

Calibration-Free Method for Cardiac Output and Vascular Profiling via Windkessel α -Parameterization

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Aims: Conventional pulse-contour analysis estimates cardiac output (CO) from arterial pressure waveforms but relies on empirical or calibrated mappings, reducing waveform morphology to a scalar estimate with limited physiological interpretability. We propose a framework based on a structurally identifiable reformulation of the Windkessel model. Four α -parameters are derived, each encoding a distinct morphological feature of the radial pressure waveform, enabling calibration-free CO estimation and simultaneous vascular parameter quantification.

Methods: A four-element Windkessel model of the arterial circulation was reformulated to yield four structurally identifiable parameters: α_c (compliance pulsatility), α_r (systolic pressure load), α_L (early-systolic inertial rise rate), and α_τ (diastolic time constant). These were extracted by fitting preprocessed radial arterial pressure (pABP) waveforms to the reformulated model and, combined with biometric covariates, used as inputs to a generalized linear model (GLM) for CO estimation, trained on 3,077 waveforms from 380 patients (VitalDB; EV1000/FloTrac comparator) and validated on 510 Vigileo waveforms without retraining.

Results: CO estimation yielded $R^2 = 0.82$, bias = $-0.02 \text{ L} \cdot \text{min}^{-1}$, and percentage error $PE = 26\%$, satisfying the interchangeability criterion ($PE < 30\%$); external validation on Vigileo without retraining gave $R^2 = 0.72$ and $PE = 28\%$. Arterial compliance (C), impedance (R_z), resistance (R_{dis}), and inductance (L) were simultaneously recovered ($R^2 = 0.82\text{--}0.93$).

Patient-specific α -profiles revealed mechanistically distinct phenotypes: stiffness-dominated loading (elevated α_c , α_r) in chronic kidney disease, and a reservoir-driven pattern (prolonged α_τ , reduced α_L) in elderly isolated systolic hypertension.

Conclusion: Our method transforms peripheral arterial pressure waveforms into mechanistically interpretable hemodynamic descriptors (α -parameters). Beyond calibration-free CO estimation, it simultaneously quantifies C , R_z , R_{dis} , and L , while patient-specific α -profiles characterize the vascular mechanisms shaping waveform morphology. This provides a mechanistic complement to scalar CO monitoring for hemodynamic assessment in perioperative and critical care settings (Taheri & Haut, Ann. Biomed. Eng., 2026).