important pressure variable, but it does not identify the hemodynamic mechanism producing it. Similar MAP values may result from different combinations of venous return, ventricular ejection, heart rate, arterial compliance, wave reflection,

peripheral runoff, and vasomotor tone. In patients undergoing major abdominal surgery, MAP showed no clinically meaningful relationship with cardiac index, demonstrating that arterial pressure cannot be assumed to represent systemic blood flow [3]. Data-driven analyses have also identified distinct mechanisms of intraoperative hypotension, including myocardial depression, bradycardia, vasodilation, hypovolemia, and mixed states [4]. A pressure threshold may therefore identify hypotension without defining its underlying physiology.

The arterial waveform reflects the interaction between ventricular ejection and the arterial system [5–7]. Commercial pulse-wave analysis systems use waveform characteristics to estimate stroke volume or cardiac output, but may require dedicated hardware, calibration, proprietary algorithms, or assumptions about vascular properties [7]. A simpler waveform-derived index could potentially support physiological interpretation without attempting to calculate absolute blood flow.

We recently proposed the Pulse Energy Ratio (PER), a dimensionless ratio of the integrated arterial pressure–time area above DBP to the DBP–time reference area over the same cardiac cycle [8]. When MAP is obtained by time-averaging that same arterial waveform, PER is algebraically determined by integrated MAP and DBP and does not constitute mathematically independent waveform information. Its proposed role is to make the relationship between the pulsatile and diastolic pressure components explicit and continuously trendable. Whether this representation provides incremental value beyond MAP, DBP, pulse pressure, or other simple pressure-derived ratios remains unknown.

To translate changes in PER into a structured physiological assessment, we use the four-interface model of hemodynamics [9]. This framework examines the left ventricular–arterial interface (Interface 1), the arteriolar–capillary interface (Interface 2), the capillary–venular interface (Interface 3), and right ventricular–pulmonary arterial coupling (Interface 4). Although PER is derived from the systemic arterial waveform and therefore most directly reflects events at Interface 1, changes in the ratio may represent the downstream expression of altered physiology elsewhere in the circulation. PER should consequently be interpreted as a prompt to assess the relevant interfaces rather than as a diagnosis in itself.

The perioperative environment may be particularly suitable for investigating this framework. Induction of anesthesia, neuraxial blockade, hemorrhage, fluid administration, vasoactive therapy, positive-pressure ventilation, positional changes, pneumoperitoneum, and surgical manipulation produce frequent and temporally defined changes in loading conditions and vascular tone. Continuous waveform acquisition also permits the relationship between physiological interventions and subsequent waveform changes to be examined. This article describes the physiological basis of PER, proposes an interface-based approach to its perioperative interpretation, and defines the principal requirements for future evaluation.

This Communication is a conceptual, hypothesis-generating article and contains no original clinical data. PER has not undergone clinical validation. Unless otherwise specified, the proposed physiological interpretations, clinical scenarios, temporal relationships, and treatment-response patterns presented herein are illustrative hypotheses rather than established diagnostic, predictive, or therapeutic findings.

2. Physiological Basis and Interpretation of PER

The arterial pressure waveform is produced by the interaction between ventricular ejection and the arterial system. Its amplitude and contour are influenced by stroke volume, the pattern and duration of ventricular ejection, arterial compliance, vascular tone, peripheral runoff, wave reflection, heart rate, and the site of measurement [5–7,10]. No isolated waveform-derived variable can therefore be interpreted as a direct measure of

cardiac output or ventricular performance without considering the arterial system in which the pressure wave is generated.

For each cardiac cycle, PER is calculated as:

$$\text{PER} = \int 0\text{RR}[\text{P(t)} - \text{DBP}]\,\text{dtDBP} \times \text{RR} \quad (1)$$

where P(t) is the instantaneous arterial pressure at time t throughout the cardiac cycle, DBP is diastolic blood pressure, and RR is the duration of the cardiac cycle. P(t) therefore represents the continuously sampled arterial pressure during both systole and diastole, not a single measurement taken during systole. The numerator is the integrated pressure–time area above the DBP baseline, and the denominator is the DBP–time reference area over the same cycle. Because both have pressure–time units, PER is dimensionless.

