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RESEARCH ARTICLE

Vascular Responses to Hand Posture are Preserved Despite Acute Endothelial Dysfunction: Insights from Pulse Wave Velocity and Finger-to-Finger Timing

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ABSTRACT

Limb posture alters hydrostatic pressure and vascular tone through mechanical and reflex pathways, but the contribution of endothelial function remains uncertain. To determine whether vascular responses to hand elevation and dependency persist during transient endothelial dysfunction induced by ischemia–reperfusion injury (IRI), eighteen healthy adults (9 males, 9 females) underwent ECG-gated pulse recordings at three hand positions: heart level (H-0˚), 90˚ below (H-90˚), and 50˚ above (H + 50˚), before and after IRI. Primary outcomes were pulse wave velocity (PWV) and finger-to-finger pulse arrival-time difference (f–f ΔT); secondary outcomes were wrist mean blood pressure (MBP), heart rate (HR), and ΔPWV/ΔBP slope. The result at baseline, hand elevation reduced PWV by 10–13% and increased f–f ΔT by 600–727%, while dependency increased PWV by ∼7% and decreased f–f ΔT by ∼200%. After IRI, these postural responses persisted, with only a modest 3% PWV reduction at H-90˚ and H-0˚ ($p < 0.05$). MBP shifted with posture as expected, and HR rose slightly with dependency but decreased overall after IRI. The ΔPWV/ΔBP slope was steeper in elevation, consistent with enhanced compliance. In conclusion, vascular responses to hand posture remain preserved despite acute endothelial dysfunction, indicating that short-term postural adjustments in arterial stiffness are mediated predominantly by hydrostatic and reflex mechanisms rather than endothelial pathways.

Keywords: Arterial stiffness, Finger-to-Finger differential, Hydrostatic pressure, Ischemia–reperfusion injury, Pulse transit time, Pulse wave velocity, Venoarteriolar reflex

Introduction

Arterial function is regulated by several fundamental mechanisms that preserve stable tissue perfusion under varying pressures. Autoregulation of blood flow involves myogenic, metabolic, and endothelial responses that operate at the arteriolar level to maintain vascular tone and compliance.[1–4] Experimental studies have also explored cardiovascular dynamics using exercise-induced stress and Doppler-based hemodynamic techniques to assess vascular reactivity in humans.[5,6] Endothelial modulation plays a central role in vascular function and dysfunction.[7,8] Endothelial impairment is an early marker of cardiovascular disease and is strongly associated with increased arterial stiffness and cardiovascular risk.[9,10] To obtain reliable, non-invasive data on vascular compliance, we often monitor pulse transit time (PTT) and pulse wave velocity (PWV).[11] The direction of a limb significantly decides arterial stiffness because it shifts hydrostatic pressure and venous filling. When we raise a subject's arm, the resulting reduction in transmural pressure and wall tension promotes vasodilation and improves compliance.

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Conversely, dependency increases local pressure and triggers myogenic tone. While several mechanisms drive these postural adaptations, disease states may cause them to fail.[12–14] We can quantify these shifts using PWV and PTT, or by employing the finger-to-finger arrival-time difference (f–f ΔT), a sensitive index of vascular timing and responsiveness.[15,16] Although we recognize these postural effects, we still do not fully understand the extent of endothelial involvement. Some data suggest that the endothelium releases nitric oxide to assist vasodilation during elevation,[17,18] yet other reports identify mechanical factors and the venoarteriolar reflex (VAR) as the dominant drivers.[14] Clarifying this mechanism holds clinical importance. We used ischemia–reperfusion injury (IRI) to model acute endothelial dysfunction, allowing us to test if postural vascular responses survive even when the endothelium is impaired.[7] We proposed that the characteristic vascular markers of hand elevation, reduced PWV, and elevated f–f ΔT would survive the ischemic challenge of IRI. We then employed a hand-elevation protocol alongside ECG and pulse wave analysis[19,20] to investigate whether endothelial function actually regulates these specific postural responses.

