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Case Report
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Multimodal Non-Invasive Perfusion Monitoring at the Bedside

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Abstract

Background: *The assessment of perfusion is a cornerstone in the management of critically ill patients. Over the last decade, hemodynamic monitoring has evolved toward less invasive modalities, supported by a growing understanding of vascular–tissue coupling and the availability of bedside technologies.*

Methods: *We present a case of acute coronary syndrome in which multimodal non-invasive hemodynamic monitoring was applied using a structured evaluation based on the interface model. Sequential assessments included capillary refill time (CRT), perfusion index, arterial line, near-infrared spectroscopy (NIRS), carotid VTI (VTIc), cardiac ultrasound (VTI LVOT), lactate and ΔCO_2,*

Results: *The integration of these complementary technologies provided a dynamic overview of the patient's perfusion status and guided therapeutic decisions, including the titration of vasopressors and discontinuation of inotropes. CRT and perfusion index were the earliest parameters to normalize, while lactate kinetics lagged behind other perfusion markers. NIRS and VTIc revealed preserved cerebral autoregulation despite peripheral hypoperfusion, highlighting the dissociation between systemic and local signals. The interface-based approach facilitated recognition of hemodynamic coherence and individualized treatment at the bedside.*

Conclusion: *Multimodal non-invasive perfusion monitoring enables a tissue-centered interpretation of hemodynamic states beyond traditional macrovariables. By integrating minimally invasive technologies through the interface model, clinicians can better identify perfusion conflicts, refine diagnostic precision, and guide personalized therapy. This case illustrates how real-time, physiology-driven monitoring supports a shift toward individualized critical care rather than protocolized interventions.*

Keywords: Hemodynamic Monitoring, Perfusion, Critical care, Non-Invasive Monitoring, Near-Infrared Spectroscopy, Perfusion Index, Point-of-Care Ultrasound, Interface Mode.

Introduction

The management of any acutely ill patient should include some form of perfusion monitoring, which has undergone substantial evolution over the past decade driven by a deeper understanding of the hemodynamic behavior of the critically ill patient and by the emergence of novel techniques and technology that enable non-invasive bedside evaluation in the intensive care unit (ICU), particularly point of care ultrasound We present a case of a patient with an acute coronary syndrome in which the application of multiple minimally invasive technologies allowed for precise characterization of the hemodynamic phenotype through a systematic evaluation based on the interface model.

Case Presentation

A 70-year-old man with a past medical history of psoriatic arthritis, osteoporosis, dyslipidemia, non–insulin-dependent diabetes mellitus, arterial hypertension, active smoking (40 pack-years), and coronary artery disease with a prior percutaneous coronary intervention in 2015 arrived to the emergency room by his own means.

He presented with progressive dyspnea and retrosternal chest pain of anginal characteristics, without additional accompanying symptoms. On initial physical examination: blood pressure 115/76 mmHg, heart rate 83 bpm, SpO_2 97% on room air, with no abnormal cardiac or pulmonary findings.

Complementary studies revealed:

- ECG: ST-segment elevation in aVR with diffuse ST depression in other leads.
- Chest X-ray: No acute pathology
- Troponin: baseline 67 ng/L, follow-up 80 ng/L, peak 603 ng/L.
- Serum creatinine: 0.83 mg/dL.
- Hemoglobin: 14.2 g/dL.
- Arterial blood gas: pH 7.37; HCO_3^- 26 mmol/L.
- Pro-BNP: 2541 pg/mL.

Given the findings consistent with acute coronary syndrome, the patient was promptly transferred to an affiliated institution due to the unavailability of an angiography unit at our center, and a coronary angiography was performed, revealing multivessel disease with 70% stenosis in the left anterior descending and right coronary arteries, and 95% in the circumflex artery.

During and after the procedure, he required norepinephrine infusion up to 0.1 µg/kg/min due to hypotension.

The patient was admitted to the ICU while awaiting surgical revascularization, still requiring vasopressor support. Clinically, he exhibited signs of impaired perfusion characterized by delayed capillary refill and mottling around the knees.

A systematic evaluation using the interface model was performed for a more precise characterization:

- Interface 1: VTI 11 cm. EF 20%.
- Interface 2: Prolonged capillary refill (finger 4 s, knee 5 s).
- Interface 3: VExUS 0.
- Interface 4: TAPSE >17 mm.

