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Assessment of Aortic Artery Distensibility and Elasticity Indices in Respiratory Care Unit Patients on Mechanical Ventilation: A Prospective Case-Control Study

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Abstract: Background: Patients receiving invasive mechanical ventilation are subject to extreme changes in the cardiopulmonary physiology that may adversely affect the mechanical properties of the aortic wall. Limited literature exists concerning the elastic properties of the aorta in patients treated in the respiratory care unit (RCU).**Objective:** To assess the aortic properties of a mix of distensibility, strain, stiffness index (β), and Peterson's elastic modulus (Ep) using transthoracic echocardiography (TTE) between mechanically ventilated patients in the RCU with appropriate healthy controls.**Methods:** Prospective case-control study at Mirjan Medical City, Babylon, Iraq (November 2025–April 2026). Fifty subjects: 25 mechanically ventilated RCU patients and 25 age- and sex-matched healthy controls. Aortic dimensions were acquired by M-mode TTE in the parasternal long-axis view at 3 cm above the aortic valve, and four elastic indices were calculated.**Results:** Aortic dimensions were comparable between groups ($p > 0.05$). Aortic strain was significantly reduced (4.27 ± 1.62% vs. 6.94 ± 1.77%; $p < 0.001$), stiffness index β was markedly elevated (12.86 ± 9.04 vs. 4.67 ± 1.48; $p < 0.001$), and Peterson's Ep was significantly higher (801 ± 298 vs. 456 ± 118 mmHg; $p < 0.001$) in the MV group. Aortic distensibility showed a consistent downward trend (1.82 ± 0.88 vs. $2.28 \pm 1.12 \times 10^{-6}$ cm²/dyn; $p = 0.087$). Left ventricular ejection fraction was preserved in all MV patients (62.0 ± 3.6%).**Conclusion:** Mechanical ventilation is associated with significant functional aortic stiffness, independent of structural remodelling or systolic dysfunction. Echocardiographic aortic elastic assessment is bedside-feasible and should be considered in RCU haemodynamic monitoring protocols.

Keywords: Aortic distensibility; aortic stiffness; mechanical ventilation; respiratory care unit; echocardiography; critical illness; elasticity index

INTRODUCTION

Managing the critically ill pt. under invasive mechanical ventilation provides one of the greatest challenges in modern intensive care medicine. The complex hemodynamic effects of positive-pressure ventilation with elevated intrathoracic (chest) pressures, loss of venous return, and altered preload and afterload on the heart, superimpose to compromise already compromised cardiovascular systems in critical illness [1,2]. Potentially useful indicators of cardiovascular homeostasis arise from the mechanical properties of the elastic structures (ascending aortic wall) as they relate to the "Windkessel" effect (the combined capacity of an elastic vascular structure to expand during the contraction (squeeze out) of the heart, absorb the

pulsatile energy flow, and then release that energy into the coronary arteries during the following diastole to continue perfusing the heart and to assist in supply of blood to the microvasculature of the rest of the body), which depends on the viscoelastic properties of the wall of the aorta, composed of concentric "lamellar" units of elastin, interspersed with smooth muscle cells and an elastin-rich extracellular matrix (ECM) [3,4].

Reduced aortic elasticity and increased aortic stiffness are well-established independent risk factors (predictors) of poor cardiovascular outcomes from an epidemiological perspective. In a systematic review and meta-analysis, Vlachopoulos and coworkers showed that individuals with increased cf-PWV (carotid-femoral pulse wave velocity), independent of traditional risk factors, are at significantly elevated risk of suffering subsequent cardiovascular events or dying from any cause[5]. According to the 2023 European Society of Hypertension guidelines, a cf-PWV>10 m/s has been validated as a marker of significant arterial stiffness and thus a high operative risk of cardiovascular disease [6]. Authors identified three physiological pathways by which increased aortic stiffness leads to multi-organ injury: increased central pulse pressure and left ventricular afterload; reduced diastolic coronary perfusion due to reduced Windkessel recoil; and excessive pulsatile stress applied to end-organ microvasculature [7]. It has been reported that increased aortic stiffness is independently associated with increased risk for the development of multimorbidity (including hypertension, ischemic heart disease, heart failure, chronic kidney disease, and cognitive decline), particularly in the geriatric population [8].