Materials and methods

Ethical approval and participants

Following the principles of the Declaration of Helsinki (2013), we received ethical permission for this research from the College of Medicine, Mustansiriyah University in Baghdad. We ensured every volunteer gave verbal informed consent prior to the start of the investigation. Between March and April 2025, we enrolled 18 normotensive subjects, split equally between nine men and nine women. We limited our cohort to non-smokers who were free from any chronic illness. For our female participants, we mandated regular menstrual cycles and conducted all trials during the early follicular stage—specifically between days 1 and 4—to account for potential hormonal shifts.

Anthropometric and hemodynamic assessment

Height and weight were recorded, and body mass index (BMI) was calculated as weight (kg) / [height (m)]^2. After 10 minutes of rest, brachial blood pressure (SBP, DBP) and heart rate (HR) were measured using an automatic device (Rossmax, Switzerland). Wrist blood pressure was assessed at three hand positions (H-0°, H-90°, H + 50°) using an automatic OromED sphygmomanometer.

Experimental protocol

Participants remained seated throughout. Digital pulse wave (DPW) signals were recorded from both hands using piezoelectric transducers, and ECG (Lead II) was recorded simultaneously. Signals were sampled at 1 kHz using a data acquisition unit 26T and LabChart Pro 7.2 (ADInstruments, Australia) for recording and offline signal analysis.

- **Phase I – Baseline:** Following 10 minutes at heart level (H-0°), 1-minute DPW and ECG signals were recorded. The left hand was then lowered (H-90°) and elevated (H + 50°) for 2 minutes each, followed by 1-minute recordings.
- **Phase II – Endothelial Dysfunction:** IRI was induced by inflating a pneumatic cuff on the left brachial artery (230 mmHg for 20 minutes), followed by 15 minutes of reperfusion. Post-IRI recordings were repeated for all three positions.[7]

PWV and PTT measurement

PTT was defined as the interval between the ECG R wave and the peak of the first derivative of the DPW waveform (FDDPW), corresponding to the 50% rise point.[21] PWV was calculated as:

$$\text{PWV (m/s)} = \text{D/PTT}$$

where D = 0.5 × body height[22]

Finger-to-Finger pulse arrival time difference (f–f ΔT)

Calculated as the difference in pulse arrival times between left and right middle fingers[20] using FDDPW peaks:

$$\text{f} - \text{f } \Delta\text{T} = \text{PTT (left)} - \text{PTT (right)}$$

Negative values indicated a higher PTT (longer pulse arrival time) at the right finger. Measurements were obtained from 13 participants rather than from 18 volunteers due to technical limitations in some recordings.

Arm positioning and angle verification

The elevated hand (H + 50°) was supported on a padded frame, with fingers free. Subjects were instructed to minimize movement. Elevation angles were verified via lateral photographs and protractor-based analysis. The right arm remained supported at heart level.

Table 1. Anthropometric and systemic measurements of the participants.

Parameters:	Male (n = 9)	Female (n = 9)	P<
Age (years)	23.2 ± 4.4	20.6 ± 3.3	NS
BMI (kg/m^2)	23.6 ± 3.7	24.4 ± 3.8	NS
SBP (mmHg)	121.1 ± 7.5	110.6 ± 9.5	0.02
DBP (mmHg)	64.3 ± 6.6	67.9 ± 11.5	NS
MBP (mmHg)	83.3 ± 6.5	82.1 ± 9.8	NS
HR (beat/min)	80.0 ± 14.4	78.9 ± 11.2	NS
PWV (m/sec) at H-0° position before IRI	3.1 ± 0.3	3.0 ± 0.2	NS
PWV (m/sec) at H-0° position after IRI	3.1 ± 0.2	2.9 ± 0.2	NS
f-f ΔT (msec) at H-0° position before IRI	10.9 ± 6.6	3.8 ± 10.8	NS
f-f ΔT (msec) at H-0° position after IRI	5.6 ± 4.6	5.6 ± 9.7	NS

Note: P-values represent the differences between genders at baseline (H-0°). Pre- vs post-IRI in between groups of pooled data are presented in Figs. 1 to 3. BMI = Body mass index; SBP, DBP, and MBP = Systolic, diastolic, and mean blood pressure; HR = Heart rate; PWV = Pulse wave velocity; H-0 position = The hand at the level of the heart; IRI = Ischemic reperfusion injury; f-f ΔT = The time difference between pulses that reach each hand's middle fingers.

