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Reactive hyperemia index and arterial stiffness following the arm and the forearm occlusion: Comparative study

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Abstract

Reactive hyperemia (RH) is a temporary increase in blood flow after ischemia, associated with reduced arterial stiffness and used as a marker of vascular health. Arterial stiffness, a key cardiovascular risk factor, is commonly assessed using pulse transit time (PTT), a non-invasive method. This study examines the effect of occlusion site (arm vs. forearm) on RH magnitude and its relationship with vascular stiffness.Ten healthy males (23.1 ± 5.0 years) participated. PTT and digital pulse amplitude (DPWA) were measured before and after RH induction at baseline and one-minute intervals for five minutes post-induction. Arm occlusion led to a post-occlusion reactive hyperemia index (RHI) increase of 23% at 2 minutes ($P < 0.03$). Forearm occlusion resulted in RHI increases of 36%, 27%, 27%, and 18% at 2, 3, 4, and 5 minutes, respectively, compared to 1 minute. No significant differences in RHI were found between arm and forearm occlusion, with RH sustained up to 5 minutes after forearm occlusion.PTT increased significantly by 6% and 4% at 1 and 2 minutes after arm occlusion release. Following forearm occlusion, PTT was significantly higher (5%) than baseline only at 1-minute post-release. The peak RHI occurred in the second minute after occlusion release, with forearm RH lasting longer (up to 5 minutes), though magnitudes were similar. Peak PTT, indicating maximum vascular stiffness reduction, occurred in the first minute post-release and may extend into the second minute.

Introduction:

Reactive hyperemia (RH) refers to the transient increase in blood flow that occurs following a period of ischemia (temporary restriction of blood supply). This physiological response plays a crucial role in vascular health, as it reflects endothelial function and microvascular reactivity [1,2]. Vasodilator metabolites, tissue hypoxia, and the shear stress-induced, endothelium-dependent release of nitric oxide (NO) from the endothelium widen arterioles and reduce vascular resistance during the occlusion period to counteract ischemic stress [3]. Because reduced vascular resistance persists when perfusion pressure is restored, blood flow increases [3]. Arterial stiffness, on the other hand, is a key indicator of cardiovascular disease (CVD) risk and is commonly assessed using measures like pulse wave velocity (PWV) or its derived variable, pulse transit time (PTT) [4].

The relationship between reactive hyperemia and arterial stiffness is complex, with emerging evidence suggesting that impaired hyperemic responses may be associated with increased arterial stiffness. One of the primary mechanisms linking these two phenomena is endothelial dysfunction. The endothelium regulates vascular tone through the release of vasodilator substances such as NO. In individuals with arterial stiffness, endothelial dysfunction may lead to a diminished hyperemic response due to reduce NO bioavailability [5]. Additionally, arterial stiffness increases pulsatile load, which can damage the microcirculation and further impair reactive hyperemia [6]. Several studies have demonstrated an inverse relationship between reactive hyperemia and arterial stiffness. For example, research has shown that individuals with higher PWV (lower PTT), indicative of increased arterial stiffness, tend to exhibit blunted reactive hyperemia responses [7]. This suggests that microvascular dysfunction may contribute to the progression of arterial stiffness and related cardiovascular complications.

In conclusion, RH and arterial stiffness are interrelated through mechanisms involving endothelial function and vascular compliance. A reduced hyperemic response may serve as an early marker of arterial stiffening and cardiovascular risk. Further research is needed to explore potential interventions aimed at improving vascular health through endothelial function enhancement.

The placement of the occlusion cuff whether on the upper arm or forearm—can significantly affect the magnitude of reactive hyperemia and its relationship with arterial stiffness. Cuff placement influences the degree of ischemia and subsequent vasodilation. Studies suggest that upper arm occlusion elicits a greater hyperemic response compared to forearm occlusion due to the involvement of larger conduit arteries, such as the brachial artery, which have a greater capacity for vasodilation [8]. In contrast, forearm occlusion primarily affects smaller resistance arteries, resulting in a more localized response that may not fully reflect systemic vascular function. Research has shown that upper arm occlusion leads to a more pronounced reduction in arterial stiffness post-occlusion compared to forearm occlusion, likely due to a more substantial shear stress-induced, endothelium-dependent NO release [9]. Conversely, forearm occlusion may produce a weaker response, potentially underestimating vascular endothelial function in individuals with early-stage arterial stiffening.