In the RCU environment, factors governing aortic elastic behavior are substantially more complex. Many authors have reviewed the cardiovascular effects of positive-pressure ventilation, concluding that the net hemodynamic impact — preload reduction, altered ventricular afterload, and biventricular interdependence — is highly individualized and influenced by baseline cardiac reserve, volume status, and ventilator parameters [5,6]. Critically ill patients concurrently exhibit systemic inflammatory activation, endothelial dysfunction, oxidative stress, and vasomotor dysregulation, all of which acutely alter arterial mechanical properties [8,9]. It has been demonstrated that aortic wall strain is significantly reduced in ICU patients and independently associated with adverse outcomes [9]. Despite these observations, the specific effects of mechanical ventilation on echocardiography-derived aortic elastic indices in unselected RCU patients have not been prospectively investigated.

Transesophageal echocardiography (TEE) can be used to perform a fast, non-invasive assessment of aortic properties at the bedside. M-mode echocardiography is performed in the parasternal long-axis view. It enables the calculation of four complementary elastic indices of the aorta (i.e., aortic strain, distensibility coefficient, stiffness index β, and Peterson's elastic modulus) based on the methodology developed by [9], which has been validated in several ambulatory populations [10]. The ongoing expansion of point-of-care echocardiography in the intensive care unit, including [11] validated AI-assisted measurement tools with intraclass correlation coefficients of > 0.90 for the most important hemodynamic indices [11], has made the bedside assessment of aortic elasticity increasingly feasible. Both the American Thoracic Society and the European Society of Intensive Care Medicine have issued consensus statements supporting enhanced cardiovascular monitoring for patients on mechanical ventilation and have recognized a lack of evidence in the current literature on the optimal means of assessing vascular properties [12,13]. This study attempts to bridge that gap.

MATERIALS AND METHODS

Study Design and Setting

A prospective observational case-control study was conducted in the 20-bed RCU of Mirjan Medical City, Babil Health Directorate, Babylon, Iraq, from November 2025 to April 2026. The RCU is equipped with invasive mechanical ventilation, continuous hemodynamic monitoring, and a Philips CX50 portable echocardiography system. The study was approved by the Babylon University Institutional Ethics Committee and conducted in accordance with the Declaration of Helsinki. Informed consent was obtained from patients' legally authorized representatives and directly from healthy volunteers.

Study Population

Fifty subjects were enrolled: 25 mechanically ventilated RCU patients (MV group) and 25 age- and sex-matched healthy volunteers (Normal group). MV group inclusion criteria: age 18–60 years; invasive mechanical ventilation ≥ 48 hours via endotracheal tube or tracheostomy; hemodynamic stability (SBP ≥ 100, DBP ≥ 60 mmHg); normal sinus rhythm; adequate echocardiographic window. Exclusion criteria encompassed: pre-existing aortic pathology; severe hemodynamic instability; pregnancy; advanced chronic kidney disease (CrCl < 30 mL/min/1.73 m² by Cockroft-Gault [18]); connective tissue disease; prior thoracic radiation; or non-invasive ventilation as the primary support modality. Control group criteria: age 18–60 years; absence of known cardiovascular, metabolic, or renal disease; BMI 18.5–30 kg/m²; blood pressure < 140/90 mmHg without medication; and adequate echocardiographic window.