When MAP is obtained by time-averaging the same arterial waveform over the same cardiac cycle:

$$\text{MAPint} = 1\text{RR} \int 0\text{RRP(t)}\,\text{dt} \quad (2)$$

PER is algebraically equivalent to:

$$\text{PER} = \text{MAPint} - \text{DBPDBP} = \text{MAPintDBP} - 1 \quad (3)$$

PER therefore contains no information mathematically independent of integrated MAP and DBP under these conditions. When MAP is conventionally estimated from SBP and DBP using a fixed formula, this equivalence does not necessarily apply because the estimated MAP does not account for the measured waveform morphology or the relative durations of systole and diastole. Any difference between PER and a conventionally estimated MAP would therefore reflect the measured waveform integral and the limitations of the estimation formula. Pulse pressure depends on the systolic and diastolic extrema, whereas PER relates the time-averaged pressure contribution to its diastolic reference. Whether this representation provides clinically useful information beyond MAP, DBP, pulse pressure, or other simple pressure-derived ratios remains unknown.

The term "energy" is retained as the operational name of the proposed index. PER does not measure energy in the physical sense because its calculation does not incorporate blood flow or displaced volume. It therefore cannot quantify hydraulic energy, ventricular work, stroke volume, cardiac output, or myocardial energetics. A more physically precise description is a normalized pulsatile pressure–time area.

DBP both defines the baseline subtracted from the waveform and forms the denominator of PER. A marked decrease in DBP may therefore increase PER without an increase in the absolute pulsatile pressure–time area. Conversely, an increase in DBP may reduce PER despite preservation of integrated MAP. PER should consequently be examined together with integrated MAP, DBP, SBP, pulse pressure, and the underlying waveform rather than interpreted as an isolated value.

Waveform fidelity must be assessed before interpreting a change in PER. The transducer should be correctly zeroed and levelled, the catheter and tubing inspected for air bubbles, kinking, obstruction, or clot, and the dynamic response assessed with a fast-flush test when appropriate. The waveform should also be examined for damping, resonance, implausible morphology, and changes related to rhythm or the arterial sampling site. Comparison with a non-invasive cuff pressure or a more central arterial site may be useful when a clinically important discrepancy is suspected [10,11]. In irregular rhythms, beat-to-beat PER values may require averaging or median filtering across several cardiac cycles.

PER should preferentially be trended within the same patient using the same monitoring system and arterial sampling site. An otherwise unexplained change may serve

as a prompt to assess the circulatory interfaces, but it does not identify the mechanism responsible for that change or determine the appropriate treatment [9].

3. Hypothesized Perioperative PER Patterns

PER should primarily be interpreted through directional changes from an individual patient's stable baseline. The combined behaviour of MAP, DBP, pulse pressure, PER, and the underlying waveform may provide a framework for recognizing changes that warrant physiological reassessment. The patterns described below are neither mutually exclusive nor diagnostic, and mixed hemodynamic states are common.

3.1. Decreasing PER Before Hypotension

A progressive decrease in PER may occur while MAP remains above a conventional hypotension threshold. This may reflect a decrease in integrated MAP relative to DBP, including a situation in which a rise in DBP initially preserves MAP while systolic pressure, pulse pressure, and PER decrease. Such a pattern could accompany an evolving hemodynamic disturbance before conventional hypotension develops, although this temporal relationship has not been demonstrated clinically.

Waveform fidelity should first be confirmed using the criteria described above [10,11]. If the signal is reliable, the change may prompt assessment of ventricular ejection and loading conditions, vascular tone, blood loss, ventilation, right ventricular function, and venous congestion. Relevant measurements may include left ventricular outflow tract velocity-time integral (LVOT-VTI), ventricular systolic function, dynamic left ventricular outflow tract obstruction, right ventricular–pulmonary arterial coupling, and indices of venous congestion [9]. The clinical context should determine which interfaces receive initial attention.

Arterial-waveform analysis has demonstrated that information preceding a conventional hypotension threshold may be identified within the continuous pressure signal [12,13]. However, these studies evaluated machine-learning approaches rather than PER and provide no evidence that PER itself predicts hypotension. The sensitivity, specificity, and lead time of any change in PER remain unknown.

3.2. Hypotension with Decreasing PER

When MAP and PER decrease together, the pulsatile pressure contribution has fallen relative to the diastolic pressure reference. This pattern is compatible with reduced ventricular ejection but is not specific for it. Potential determinants include reduced venous return, hemorrhage, impaired ventricular contractility, dynamic left ventricular outflow tract obstruction, altered arterial compliance or vascular tone, changes in wave reflection or peripheral runoff, heart rate, ventilation, and the arterial sampling site [5–7,10].

After confirming waveform fidelity, a decreasing PER may prompt broad physiological reassessment rather than preferentially identify a single mechanism. Assessment may include ventricular filling and systolic function, LVOT-VTI, right ventricular–pulmonary arterial coupling, venous congestion, blood loss, vascular tone, and relevant changes in ventilation or surgical conditions [9]. The clinical context should determine the relative priority assigned to each component.