Additionally, the choice of cuff placement may have clinical implications when assessing cardiovascular risk. A study by Naka et al. (2006) [10] demonstrated that upper arm occlusion is more predictive of systemic endothelial dysfunction and arterial stiffness compared to forearm occlusion, suggesting that it may be the preferred method for evaluating cardiovascular health. The aim of the current study is to ascertain how the positions of the occlusion cuff affect the vascular stiffness and RH peak's magnitude and temporal pattern.

Subjects and Methods

Ten healthy male subjects, aged 23.1 ± 5.0, were recruited for the current investigation. All participants had no known medical conditions and were not taking any medications. They were told to abstain from tea, coffee, and tobacco for two hours prior to the test, fast for two hours beforehand, and avoid vigorous physical activity for 12 hours before the study. Before providing written informed consent, each participant was fully briefed about the study. The study adhered to the principles of the Declaration of Helsinki, and all procedures were approved by the institutional ethics committee.

Experimental Protocol

An automated sphygmomanometer (Rossmax, Switzerland) was used to measure resting heart rate (HR) and systolic and diastolic blood pressure (SBP, DBP). To guarantee hemodynamic stability, repeated measurements of the right brachial SBP, DBP, and HR were made in a semi-supine position following 10 minutes of rest.

Digital pulse wave (DPW) signals were measured from the left and right middle fingertips using piezoelectric crystal mechanotransducers. A Lead-II ECG (using three surface electrodes) was simultaneously recorded for five minutes. The mechanotransducers' analog signals were converted into digital signals by the PowerLab Data Acquisition System (Acquisition Unit 26T, AD Instruments Pty Ltd, New South Wales, Australia). Offline analysis of ECG and DPW signals was performed using LabChart Pro software, with pulse wave parameters extracted via parameter-specific peak-detection algorithms. ECG and DPW signals were acquired at a sampling rate of 1 kHz. All participants were subjected to the following procedure:

1. A two-minute baseline recording of ECG and DPW signal. 2. Complete arterial occlusion was achieved by inflating a blood pressure cuff around the left upper arm to ≥50 mmHg above baseline SBP for five minutes, ensuring minimal discomfort and consistent application across participants. Occlusion efficacy was verified in real time by monitoring the absence of the DPW signal. Real-time occlusion verification was achieved by monitoring the absence of the DPW signal. 3. Following cuff deflation, the RH phase began, and DPW/ECG signals were continuously recorded for five additional minutes [11]. PTT was measured as the interval between the R-wave peak of the ECG and the first derivative peak of the DPW signal [12]. DPWA was used to quantify changes in blood flow during RH. The relative change in DPWA from the five-minute post-ischemic phase compared to the pre-ischemic baseline served as an index of RH magnitude. To normalize for systemic effects, the reactive hyperemia index (RHI) was calculated as follows: the ratio of the occluded arm's mean DPWA at each minute post-occlusion (A) to the mean DPWA from the same arm's baseline readings (B), divided by the same ratio obtained from the control (non-occluded) arm (C/D). This ratio accounts for systemic vascular changes during testing.
RHI = [A/B] / [C/D] [11].

Statistical Analysis

All data are presented as mean ± standard deviation (SD). Paired Student's t-tests were used for within-group comparisons, and unpaired Student's t-tests were used for between-group comparisons. A p-value < 0.05 was considered statistically significant. Analyses were performed using GraphPad InStat (version 3.06).

Results

The characteristics of the recruited volunteers in the current study are presented in Table 1. Although the participants were randomly sampled, the BMI of the recruited volunteers indicates that they were overweight. Arm occlusion resulted in a post-occlusion

RHI that was 23% higher at 2 minutes compared to the RHI at 1 minute ($P < 0.03$) (Fig.1). Baseline values were normalized to 1. Following forearm occlusion, post-occlusion RHI at 2, 3, 4, and 5 minutes was significantly higher by 36%, 27%, 27%, and 18%, respectively, compared to the RHI at 1 minute. No significant differences in RHI were observed at any time point between arm and forearm occlusion. Additionally, RH was sustained beyond 5 minutes in the current study. The apparent cessation of hyperemia at 3, 4, and 5 minutes after arm occlusion release may reflect the elevated mean RHI at 1 minute, which served as the comparison baseline. Relative to baseline values, PTT increased significantly at 1 and 2 minutes after arm occlusion release (by 6% and 4%, respectively) (Fig. 2). Following forearm occlusion release, PTT was 5% higher than baseline only at 1 minute. No significant differences were observed between arm and forearm baseline PTT values (260.9 ± 19.1 msec and 263.6 ± 19.7 msec, respectively), nor at 1- or 2-minutes post-occlusion release.
In conclusion, vascular stiffness (compliance), as indexed by PTT changes, improved similarly at both sites but persisted only up to 2 minutes.