Statistical analysis

Data were expressed as mean ± standard deviation. Normality was assessed using the Kolmogorov–Smirnov test. Depending on distribution, paired and unpaired t-tests or non-parametric Wilcoxon matched-pairs and Mann–Whitney U tests were used for intra- and inter-group comparisons. Significance was set at $p < 0.05$. Analyses were performed using GraphPad InStat (version 3.06).

Results

Participant characteristics

Eighteen healthy, normotensive participants completed the study. Anthropometric and baseline cardiovascular parameters are summarized in Table 1. No significant differences were observed between genders, except for SBP, which was significantly higher in males ($p < 0.02$). Therefore, data from both genders were pooled and considered as one group.

PWV across arm positions

At baseline (pre-IRI), PWV in the left heart–finger pathway at H-0° was 3.1 ± 0.3 m/s Fig. 1. Hand dependency (H-90°) significantly increased PWV by 7%, while hand elevation (H + 50°) significantly reduced PWV by 13%. After IRI, similar trends were observed in which the PWV of the left heart–finger pathway at the H-0° hand position was 3.0 ± 0.2 m/s. On moving the left hand to the lowest position (i.e., H-90°, 90° below heart level), the PWV increased significantly by 7%, and decreased significantly by 10% when the hand was moved to the H + 50° position Fig. 1.

Notably, IRI caused a small but significant reduction in PWV at H-90° and H-0° compared to pre-IRI values (3% decrease), but no significant change was found at H + 50° Fig. 1. The maximal PWV difference between H + 50° and H-90° positions was not significantly altered by IRI.

Wrist mean blood pressure (MBP)

Wrist MBP at H-0° was 84.8 ± 10.8 mmHg. At H-90°, MBP rose significantly by 36%, while at H + 50°, it dropped by 30%. Despite these shifts, the ΔPWV/ΔBP at H + 50° (0.016 m/s/mmHg) was nearly twice that at H-90° (0.007 m/s/mmHg), indicating enhanced vascular sensitivity with steeper PWV change during elevation.

Heart rate (HR)

Baseline HR at H-0° was 78.6 ± 10.9 bpm and decreased to 75.7 ± 9.6 bpm after IRI. HR increased by 3–5% upon moving the hand to H-90°, in both pre- and post-IRI Fig. 2. No significant HR change was observed with hand elevation. IRI was associated with a significant reduction in HR at all positions.

Finger-to-Finger pulse arrival time difference (f–f ΔT)

At baseline, f–f ΔT was 7.1 ± 9.5 ms at H-0°, decreasing to –14.2 ± 7.9 ms at H-90° (200% reduction), and increasing to 49.6 ± 18.3 ms at H + 50° (600% increase) Fig. 3. Post-IRI, f–f ΔT values followed a similar pattern: f–f ΔT at H-0° was 5.6 ± 7.5 ms, dropped to –13.4 ± 8.3 ms at H-90° (a decrease of 239%), and rose to 46.3 ± 16.1 ms at H + 50° (727% increase). No significant changes in the f–f ΔT response were observed after IRI. The maximum differences in f–f ΔT