A falling PER does not establish fluid responsiveness or demonstrate that fluid administration will improve the circulation. Impaired forward flow may coexist with elevated venous pressure or organ congestion. Treatment should therefore be directed toward the physiological constraint identified through clinical and hemodynamic assessment rather than toward PER itself.

3.3. Hypotension with Preserved or Increasing PER

When MAP decreases while PER remains stable or increases, DBP has generally fallen proportionately as much as, or more than, the integrated pulsatile pressure contribution. Because DBP forms the denominator of PER, a marked decrease in DBP may mathematically increase the ratio without an increase in the absolute pulsatile pressure–time area. This pattern should not be interpreted as evidence of preserved stroke volume or isolated vasoplegia.

In an appropriate clinical context, reduced vascular tone represents one possible explanation. This pattern might occur following induction of general anesthesia, neuraxial blockade, administration of vasodilating medications, or the development of an inflammatory vasodilatory state. In one prospective perioperative cohort, hypotension after induction with sufentanil, propofol, and rocuronium was predominantly associated with decreased systemic vascular resistance, while stroke volume index and cardiac index remained relatively stable [14]. This supports considering vasodilation as a possible mechanism of peri-induction hypotension but does not validate PER as a marker of vascular tone.

Changes in arterial compliance, wave reflection, peripheral runoff, heart rate, ventricular ejection, and arterial sampling site may produce a similar PER pattern [5–7,10]. Vasodilation may also coexist with reduced venous return or ventricular dysfunction. A preserved or increasing PER may consequently prompt assessment of vascular tone and Interface 2, while ventricular ejection, venous return, right ventricular function, and waveform fidelity are also evaluated [9].

3.4. PER Responses to Perioperative Interventions

Changes in PER following a defined intervention may be examined as a corroborative trend but cannot independently establish treatment response. Following fluid administration, an increase in MAP accompanied by a rise in PER might be consistent with improved pulsatile pressure generation, but this interpretation should be supported by LVOT-VTI or another measure of forward flow. The absence of an increase in PER does not exclude fluid responsiveness because simultaneous changes in DBP, vascular tone, and arterial compliance may alter the ratio.

Vasopressor administration may increase MAP predominantly through an increase in DBP. PER may consequently remain unchanged or decrease even when the intended vascular effect has occurred. Such a decrease should not automatically be interpreted as worsening cardiac performance. Conversely, increased arterial load may reduce ventricular ejection in a susceptible patient. Distinguishing a denominator effect from afterload-related deterioration requires reassessment of Interface 1 and an independent measure of forward flow [5,9].

Inotropic therapy might increase PER if pulsatile pressure generation increases, but concurrent changes in heart rate, vascular tone, and loading conditions may modify the response. Changes in positive end-expiratory pressure, tidal volume, patient position, pneumoperitoneum, or surgical retraction may similarly alter venous return and right ventricular afterload. A temporally associated change in PER may identify a moment for reassessment, but it cannot establish the responsible mechanism.

The proposed role of PER is to display changes in the relationship between the pulsatile and diastolic pressure components and potentially prompt structured physiological assessment. It should not independently determine treatment or serve as a therapeutic endpoint. The derivation of PER and the hypothesized approach to its perioperative interpretation are summarized in Figure 1.