Table 1: The characteristics of the participants recruited in the present study

N = 10	
Age (years)	23.1±5.0 (18-31)
BMI (Kg/m^2)	29.0±5.8 (23.1-41.2)
SBP (mm Hg)	132.5±7.0 (122-146)
DBP (mm Hg)	77.5±9.5 (64-93)
MBP (mm Hg)	95.8±7.4 (87.3-106.3)
Heart rate (beat/min)	74.9±12.1(59-94)

BMI = Body mass index. SBP, DBP, & MBP = Systolic, Diastolic, & Mean Blood Pressure

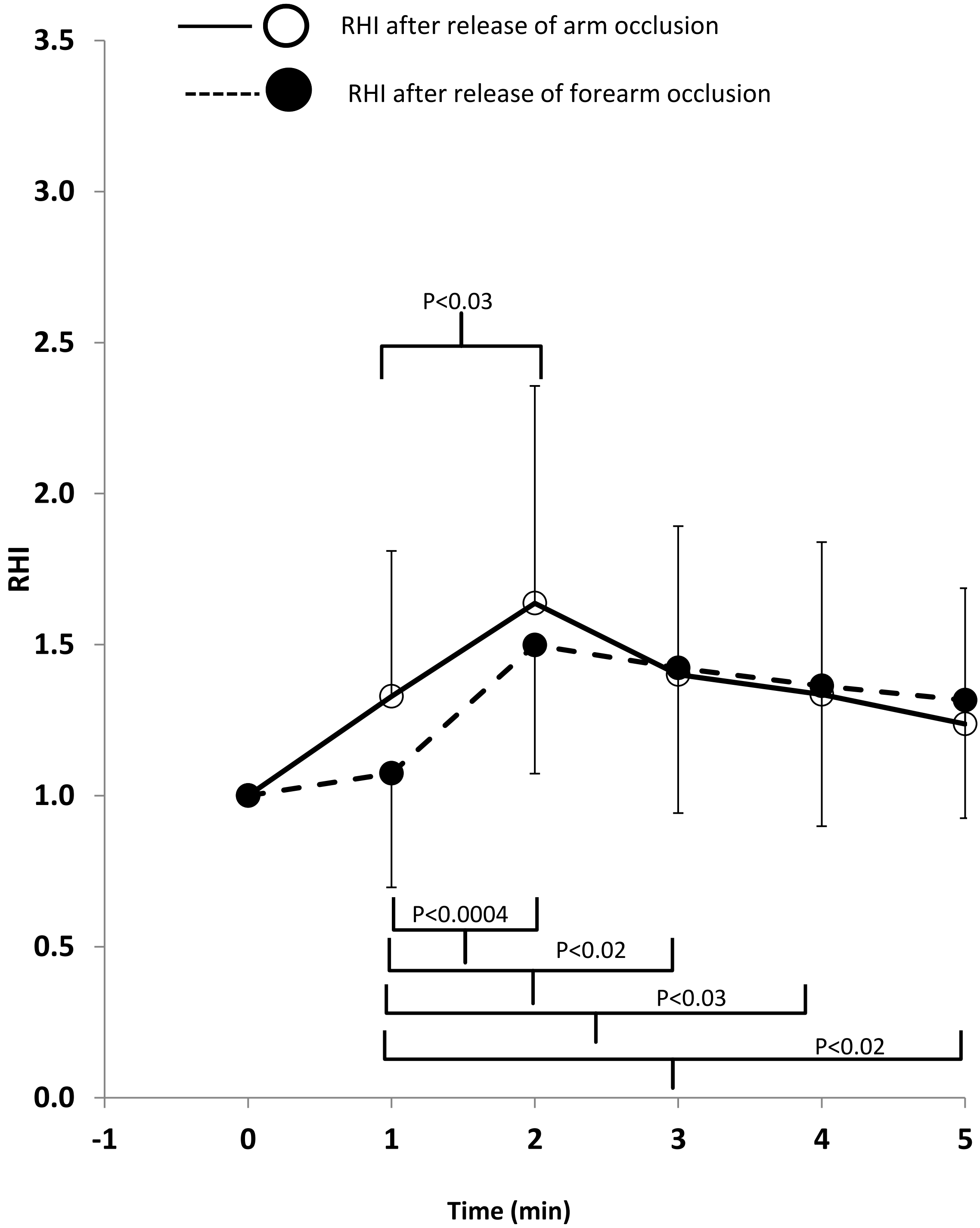

Fig.1: *Reactive hyperemia index (RHI) following the arm and forearm post-occlusion release. N = 10. Time "0" represents baseline values.*