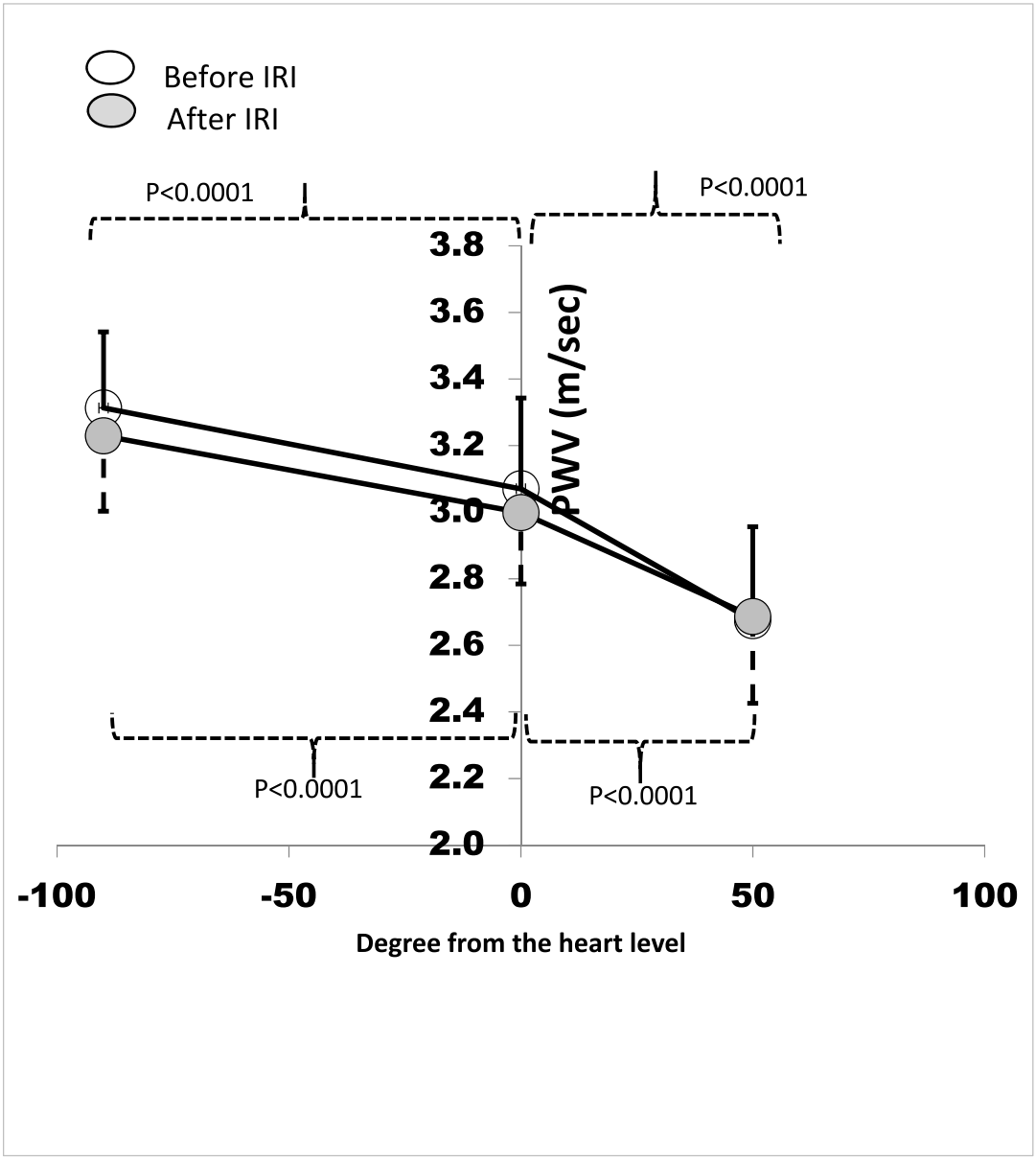

Fig. 1. Heart-Finger pulse wave velocity (PWV) when the hand is at –90°, 0°, and 50° from the heart level before and after ischemic re-perfusion injury. (n = 18).

(by subtracting the value at H-90° from the value at H + 50°) indicate that no significant changes occurred before and after IRI at any hand position Fig. 3.