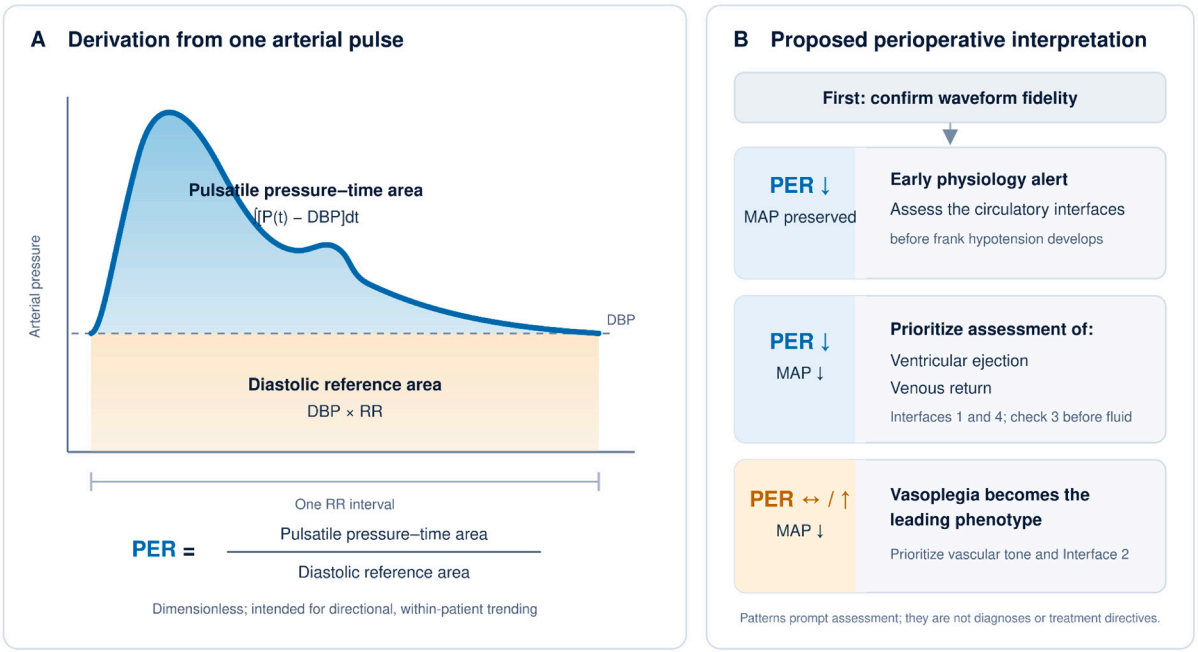

Figure 1. Derivation and hypothesized perioperative interpretation of the Pulse Energy Ratio. PER represents the integrated arterial pressure–time area above diastolic blood pressure, normalized to the diastolic pressure–time reference area over the same cardiac cycle. A decreasing PER may prompt assessment of ventricular ejection, loading conditions, vascular properties, ventilation, and signal quality. During hypotension, a preserved or increasing PER may prompt consideration of reduced vascular tone while accounting for the DBP denominator effect and other determinants of waveform morphology. The potential occurrence of a PER change before hypotension is hypothetical and has not been clinically demonstrated. These patterns are assessment prompts rather than diagnostic phenotypes or treatment directives. Abbreviations: DBP, diastolic blood pressure; MAP, mean arterial pressure; PER, Pulse Energy Ratio; RV–PA, right ventricular–pulmonary arterial.

4. Illustrative Clinical Scenarios

The following hypothetical scenarios illustrate how changes in PER might prompt focused physiological assessment and be followed during mechanism-directed treatment. They are not treatment algorithms. PER should be interpreted alongside its component pressure variables, the arterial waveform, clinical context, and assessment of the circulatory interfaces.

4.1. Hypothetical Compensated Decrease in PER

During major abdominal surgery, a patient has an arterial pressure of 100/60 mmHg, a time-integrated MAP of 75 mmHg, a pulse pressure of 40 mmHg, a PER of 0.25, and an LVOT-VTI of 20 cm. Over several minutes, the arterial pressure changes to 90/65 mmHg. Although integrated MAP remains 75 mmHg, pulse pressure narrows to 25 mmHg, PER decreases to 0.15, and LVOT-VTI decreases to 14 cm. After waveform fidelity is confirmed [10,11], this pattern might prompt physiological assessment rather than reassurance based on the preserved MAP alone.

Changes in vascular resistance may preserve arterial pressure despite a reduction in cardiac output, and MAP should not be interpreted as a direct surrogate for flow [5]. Assessment of Interfaces 1, 3, and 4 might examine ventricular filling and contractility, venous return, right ventricular function, and venous congestion [9]. Serial LVOT-VTI can provide an independent estimate of directional changes in stroke volume when the LVOT cross-sectional area remains unchanged [15]. An underfilled left ventricle with preserved contractility and no significant venous congestion might support reduced filling, whereas

adequate filling with impaired systolic function might suggest decreased contractility. PER alone cannot distinguish these mechanisms.

In this hypothetical sequence, the disturbance subsequently progresses to an arterial pressure of 80/55 mmHg, integrated MAP of 62 mmHg, PER of 0.13, and LVOT-VTI of 10 cm. If mechanism-directed treatment improves ventricular filling or ejection, LVOT-VTI and PER might move toward their previous values. Any apparent response would still require interpretation alongside DBP, MAP, pulse pressure, perfusion, and the underlying waveform. Figure 2 illustrates this proposed sequence.