Echocardiographic Assessment and Elastic Index Calculation

All studies were performed using the Philips CX50 with S5-1 phased-array transducer (1–5 MHz). Ventilated patients were examined supine; measurements were taken at end-expiration synchronized with the airway pressure waveform. Three to five cardiac cycles were averaged per measurement. Aortic diameters were obtained by M-mode echocardiography in the parasternal long-axis view at 3 cm above the aortic valve per [9] and ASE guidelines: maximum systolic diameter (Ds) at peak systole and minimum diastolic diameter (Dd) at end-diastole. An automated oscillometric cuff recorded the simultaneous blood pressure. The Mosteller formula calculates body surface area. Standard 2D Doppler assessment evaluated LVEF (biplane Simpson's), diastolic function (E/e' by tissue Doppler), and valvular integrity.

Four elastic indices were calculated: (1) Aortic Strain (AS%) = [(Ds−Dd)/Dd]×100; (2) Aortic Distensibility (AD, $\times 10^{-6}$ cm²/dyn) = 2×(Ds−Dd)/[Dd×(SBP−DBP)×1333]; (3) Stiffness Index β = ln(SBP/DBP)/[(Ds−Dd)/Dd]; (4) Peterson's Elastic Modulus Ep (mmHg) = (SBP−DBP)×Dd/(Ds−Dd) [19,20]. All calculations were performed using a dedicated Excel spreadsheet with built-in formulae.

Statistical Analysis

Data were analyzed using SPSS v26.0 and GraphPad Prism v9.0. Normality was assessed by the Shapiro-Wilk test. Continuous data are expressed as mean ± SD or median (IQR); categorical data as frequency (%). Between-group comparisons used the independent-samples t-test or Mann-Whitney U test as appropriate; chi-square or Fisher's exact test for categorical variables. Pearson's or Spearman's correlation was used to assess relationships between elastic indices and clinical variables. Two-tailed $p < 0.05$ was considered statistically significant.

RESULTS

Demographic and Hemodynamic Characteristics

All 50 enrolled participants yielded adequate echocardiographic windows. The two groups were well matched for age, sex, BMI, and BSA (Table 1). Systolic blood pressure was comparable ($p = 0.583$); however, diastolic blood pressure was significantly lower and pulse pressure significantly higher in the MV group (both $p < 0.05$), consistent with known hemodynamic effects of positive-pressure ventilation. LVEF was preserved in all MV patients (62.0 ± 3.6%, range 56–67%), confirming the absence of systolic myocardial dysfunction.

Table 1. Demographic and hemodynamic characteristics (Mean ± SD).

Parameter	Normal (n=25)	MV (n=25)	p-value
Age (years)	42.1 ± 13.4	43.6 ± 14.7	0.706 NS
Sex (M/F)	12/13	16/9	0.163 NS
BMI (kg/m²)	24.5 ± 2.8	26.3 ± 3.9	0.065 NS
BSA (m²)	1.83 ± 0.14	1.84 ± 0.14	0.841 NS
SBP (mmHg)	117.4 ± 7.1	115.6 ± 16.2	0.583 NS
DBP (mmHg)	86.8 ± 3.5	76.5 ± 10.9	<0.001*
PP (mmHg)	30.7 ± 5.8	39.1 ± 11.3	0.002*
LVEF (%)	—	62.0 ± 3.6	—

SBP = systolic blood pressure; DBP = diastolic blood pressure; PP = pulse pressure; LVEF = left ventricular ejection fraction; NS = not significant; * $p < 0.05$.

Ventilator Parameters

All MV patients were ventilated in SIMV-PC mode. Mean PaO_2/FiO_2 ratio of 228.4 ± 68.2 was consistent with mild-to-moderate hypoxaemic respiratory failure. Mean duration of ventilation before the study echocardiogram was 72.3 ± 24.6 hours (Table 3).

Table 3. Ventilator parameters at time of echocardiographic assessment (MV group, n=25).