Discussion

Using PWV and f–f ΔT as non-invasive markers of compliance, we found that hand elevation decreased PWV and increased f–f ΔT—indicating enhanced compliance—while hand dependency had the opposite effect. These trends were maintained after IRI-induced endothelial dysfunction, suggesting minimal endothelial involvement in posture-driven vascular changes. PWV changes align with established hemodynamic principles: elevation reduces hydrostatic pressure and wall tension, favoring vasodilation and compliance; dependency increases transmural pressure, enhancing myogenic tone and stiffness.[11,23–25] This study evaluated whether vascular stiffness responses to hand posture are modulated by endothelial function. Although differences in PWV in young males and females were reported in some reports,[26] however, in our experiment such differences were not observed and it was consistent with other reports.[27] Again, if PWV shows a gender-dependent trend, having

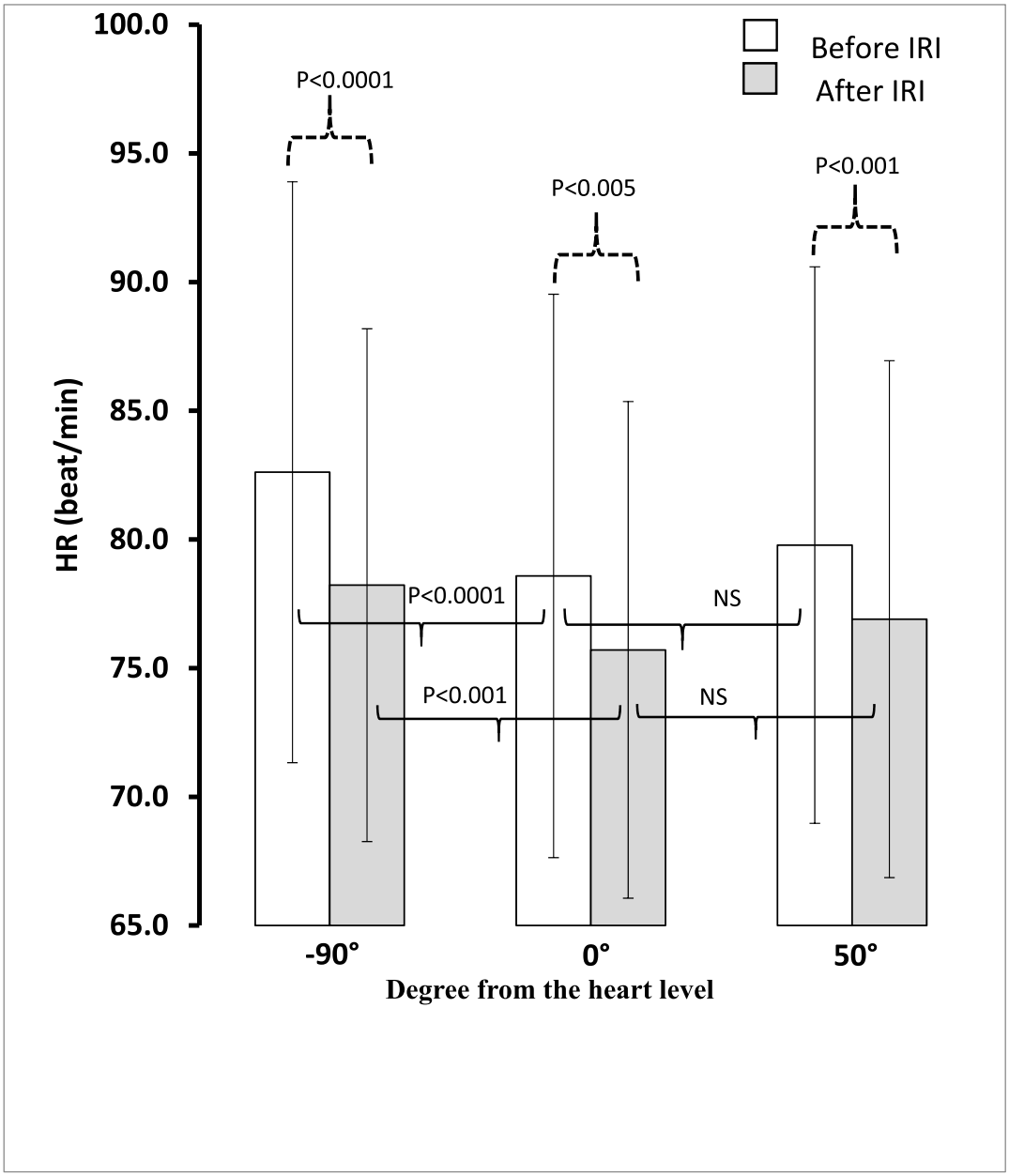

Fig. 2. Comparison of heart rate (HR) at the three hand positions before and after ischemic re-perfusion injury (IRI). (n = 18).

an equal number of male and female participants minimizes the impact of this difference. Our findings support a key role for the VAR—a neuro-myogenic response to venous distension—which induces arteriolar vasoconstriction in dependent limbs.[12] Its attenuation during elevation allows vasodilation to dominate. Previous studies show that hand elevation delays systolic peaks and increases PTT (i.e., decreases in PWV), consistent with our results.[28,29] HR increased only at H-90°, suggesting a systemic VAR-mediated sympathetic response.[30] The persistence of maximum ΔPWV and maximum difference in f–f ΔT after IRI reinforces the notion that endothelial NO does not dominate short-term postural vascular adjustments.[17,18] At present, there is no evidence that hand posture directly influences pulse wave velocity (PWV) via endothelial nitric oxide (NO) release. The mention of NO in this context was intended only as a hypothetical explanation of the vascular response to changes in hand posture. Nitric oxide (NO) has no role in the context of vascular response during hand posture changes. Although the NO plays a central role in modulating the vascular diameter and stiffness in many other conditions, yet no production of this agent was reported during hand posture changes according to