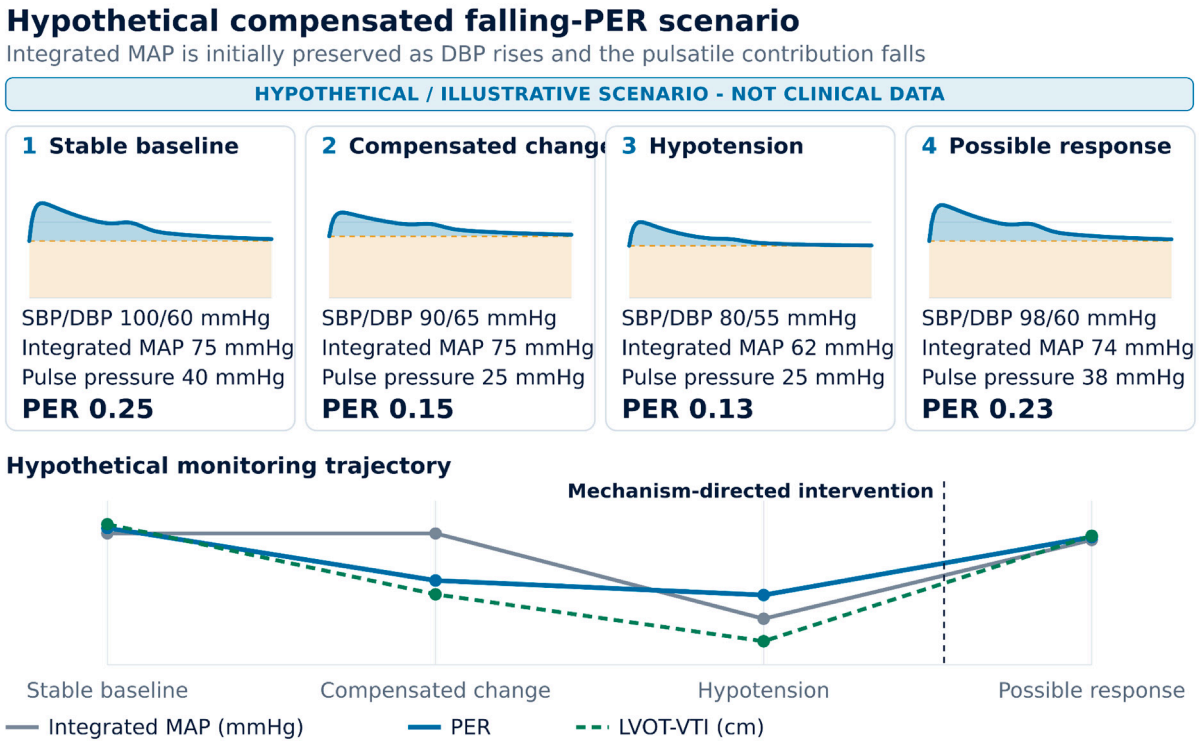

Figure 2. Hypothetical compensated decrease in Pulse Energy Ratio despite initially preserved mean arterial pressure. Arterial pressure changes from 100/60 to 90/65 mmHg. The increase in DBP initially preserves time-integrated MAP at 75 mmHg, while pulse pressure, PER, and LVOT-VTI decrease. The scenario then illustrates subsequent hypotension and a possible response to mechanism-directed treatment. The values and trajectories are illustrative and are not clinical data. PER and time-integrated MAP are mathematically coupled, and pulse pressure is displayed for comparison. Abbreviations: DBP, diastolic blood pressure; LVOT-VTI, left ventricular outflow tract velocity-time integral; MAP, mean arterial pressure; PER, Pulse Energy Ratio; PP, pulse pressure; SBP, systolic blood pressure.

4.2. Hypothetical Increase in PER During Vasodilatory Hypotension

Following induction of general anesthesia, arterial pressure begins to decrease because of a disproportionate fall in DBP. PER remains preserved or increases as the diastolic reference decreases relative to the integrated pulsatile pressure–time area. This pattern might prompt consideration of reduced vascular tone while avoiding the assumption that PER demonstrates preserved stroke volume or confirms vasoplegia.

In this hypothetical scenario, focused assessment shows preserved ventricular contractility and LVOT-VTI without major blood loss, dynamic outflow obstruction, right ventricular dysfunction, or a marked reduction in venous return. Anesthetic-induced vasodilation would therefore represent one possible explanation. This mechanism is consistent with a prospective perioperative cohort in which hypotension after induction with

sufentanil, propofol, and rocuronium was predominantly associated with reduced systemic vascular resistance while stroke volume index and cardiac index remained relatively stable [14]. That study did not evaluate PER.

If vasopressor therapy restores vascular tone and DBP without impairing ventricular ejection, MAP might increase, and the elevated PER might decrease toward its previous value. The observed response could differ if the intervention also changes stroke volume, ventricular afterload, arterial compliance, or waveform morphology. A similar pattern arising later in the perioperative course might prompt consideration of an inflammatory or septic vasodilatory state, guided by the clinical context and assessment of Interface 2. Figure 3 illustrates this proposed sequence.