Given the suspicion of excessive afterload induced by vasopressor therapy, norepinephrine was progressively titrated down and eventually discontinued. In parallel, he received a 250 ml of 3% saline as a bolus. However, the uncoupling between interfaces 1 and 2 persisted, as indicated by persistent signs of left ventricular dysfunction associated with lower extremity mottling and increased capillary refill (Table 1, Baseline).

A multimodal hemodynamic assessment was then conducted, including continuous multiparametric monitoring with arterial line and perfusion index, near-infrared spectroscopy (NIRS), carotid VTI (VTIc) using a portable ultrasound device (Fig. 1); bedside cardiac ultrasound (VTI LVOT), serial lactate and ΔCO_2 measurements.

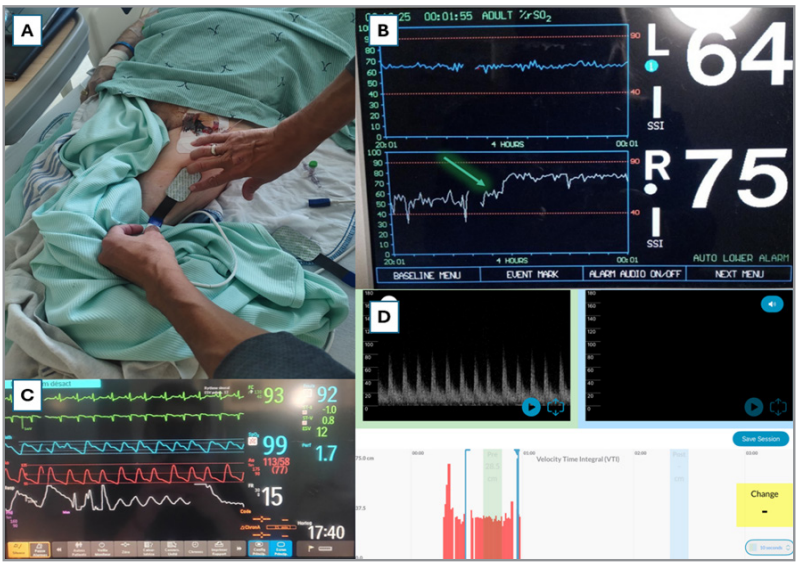

Figure 1: Multimodal Hemodynamic Monitoring

A: Application of the cutaneous NIRS sensor (leg).
B: NIRS monitor with cerebral probe (upper trace) and cutaneous probe (lower trace). Note the quantitative increase in the cutaneous signal following the initiation of dobutamine (green arrow).
C: Multiparameter monitor display including arterial line and perfusion index.
D: Carotid artery velocity time integral (VTIcarotid) display obtained using a FloPatch device.

Formal transthoracic echocardiography revealed anteroseptal and apical hypokinesia, inferior akinesia, a left ventricular ejection fraction of 40%, normal right ventricular function, and no significant valvular abnormalities. During the ICU stay, the patient did not experience recurrence of chest pain, arrhythmias, or dynamic ECG changes. Table 1 summarizes the values obtained from the multimodal hemodynamic monitoring through the follow up.

Table 1: Serial Trends of the Evaluated Hemodynamic and Perfusion parameters

Temporal phase	Capillary refill (s)	Perfusion Index	Arterial Line (mmHg)	Cerebral NIRS (%)	Leg NIRS (%)	VTI carotid (cm)	VTI LVOT (cm)	Lactate / ΔCO_2
Baseline	4	0,8	120/60	66	47	25	12	1,9 / 15
Dobutamine 5 µg/kg/min (start)	2	3,8	115/60	64	53	34	13	1,8 / 13
Dobutamine 5 µg/kg/min (2 h)	2	4,9	136/72	64	75	28	13	1,8 / 7
Dobutamine 5 µg/kg/min (12 h)	2	4,0	144/86	—	70	30	14	—
Dobutamine 2.5 µg/kg/min	2	5,5	110/54	—	66	33	15	—
Dobutamine 1 µg/kg/min	2	5,7	113/58	—	65	17	14	—
Dobutamine discontinued	2	4,7	114/64	—	67	22	14	—

With progressive clinical improvement and stabilization of parameters obtained through multimodal non-invasive hemodynamic monitoring, dobutamine dose was gradually reduced and eventually discontinued. Figure 2 shows the monitor values at the time of discontinuation.

Figure 2: Multiparameter Monitor at the Time of Dobutamine Discontinuation.