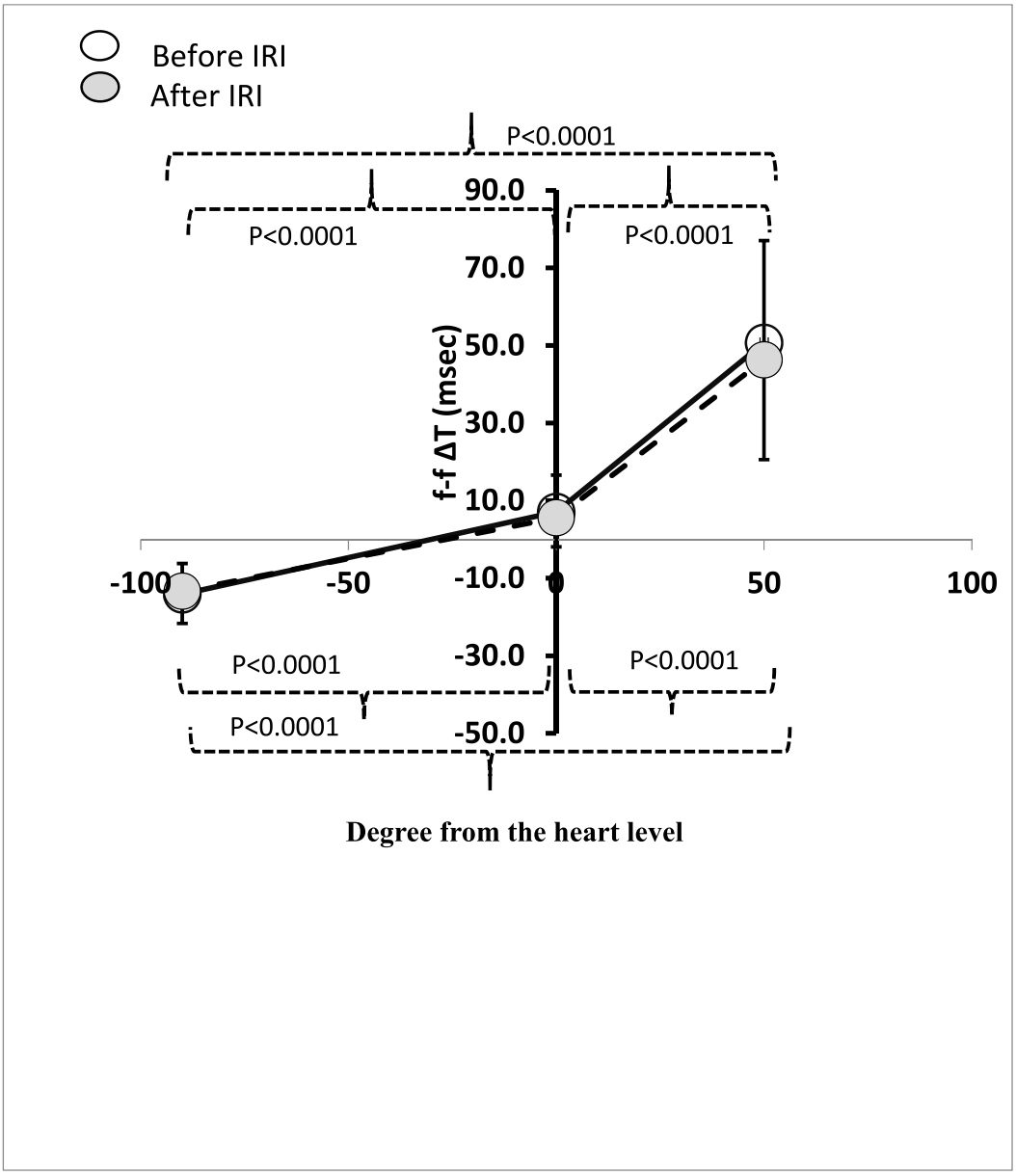

Fig. 3. The time difference between pulses that reach each hand's middle fingers (f-f ΔT) at the three hand positions before and after ischemic re-perfusion injury (IRI). (n = 13).

the available literatures so far. Basically, the vascular stiffness (and PWV) is due to vascular smooth muscle tone, which is affected by transluminal distending pressure. An increase in transluminal distending pressure stimulates reflex contraction of vascular smooth muscle and consequently increases the vascular stiffness. On the contrary, a decrease in transluminal distending pressure abolishes the reflex contraction of vascular smooth muscle and consequently decreases the vascular stiffness. This transluminal distending pressure is affected by vascular smooth muscle contraction (sympathetic nervous system activation) and relaxation by the release of NO. IRI, used to transiently impair endothelial function,[7,31] modestly reduced PWV but did not alter overall postural trends. The general small reduction in PWV specifically after IRI may reflect HR reduction due to sympathetic withdrawal and parasympathetic dominance, as reported in short cyclic intermittent ischemia-reperfusion studies.[7,32] Anatomical asymmetries, such as differences in subclavian artery length, likely contributed to the directionality of f–f ΔT values.[33] f–f ΔT changes paralleled PWV patterns, validating it as a reliable compliance marker.[20] Our

data revealed an elevated ΔPWV/ΔBP ratio during arm elevation, which points toward improved vascular compliance despite the relatively small pressure changes. This specific observation underscores the non-linear behavior of arterial stiffness, a property dictated by vascular elasticity and smooth muscle tone in addition to transmural pressure.[34] To summarize, we conclude that mechanical and reflexive drivers, namely hydrostatic gradients and the venoarteriolar reflex (VAR) serve as the primary mediators for these acute postural adaptations. Our results further demonstrate that transient endothelial function plays only a negligible role in these vascular shifts.