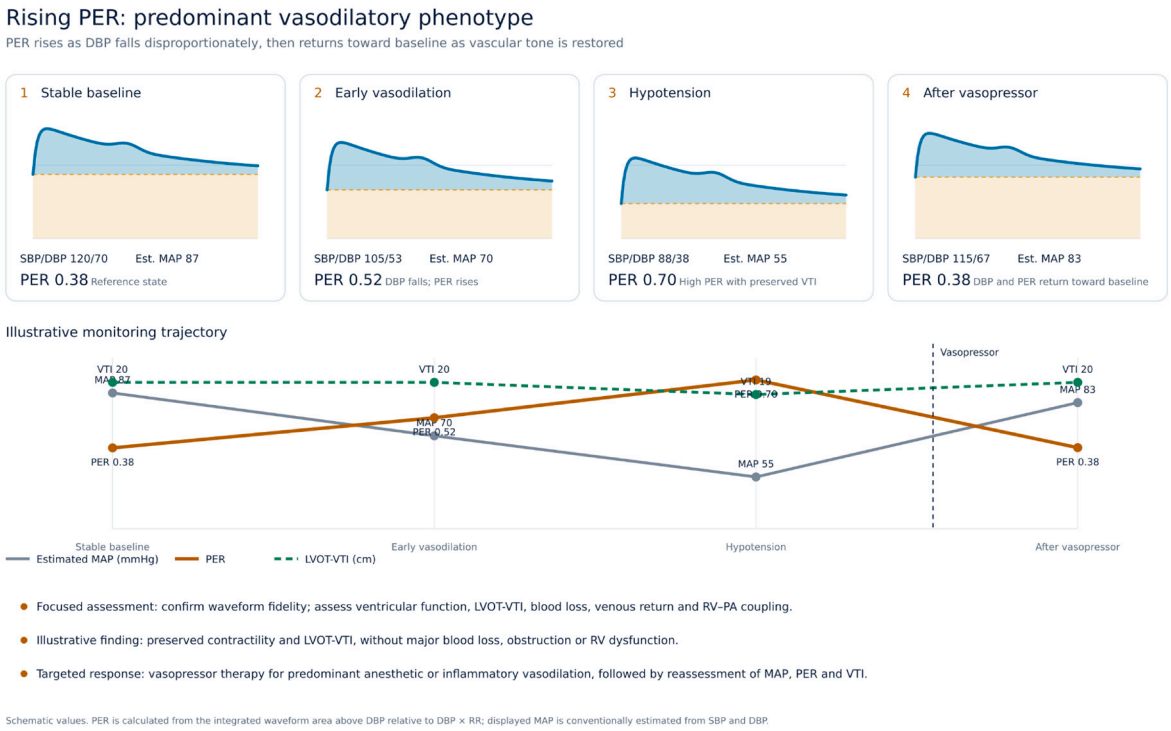

Figure 3. Hypothetical Pulse Energy Ratio trajectory during vasodilatory hypotension. A disproportionate decrease in DBP is accompanied by a decrease in MAP and an increase in PER while LVOT-VTI remains relatively preserved. If reduced vascular tone is the principal mechanism, restoration of DBP with vasopressor therapy might be accompanied by movement of PER toward its preceding value. The values and trajectories are illustrative and are not clinical data. Abbreviations: DBP, diastolic blood pressure; LVOT-VTI, left ventricular outflow tract velocity-time integral; MAP, mean arterial pressure; PER, Pulse Energy Ratio; SBP, systolic blood pressure.

4.3. PER as a Treatment-Response Variable

These scenarios illustrate that movement of PER toward a previous baseline would not necessarily occur in the same direction after every intervention. Improvement in ventricular filling or ejection might be accompanied by an increase in PER. Restoration of DBP during treatment of predominant vasodilation might instead be accompanied by a decrease in PER. Simultaneous changes in vascular tone, ventricular afterload, heart rate, arterial compliance, and waveform morphology may alter either response.

PER should not serve as a therapeutic target or independently confirm treatment success. Its trajectory may provide corroborative information when interpreted with MAP, DBP, pulse pressure, independent measures of forward flow, tissue perfusion, and venous congestion. Discordance between these variables should prompt reassessment of the proposed physiological mechanism.