Discussion

Hemodynamic monitoring remains one of the pillars of critical care management and represents a dynamic concept that has undergone important iterations over the past decade [1]. Multimodal monitoring—incorporating non-invasive and minimally invasive devices—has emerged as a valid option for characterizing hemodynamic parameters that guide tissue perfusion in critically ill patients and tracking response to therapy [2].

Interface Model and a Tissue-Centered Approach

The interface-based approach enables systematic communication of vascular–tissue coupling, identifying perfusion conflict points and guiding targeted therapeutic interventions (Fig. 3).

Furthermore, integrating this model represents a conceptual step forward—moving away from a cardio-centric perspective toward a tissue-centered paradigm of hemodynamic assessment and optimization [3].

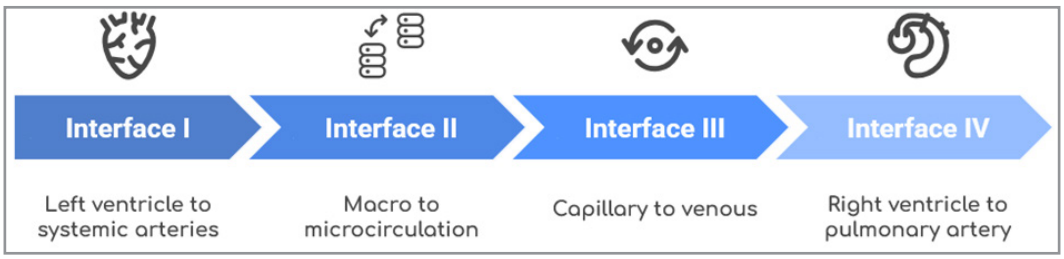

Figure 3: Coupling Interfaces.

Capillary Refill Time

Capillary refill time (CRT) has demonstrated a strong correlation with lactate levels in septic shock, supported by robust physiological foundations as a marker of tissue hypoperfusion, and represents the most accessible manoeuvre to assess the second hemodynamic interface (macro to microcirculation). It is reproducible, easy to perform, and has low interobserver variability [4, 5]. In this case, the initially prolonged CRT suggested suboptimal tissue perfusion despite reasonable macrohemodynamic parameters, and likely resulted from interface 1 uncoupling as a consequence of ischemic cardiomyopathy. Moreover, CRT was the earliest marker to normalize and mirrored the patient's hemodynamic trajectory throughout recovery.

Perfusion Index

The perfusion index is derived from the pulsatile and non-pulsa-

tile components of the plethysmographic waveform and is available in most modern monitors as a continuous ancillary indicator within the pulse oximetry module. Changes in vascular tone and congestion inversely affect its magnitude (i.e., increased vascular tone, and/or increased congestion, reduces its value), making it useful for assessing local perfusion trends. Although consistent cutoff values have not been firmly established, downward trends are associated with worse prognosis. In this patient, and in the absence of confounding factors (ambient temperature, analgesic control, limb compression, or signal loss), the perfusion index closely followed the trend of other dynamic parameters during the clinical course, serving as an early indicator of improvement in the hemodynamic profile [6-8].

Arterial Line Monitoring

An arterial line allows continuous visualization of key macrohemodynamic variables such as systolic pressure (reflecting left ventricular afterload), diastolic pressure (reflecting vascular tone), and mean arterial pressure (determinant of tissue perfusion). It also provides an estimate of stroke volume via pulse pressure analysis. The insertion site selection is relevant: more central sites (i.e., closer to the aorta) yield more accurate hemodynamic data in patients with perfusion mismatch. In this case, femoral access was chosen due to obesity, previous radial access during coronary angiography, and local difficulty obtaining a reliable signal [9-11].

Near-Infrared Spectroscopy (NIRS)

NIRS operates through the emission of near-infrared light (680–800 nm) across tissues where sensors are placed, assessing the absorption patterns of chromophores such as hemoglobin predominantly in small vessels (<1 mm), thus reflecting mainly the venous component.

Consequently, NIRS provides a continuous measure of the balance between oxygen delivery and consumption at the tissue level. Although most data derive from thenar-site measurements using vascular occlusion tests in septic shock patients, downward or upward trends in both magnitude and rate of change are associated with poorer and better outcomes, respectively[12-14]. In our case, differential probe positioning demonstrated stable cerebral (frontal) values despite pronounced peripheral (cutaneous) changes. Therefore, subsequent cerebral NIRS monitoring was deemed unnecessary to assess hemodynamic coherence. The prognostic extrapolation of spontaneous recovery rates and cerebral–cutaneous configuration may warrant further investigation.