Conclusion

Our research confirms that vascular reactions to hand posture remain remarkably stable, even when we induce acute endothelial dysfunction through ischemia–reperfusion injury (IRI). We documented that pulse wave velocity (PWV) and finger-to-finger timing (f–f ΔT) shifted in the anticipated directions: hand elevation lowered PWV and extended f–f ΔT, whereas dependency raised PWV and shortened f–f ΔT. Most importantly, these posture-induced patterns endured throughout the IRI challenge. Our analysis of f–f ΔT helps resolve existing contradictions in scientific literature; specifically, rising positive values signal a pulse delay in the raised hand, while shifts toward zero or negative values indicate accelerated transit in the dependent limb. Therefore, f–f ΔT functions as a metric that reinforces rather than opposes PWV findings. These results validate our conclusion that mechanical and reflexive drivers, primarily the venoarteriolar reflex (VAR) and hydrostatic loading, govern postural vascular changes, with no detectable role for the endothelium. The steeper ΔPWV/ΔBP slope we observed during elevation, despite the lower pressure environment, further emphasizes a state of heightened vascular compliance. From a clinical perspective, we recommend hand-elevation testing as a dependable method for probing vascular compliance, even in populations with impaired endothelial function, such as those suffering from early atherosclerosis, diabetes, or hypertension.

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images that are not ours have been included with the necessary permission for republication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Author(s) signed off on ethical considerations approval.
- Ethical Clearance: The project was approved by the local ethical committee at Mustansiriyah University.

Authors' contributions statement

ZAFA: theorized the study design, carried out all experimental procedures, handled data acquisition and analysis, and wrote the initial manuscript draft. FSN: provided overall research supervision and checked the validity of our results. BTA: Managed research resources, oversaw the project's progression, and contributed to the critical editing of the text. All authors reviewed the final version of the manuscript and granted their approval for this submission.

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الاستجابات الوعائية لتغير وضع اليد تبقى محفوظة رغم الخلل البطاني الحاد: دراسة باستخدام سرعة موجة النبض والفارق الزمني بين الإصبعين

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قسم الفسلجة، كلية الطب، الجامعة المستنصرية، بغداد، العراق.

الخلاصة

تؤثر التغيرات في الضغط الهيدروستاتيكي الناتجة عن تغيير وضع الطرف العلوي، إلى جانب منعكس الوريد-الشرياني (VAR)، على التفاعلات الوعائية ولا سيما صلابة الأوعية الدموية، وذلك عبر آليات ميكانيكية وانعكاسية إضافة إلى بعض التأثيرات البطانية. وتُعد سرعة موجة النبض (PWV) والفارق في زمن وصول النبضة بين الإصبعين (f–f ΔT) من المؤشرات غير الجراحية الموثوقة لقياس صلابة الشرايين.هدفت هذه الدراسة إلى تقييم ما إذا كانت الاستجابات الوعائية الناتجة عن رفع أو خفض اليد تعتمد على سلامة البطانة الوعائية، وذلك من خلال قياس كل من PWV وf–f ΔT قبل وبعد إحداث خلل بطاني عابر باستخدام نموذج إصابة نقص التروية وإعادة التروية ((IRI. شملت الدراسة 18 متطوعًا سليمًا خضعوا لتسجيل إشارات النبض وتخطيط القلب (ECG) في ثلاثة أوضاع لليد: بمستوى القلب، و90 درجة أسفل مستوى القلب، و50 درجة أعلى مستوى القلب. أظهرت النتائج أن رفع اليد أدى إلى زيادة f–f ΔT بنسبة 600%–727% وانخفاض PWV بنسبة 10%–13%، مما يشير إلى زيادة الامتثال الوعائي. بينما أدى خفض اليد إلى ارتفاع PWV بنسبة 7% وانخفاض f–f ΔT بنسبة 200%–239%. بعد تحفيز الخلل البطاني (IRI) لم تتغير هذه الأنماط بشكل يُذكر، مما يدل على أن الاستجابة الوعائية بقيت محفوظة وتعتمد بصورة أساسية على الآليات الميكانيكية والانعكاسية أكثر من اعتمادها على التعديلات البطانية الحادة.

الكلمات المفتاحية: إصابة نقص التروية وإعادة التروية، الاستجابة الوريدية-الشريانية، الفارق الزمني بين الإصبعين، سرعة موجة النبض، صلابة الشرايين، ضغط هيدروستاتيكي، زمن انتقال النبضة.