4.4. Influence of Arterial Sampling Site

PER is derived from the locally recorded arterial waveform and may vary with the arterial sampling site. Peripheral wave amplification and site-dependent differences in waveform morphology are particularly relevant during profound hemodynamic instability, cardiac surgery, substantial vasopressor support, and right ventricular dysfunction. Under these conditions, radial pressure may underestimate femoral or central arterial pressure, altering both the measured pressure and the waveform area used to calculate PER. Radial-to-femoral gradients have been reported in approximately one-third of cardiac surgical patients [16], and marked discordance has also been described during severe perioperative right ventricular dysfunction [17]. PER should therefore be trended from a consistent arterial site. An unexpected value or abrupt change should prompt reassessment of waveform quality, comparison with non-invasive pressure when appropriate, and consideration of a more central arterial measurement. Periodic comparison of the invasive radial arterial pressure with an intermittent non-invasive brachial pressure measurement may provide a practical means of detecting a present or evolving central-to-peripheral arterial pressure gradient. When a clinically important discrepancy is suspected, confirmation using a more proximal invasive site, such as the brachial, axillary, or femoral artery, may be appropriate. In patients with shock, particularly those with right ventricular dysfunction or substantial vasopressor requirements, a femoral arterial waveform may provide a more representative basis for interpretation.

5. Limitations and Future Validation

PER has a fundamental mathematical limitation. When MAP is obtained by time-averaging the same arterial waveform, PER is determined entirely by integrated MAP and DBP and contains no mathematically independent information beyond those variables. When MAP is instead estimated from SBP and DBP using a fixed formula, differences between PER and calculated MAP may reflect waveform morphology and the limitations of the estimation formula. Whether expressing this relationship as a normalized and continuously trendable ratio provides useful information beyond MAP, DBP, pulse pressure, or other simple pressure-derived ratios is unknown.

PER is also sensitive to its DBP denominator. A disproportionate decrease in DBP may increase PER without an increase in the absolute pulsatile pressure–time area, while an increase in DBP may reduce PER despite preserved integrated MAP. Measurement may additionally be affected by arterial compliance, wave reflection, peripheral runoff, heart rate, rhythm, damping or resonance, transducer levelling and zeroing, signal filtering, sampling frequency, monitoring system, vasoactive therapy, and arterial sampling site [5–7,10,11]. Radial–femoral pressure gradients may be particularly relevant during profound hemodynamic instability, cardiac surgery, substantial vasopressor support, and right ventricular dysfunction [16,17].

Initial studies should establish within-patient and between-device reproducibility and define acceptable waveform-quality and averaging criteria. PER should then be compared with independent measures of stroke volume, cardiac output, ventricular function, and vascular tone. Direct comparison with MAP, DBP, SBP, pulse pressure, and simple pressure-derived ratios will be necessary to determine whether PER provides incremental discriminatory, temporal, or predictive value. Its ability to track responses to hemodynamic interventions should also be evaluated before it is considered for clinical use.

Finally, a clinically relevant central–peripheral arterial pressure gradient may also influence PER when derived from a peripheral arterial waveform; comparison with intermittent brachial cuff pressure should therefore be incorporated into future validation studies.

6. Conclusions

PER is a simple, dimensionless representation of the relationship between the integrated pulsatile pressure–time area and its diastolic pressure reference. Its potential value lies not in providing mathematically independent information from integrated MAP and DBP, but in making this relationship explicit and continuously trendable from the arterial waveform. Within the four-interface framework, changes in PER may serve as prompts for structured physiological reassessment rather than as diagnostic phenotypes or treatment targets. Whether PER provides clinically useful information beyond conventional pressure variables and simple pressure-derived ratios remains to be established through prospective validation.