Ventilator Parameter	Mean ± SD	Range
Mode	SIMV-PC (all)	—
Tidal Volume (mL)	452 ± 68	350–590
Tidal Volume (mL/kg PBW)	7.1 ± 0.8	5.6–8.5
Respiratory Rate (breaths/min)	16.4 ± 2.1	12–20
PEEP (cmH_2O)	6.8 ± 1.7	4–10

FiO_2 (%)	48.2 ± 12.4	30–70
Peak Insp. Pressure (cmH_2O)	22.4 ± 3.8	15–30
Mean Airway Pressure (cmH_2O)	12.1 ± 2.3	8–17
PaO_2/FiO_2 Ratio	228.4 ± 68.2	118–360
Duration MV before study (h)	72.3 ± 24.6	48–144

PEEP = positive end-expiratory pressure; SIMV-PC = synchronized intermittent mandatory ventilation – pressure-controlled; PBW = predicted body weight.

Aortic Dimensions and Elastic Indices

Absolute and indexed aortic diameters were statistically equivalent between groups (all $p > 0.05$), ruling out structural remodeling as a confounder. In marked contrast, three of four elastic indices showed highly significant impairment in the MV group: aortic strain was reduced by ~38.5%, stiffness index β was elevated by ~175%, and Peterson's Ep was elevated by ~76% (all $p < 0.001$). Aortic distensibility showed a consistent downward trend that did not reach statistical significance ($p = 0.087$), likely reflecting measurement variability across both diameter and pressure components (Table 2).

Table 2. Aortic dimensions and elastic indices (Mean ± SD).

Parameter	Normal (n=25)	MV (n=25)	p-value
AOS (mm)	31.68 ± 2.63	31.33 ± 2.85	0.641 NS
AOD (mm)	29.68 ± 2.43	29.87 ± 2.63	0.773 NS
Indexed AOS (cm/m^2)	1.75 ± 0.11	1.72 ± 0.13	0.413 NS
Indexed AOD (cm/m^2)	1.64 ± 0.10	1.63 ± 0.10	0.714 NS
Aortic Strain (%)	6.94 ± 1.77	4.27 ± 1.62	<0.001*
Distensibility ($\times 10^{-6}$ cm^2/dyn)	2.28 ± 1.12	1.82 ± 0.88	0.087 NS
Stiffness Index β	4.67 ± 1.48	12.86 ± 9.04	<0.001*
Peterson's Ep (mmHg)	456 ± 118	801 ± 298	<0.001*

AOS = aortic systolic diameter; AOD = aortic diastolic diameter; Ep = Peterson's elastic modulus; NS = not significant; * $p < 0.001$.

DISCUSSION

This prospective case-control study provides the first systematic echocardiographic evidence that mechanically ventilated RCU patients sustain substantial functional impairment of ascending aortic elastic properties despite preserved LVEF and structurally normal aortic dimensions. The clinical significance of these findings extends to hemodynamic monitoring, fluid management, and weaning decision-making in the critically ill.

The Windkessel function of the ascending aorta — buffering pulsatile stroke volume, storing systolic elastic energy, and releasing it during diastole to sustain coronary perfusion — is fundamentally dependent on aortic viscoelastic properties [14,15]. When impaired, the consequent hemodynamic perturbations are far-reaching: increased central pulse pressure and left ventricular afterload, diminished diastolic recoil, and augmented pulsatile stress on the renal, cerebral, and coronary microvasculature [16,17]. Many authors established cf-PWV as the clinical gold standard for arterial stiffness and demonstrated its independent predictive value for cardiovascular events and all-cause mortality [18,19]. The present study extends this mechanistic framework into the acute-to-subacute context of the RCU, where pathophysiological drivers are multifactorial and potentially more severe.

The most robust finding was a 38.5% relative reduction in aortic strain in the MV group (4.27 ± 1.62% vs. 6.94 ± 1.77%; $p < 0.001$). Aortic strain integrates stroke volume, proximal aortic impedance, and wall compliance across the cardiac cycle. Its reduction in the MV cohort is attributable to three converging mechanisms: PEEP-mediated preload reduction diminishing stroke volume and pulsatile aortic filling; extramural compression of the ascending aorta by elevated intrathoracic pressure attenuating outward systolic expansion; and intrinsic stiffening of the aortic wall from systemic inflammation, endothelial dysfunction, and oxidative stress [20,21]. The preservation of LVEF at 62.0 ± 3.6% critically dissociates the reduction in aortic strain from primary myocardial contractile deficit, implicating the arterial side of the ventriculo-arterial coupling system as the principal locus of impairment — consistent with the findings of [22,23].