Carotid artery velocity time integral (VTIcarotid)

The VTIcarotid is the distance blood travels per cardiac cycle up a single carotid artery. It can be continuously measured by a wireless, wearable Doppler ultrasound device. The relationship between SV and the VTIcarotid is direct, but also determined by the flow fraction of blood to the head (i.e., internal and external carotid arteries, combined) relative to the body, as described in a recent theoretical framework [15-17].

In this way, VTIcarotid acts as a measure of LVOT VTI to systemic arterial coupling (i.e., interface 1). In the present case, similar to the frontal and peripheral NIRS measurements, the VTIcarotid suggested flow distribution away from the head towards the periphery with dobutamine infusion. The initial significant rise in VTIcarotid with dobutamine and then general downtrend while on the same infusion dose could have been caused by intracerebral vasoconstriction and/or arterial vasodilation of the systemic arterioles (e.g., in the skin, muscles or splanchnic vasculature). This observation matched the fall in the decrease in the saturation ratio of head-to-lower extremity.

Left Ventricular Velocity-Time Integral (VTI)

Measurement of the velocity-time integral (VTI) from a five-chamber cardiac window using bedside ultrasound (POCUS) has gained traction as a practical and reproducible diagnostic and therapeutic parameter for shock management. It assumes a constant left ventricular outflow tract diameter, allowing estimation of stroke volume by calculating the distance blood travels during systole via pulsed Doppler [18–20]. While threshold values have been proposed to define adequacy for specific shock states and to profile shock subtypes, the authors interpret it as a dynamic trend marker, comparable to other non-invasive tools, rather than a rigid target. Through its correlation with stroke volume, the VTI allows for interface 1 (left ventricle to systemic arteries) assessment and monitoring during clinical interventions [21, 22].

In this case, serial VTI assessments allowed for periodic interface 1 assessment, and demonstrated an ascending trend initially attributed to dobutamine's inotropic effect and subsequently to intrinsic myocardial recovery during its tapering phase.

Lactate

While lactate is commonly used as a marker of tissue hypoperfusion in acutely ill patients, it is in hemorrhagic, hypovolemic and cardiogenic shock where this applies, reflecting an imbalance between oxygen delivery (DO_2) and consumption (VO_2), and correlates closely with prognosis when elevated. In septic shock, most of the elevation in lactate is due to inflammatory cytokines and not tissue hypoperfusion. Sequential measurement enhances its prognostic accuracy and helps guide therapeutic interventions in the right pathologies. Recent critical care literature emphasizes the nuanced interpretation of lactate due to multiple underlying mechanisms (type A vs. type B hyperlactatemia) and its bioenergetic implications. Since perfusion conflict was revealed by other dynamic parameters, and in line with the current trend of not targeting a specific lactate clearance in isolation, further early measurements were deemed unnecessary. The authors interpret that the multimodal monitoring approach facilitated early detection of hypoperfusion, acknowledging that lactate kinetics may lag behind real-time perfusion changes [23-27].

Venous–Arterial CO_2 Difference (ΔCO_2)

The venous–arterial carbon dioxide difference (ΔCO_2) is inversely related to cardiac output, as reduced flow prolongs capillary transit time and CO_2 diffusion, assuming stable CO_2 production, temperature, and hemoglobin levels [28, 29].

Values >6 mmHg are consistent with tissue blood flow impairment. Although most evidence originates from septic shock, its prognostic relevance extends to other shock types.

In this case, the elevated initial ΔCO_2 indicated significant tissue-level repercussions of predominantly cardiogenic shock,

and along with CRT and perfusion index, was among the earliest markers reflecting hemodynamic coherence in the multimodal framework [30-32].

Conclusion

As the care of acutely ill patients slowly but surely moves towards personalized approaches at the bedside, the integration of minimally invasive technologies complementing conventional techniques may serve as a valuable strategy to identify the hemodynamic phenotype of these patients in order to provide the appropriate therapeutic interventions. We hope to see more research focusing on precision care guided by individual physiology, as opposed to "one-size-fits-all" protocols. A multimodal, tissue-centered approach enables recognition of hemodynamic disturbances not necessarily revealed by looking at vital signs alone and prompt individualized management at the bedside.

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