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References

1. Saugel, B.; Fletcher, N.; Gan, T.J.; Grocott, M.P.; Myles, P.S.; Sessler, D.I.; Auzinger, G.; Chappell, D.; Edwards, M.; Forni, L.G.; et al. PeriOperative Quality Initiative international consensus statement on perioperative arterial pressure management. *Br. J. Anaesth.* **2024**, *133*, 264–276. [CrossRef] [PubMed]
2. Wesselink, E.M.; Kappen, T.H.; Torn, H.M.; Slooter, A.J.C.; van Klei, W.A. Intraoperative hypotension and the risk of postoperative adverse outcomes: A systematic review. *Br. J. Anaesth.* **2018**, *121*, 706–721. [CrossRef] [PubMed]
3. Kouz, K.; Bergholz, A.; Timmermann, L.M.; Brockmann, L.; Flick, M.; Hoppe, P.; Briesenick, L.; Schulte-Uentrop, L.; Krause, L.; Maheshwari, K.; et al. The relation between mean arterial pressure and cardiac index in major abdominal surgery patients: A prospective observational cohort study. *Anesth. Analg.* **2022**, *134*, 322–329. [CrossRef] [PubMed]
4. Kouz, K.; Brockmann, L.; Timmermann, L.M.; Bergholz, A.; Flick, M.; Maheshwari, K.; Sessler, D.I.; Krause, L.; Saugel, B. Endotypes of intraoperative hypotension during major abdominal surgery: A retrospective machine learning analysis of an observational cohort study. *Br. J. Anaesth.* **2023**, *130*, 253–261. [CrossRef] [PubMed]
5. Magder, S. The meaning of blood pressure. *Crit. Care* **2018**, *22*, 257. [CrossRef] [PubMed]
6. Westerhof, N.; Lankhaar, J.W.; Westerhof, B.E. The arterial Windkessel. *Med. Biol. Eng. Comput.* **2009**, *47*, 131–141. [CrossRef] [PubMed]
7. Saugel, B.; Kouz, K.; Scheeren, T.W.L.; Greiwe, G.; Hoppe, P.; Romagnoli, S.; de Backer, D. Cardiac output estimation using pulse wave analysis—Physiology, algorithms, and technologies: A narrative review. *Br. J. Anaesth.* **2021**, *126*, 67–76. [CrossRef] [PubMed]
8. Rola, P.; Bhardwaj, V.; Peddi, K.Y.; Haycock, K.; Miller, A.; Spiegel, R.; Kenny, J.-E. The Pulse Energy Ratio: Validation of a Novel Hemodynamic Parameter. *Preprints* **2026**, 2026041985. [CrossRef]
9. Rola, P.; Kattan, E.; Siuba, M.T.; Haycock, K.; Crager, S.; Spiegel, R.; Hockstein, M.; Bhardwaj, V.; Miller, A.; Kenny, J.-E.; et al. Point of view: A holistic four-interface conceptual model for personalizing shock resuscitation. *J. Pers. Med.* **2025**, *15*, 207. [CrossRef] [PubMed]
10. Esper, S.A.; Pinsky, M.R. Arterial waveform analysis. *Best Pract. Res. Clin. Anaesthesiol.* **2014**, *28*, 363–380. [CrossRef] [PubMed]
11. Saugel, B.; Kouz, K.; Meidert, A.S.; Schulte-Uentrop, L.; Romagnoli, S. How to measure blood pressure using an arterial catheter: A systematic 5-step approach. *Crit. Care* **2020**, *24*, 172. [CrossRef] [PubMed]
12. Hatib, F.; Jian, Z.; Buddi, S.; Lee, C.; Settels, J.; Sibert, K.; Rinehart, J.; Cannesson, M. Machine-learning algorithm to predict hypotension based on high-fidelity arterial pressure waveform analysis. *Anesthesiology* **2018**, *129*, 663–674. [CrossRef] [PubMed]

13. Wijnberge, M.; Geerts, B.F.; Hol, L.; Lemmers, N.; Mulder, M.P.; Berge, P.; Schenk, J.; Terwindt, L.E.; Hollmann, M.W.; Vlaar, A.P.; et al. Effect of a machine learning-derived early warning system for intraoperative hypotension versus standard care on depth and duration of intraoperative hypotension during elective noncardiac surgery: The HYPE randomized clinical trial. *JAMA* **2020**, *323*, 1052–1060. [CrossRef] [PubMed]
14. Saugel, B.; Bebert, E.J.; Briesenick, L.; Hoppe, P.; Greiwe, G.; Yang, D.; Ma, C.; Mascha, E.J.; Sessler, D.I.; Rogge, D.E. Mechanisms contributing to hypotension after anesthetic induction with sufentanil, propofol, and rocuronium: A prospective observational study. *J. Clin. Monit. Comput.* **2022**, *36*, 341–347. [CrossRef] [PubMed]
15. Blanco, P. Rationale for using the velocity-time integral and the minute distance for assessing the stroke volume and cardiac output in point-of-care settings. *Ultrasound J.* **2020**, *12*, 21. [CrossRef] [PubMed]
16. Bouchard-Dechêne, V.; Kontar, L.; Couture, P.; Pérusse, P.; Levesque, S.; Lamarche, Y.; Denault, A.Y.; Rochon, A.; Deschamps, A.; Desjardins, G.; et al. Radial-to-femoral pressure gradient quantification in cardiac surgery. *JTCVS Open* **2021**, *8*, 446–460. [CrossRef] [PubMed]
17. Denault, A.Y.; Haddad, F.; Jacobsohn, E.; Deschamps, A. Perioperative right ventricular dysfunction. *Curr. Opin. Anaesthesiol.* **2013**, *26*, 71–81. [CrossRef] [PubMed]

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