Stiffness index β was elevated approximately

threefold in the MV group (12.86 ± 9.04 vs. 4.67 ± 1.48; p < 0.001). The theoretical pressure-independence of β — achieved through incorporation of a logarithmic systolic-to-diastolic pressure ratio — makes this finding particularly informative: the magnitude of elevation (175% above control values) substantially exceeds what would be predicted from the observed blood pressure differences alone, confirming genuine intrinsic mechanical alteration of the aortic wall [24,25]. The wide individual variation in β within the MV group (range 5.47–42.30, SD 9.04) reflects the heterogeneous spectrum of RCU admission diagnoses — pneumonia, ARDS, COPD exacerbation, post-operative respiratory failure — each differentially modulating systemic inflammatory burden, vasomotor dysregulation, and endothelial injury [26,27].

In the MV group, the elastic modulus Ep was significantly increased, more than double from baseline, compared with controls (801 ± 298 vs. 456 ± 118 mmHg; p < .001; ~76% relative increase). Approximately 27% of the total EP difference could be ascribed to the increased pulse pressure of the MV group over CV (39.1 vs. 30.7 mmHg). From the β-coefficient findings, 49% of the total increase in Ep likely represents intrinsic stiffness of the aortic wall, which supports pharmacological mechanisms based on load-dependent analytical models described by [28,29]. While aortic distensibility approached significant difference (p = 0.087), potentially indicating an inherent source for compound measurement error among the two variables used (i.e., meaningful size differences in the diameter and measurements) resulting in uncertainty propagating through each of the two variables [2], hence leading to a finding that, as indicated in their review, distensibility exhibits inter-measurement variability than either measure of strain or stiffness index combined in co-primary indices, thereby, when two of the co-primary indices significantly impaired, the overall trend for distensibility should be interpreted, in a biologically valid manner, rather than as providing contradictory information.

The complete preservation of absolute and BSA-indexed aortic dimensions in the MV group is a critical internal validation of the study findings. It confirms that the significantly impaired elastic indices cannot be attributed to unrecognized structural aortic dilation, aneurysmal disease, or congenital abnormality — all pre-specified exclusion criteria — and defines a pattern of functional aortic stiffness: viscoelastic impairment without structural remodeling, consistent with the acute-to-subacute critical illness time frame insufficient for matrix metalloproteinase-driven extracellular matrix remodeling [30,31]. This distinction has direct practical implications: standard RCU echocardiographic protocols that assess only aortic dimensions would fail to detect this functional mechanical impairment, underscoring the clinical value of calculating the elastic index.

The 100% technical success rate in obtaining adequate aortic M-mode windows — including all 25 supine mechanically ventilated patients with multiple monitoring devices — confirms the bedside feasibility of this assessment. This is consistent with large critical care echocardiography registries; Flower et al. demonstrated in the TRITONe study (1,015 shock patients, 178 UK ICUs) that echocardiography influenced management in 54% of critically ill patients [1]. The current study extends the echocardiographic utility to include aortic mechanical characterization as an additional hemodynamic monitoring layer. AI-assisted echocardiography, validated by [7], and technological advances reviewed by [15] will further reduce operator dependence and support routine clinical adoption of aortic elastic index assessment in resource-limited RCU settings. Competency frameworks outlined by [16] provide the training infrastructure required to ensure standardized measurement quality.

Several limitations must be acknowledged. The cross-sectional design of the study precludes determining a causal relationship between positive-pressure ventilation and increased aortic stiffness; further longitudinal studies with pre-ventilation baselines are needed. Ventilator parameters (PEEP and driving pressure) could not be consistently included as covariates when performing multivariate analyses. The unstandardized and unquantified nature of the type and dose of vasoactive medications might represent an important confounding factor, since catecholamines exert direct alpha-adrenergic effects on aortic smooth muscle tone [33]. The presence of considerable variability in M-mode-derived measurements (5-15%) may have reduced the significance of the distensibility comparisons in this study, and speckle-tracking echocardiography will likely ensure greater reproducibility in future studies. The single-center nature of this study limits extrapolation of the findings to other centers, and the absence of clinical outcomes precludes evaluation of the prognostic value of the stiffness indices measured in this cohort; further multicenter prospective studies would be required to identify such indices.

CONCLUSION

Mechanical ventilation of critically injured patients can alter the elasticity of blood vessels, such as the aorta. This study demonstrates that critically ill patients who have been mechanically ventilated exhibit significant ascending aortic dysfunction (reduced strain and increased aortic stiffness index) compared with healthy controls, despite normal left ventricular systolic function (squeezing ability) and normal aortic size. These results indicate that the acute functional aortic stiffness in critically ill patients

is primarily due to mechanical ventilation and critical illness. Measuring aortic elasticity should be performed routinely with bedside echocardiography and integrated into a comprehensive hemodynamic monitoring system for mechanically ventilated patients in the critical care unit. Future multicenter, prospective studies are needed to determine whether aortic elasticity indices can serve as a safe and reliable clinical tool to predict outcomes related to mechanical ventilation, pharmacologic support, duration of weaning from mechanical ventilation, and/or long-term cardiovascular health.

BIBLIOGRAPHY

1. Flower L, Waite A, Boulton A, Wilson P, Huang S, Christie L, et al. The use of echocardiography in the management of shock in critical care: A prospective, multi-center, observational study. Intensive Care Med. 2024;50(10):1668–80.
2. Niwińska MM, Chlabicz S. Evaluation of arterial stiffness parameters measurement with noninvasive methods: A systematic review. Cardiol Res Pract. 2024;2024:4944517.
3. Mileva N, Velikova T, Velikov T, Vassilev D. Aortic elasticity and cardiovascular risk stratification: A narrative review. J Vasc Dis. 2024;3(1):88–101.
4. Kadoglou NPE, Khattab E, Siddique M, Haddad M, Gupta I. Aortic stiffness: A major risk factor for multimorbidity in older people. J Clin Med. 2023;12(6):2321.
5. Monaco F, Belletti A, Bove T, Landoni G, Zangrillo A. Cardiovascular effects of mechanical ventilation. Compr Physiol. 2022;12(1):3121–44.
6. Cutuli SL, Grieco DL, Michi T, Cesarano M, Rosà T, Pintaudi G, et al. The cardiovascular effects of mechanical ventilation and positive end-expiratory pressure. J Clin Med. 2023;12(13):4176.
7. Gohar E, Herling A, Mazuz M, Tsaban G, Gat T, Kobal S, et al. Artificial intelligence versus POCUS expert: A validation study of three AI-based hemodynamic echocardiographic assessment tools. J Clin Med. 2023;12(4):1352.
8. Vieillard-Baron A, Matthay M, Teboul JL, Bein T, Schultz M, Magder S, et al. Experts' opinion on management of hemodynamics in ARDS patients. Intensive Care Med. 2016;42(5):739–49.
9. Qadir N, Sahetya S, Munshi L, Summers C, Abrams D, Beitler J, et al. An update on management of adult patients with ARDS: An ATS clinical practice guideline. Am J Respir Crit Care Med. 2024;209(1):24–36.
10. Battaglini D, Rocco PRM, Pelosi P. New insights in mechanical ventilation and adjunctive therapies in ARDS. Signa Vitae. 2022;18(5):1–11.
11. Chaves RCF, Quaresma VNF, Otsuki DA, Sanches LC, Ribeiro NM, Corrêa TD, et al. Assessment of fluid responsiveness using dynamic indices in mechanically ventilated patients: A systematic review and meta-analysis. Crit Care. 2024;28:289.
12. Messina A, Caporale M, Calabrò L, Lionetti G, Bono D, Gualtieri A, et al. Reliability of pulse pressure and stroke volume variation in assessing fluid responsiveness: A meta-analysis. Crit Care. 2023;27:431.
13. Monnet X, Teboul JL. Prediction of fluid responsiveness: What's new? Ann Intensive Care. 2022;12:46.
14. Muñoz F, Born P, Bruna M, Ulloa R, González C, Philp V, et al. Coexistence of fluid-responsive state and venous congestion in critically ill patients: A multicenter observational study. Crit Care. 2024;28(1):52.
15. Guevarra K, Greenstein Y. Point-of-care ultrasonography in the critical care unit: An update. Curr Cardiol Rep. 2025;27(2):1–13.
16. Sreedharan R, Khanna S, Moghekar A, Dugar S, Collier P. Critical care echocardiography: A self-assessment book. Cham: Springer Nature; 2024.
17. Santus P, Radovanovic D, Saad M, Zilianti C, Coppola S, Chiumello DA, et al. Acute dyspnea in the emergency department: A clinical review. Intern Emerg Med. 2023;18(5):1491–507.
18. Cockroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976;16(1):31–41.
19. Stefanadis C, Stratos C, Boudoulas H, Kourouklis C, Toutouzas P. Distensibility of the ascending aorta: comparison of invasive and non-invasive techniques. Eur Heart J. 1990;11(11):990–6.
20. Spronck B, Avolio AP, Tan I, Butlin M, Reesink KD, Delhaas T. Arterial stiffness index beta and cardio-ankle vascular index inherently depend on blood pressure. J Hypertens. 2017;35(1):98–104.
21. Alrekaby HAK, Ghiad IA, Al-Saad SF. Correlation between hypertension and risk factors among ischemic heart disease patients in Babylon governorate. Int J Health Sci. 2022;6(S6):3854–63.
22. Laurent S, Cockcroft J, Van Bortel L, Boutouyrie P, Giannattasio C, Hayoz D, et al. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J. 2006;27(21):2588–605.
23. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. J Am Coll Cardiol. 2010;55(13):1318–27.
24. Westerhof N, Lankhaar JW, Westerhof BE. The arterial Windkessel. Med Biol Eng Comput. 2009;47(2):131–41.
25. O'Rourke MF, Hashimoto J. Mechanical factors in arterial aging: a clinical perspective. J Am Coll

Cardiol. 2007;50(1):1–13.
26. Pinsky MR. Cardiovascular issues in respiratory care. Chest. 2005;128(5 Suppl 2):592S–7S.
27. Chirinos JA. Arterial stiffness: basic concepts and measurement techniques. J Cardiovasc Transl Res. 2012;5(3):243–55.
28. Mitchell GF. Arterial stiffness and hypertension: chicken or egg? Hypertension. 2014;64(2):210–4.
29. Ben-Assa E, Bader Y, Reiter Y, Grupper A, Volodarsky O, Fudim M, et al. Aortic wall strain as a predictor of outcome in patients admitted to the intensive care unit. Intensive Care Med. 2019;45(7):966–74.
30. Safar ME, Levy BI, Struijker-Boudier H. Current perspectives on arterial stiffness and pulse pressure in hypertension and cardiovascular diseases. Circulation. 2003;107(22):2864–9.
31. Cavalcanti AB, Suzumura EA, Laranjeira LN, Paisani DM, Damiani LP, Guimarães HP, et al. Effect of lung recruitment and titrated PEEP vs low PEEP on mortality in ARDS: A randomized clinical trial. JAMA. 2017;318(14):1335–45.
32. Lantelme P, Mestre C, Lievre M, Gressard A, Milon H. Heart rate: an important confounder of pulse wave velocity assessment. Hypertension. 2002;39(6):1083–7.
33. Wilkinson IB, Qasem A, McEniery CM, Webb DJ, Avolio AP, Cockcroft JR. Nitric oxide regulates local arterial distensibility in vivo. Circulation. 2002;105(2):213–7.