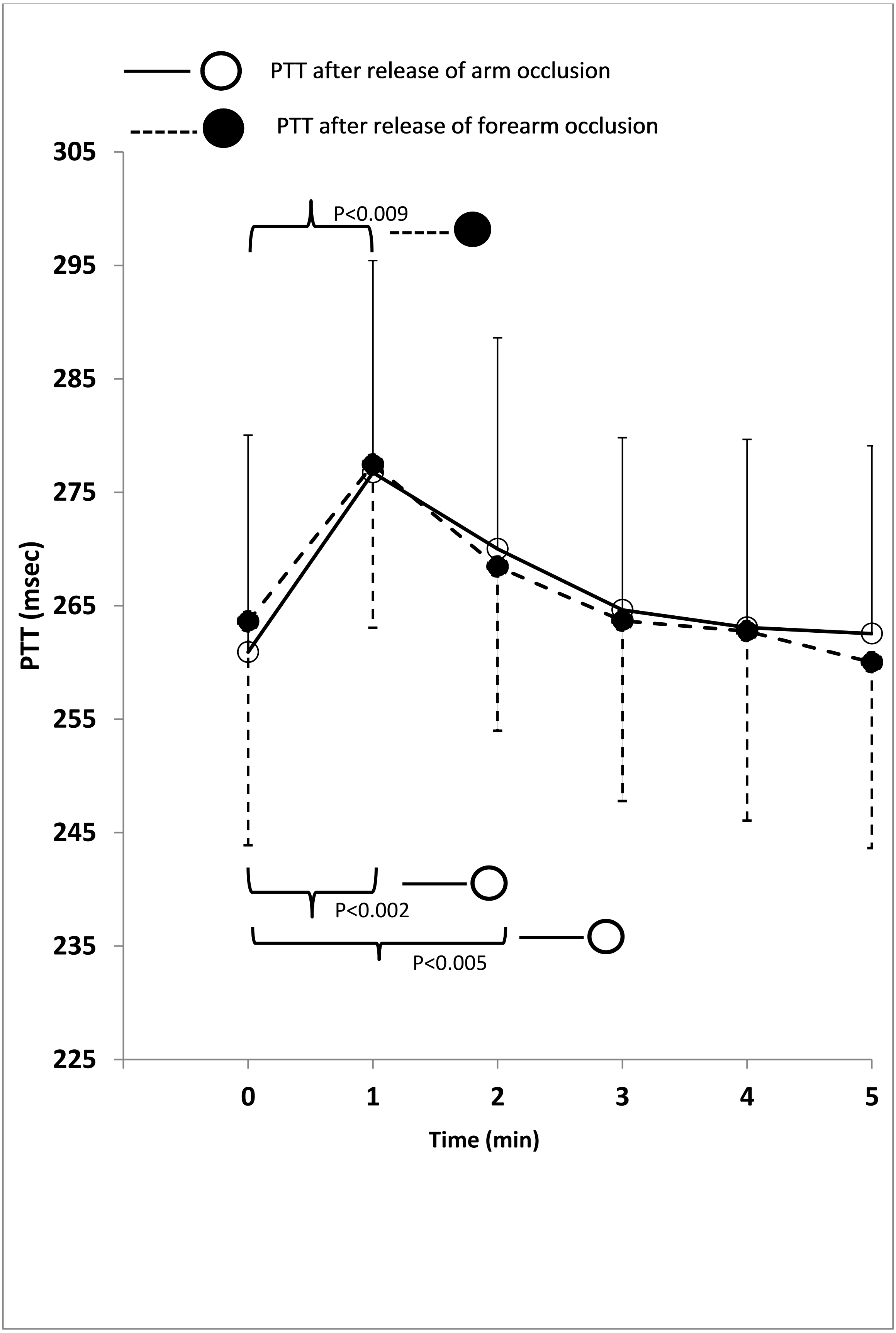

Fig. 2*: Pulse transit time (PTT) following the arm and forearm post-occlusion release. N = 10. Time "0" represents baseline values*

Discussion

RH reflects endothelial function, mainly mediated by NO [13]. Impaired NO activity contributes to atherosclerosis and cardiovascular risk. The current study found no significant difference in RHI between 5 min forearm and 5 min arm occlusions, consistent with others [14]. All subjects reported reduced discomfort after forearm occlusion compared to upper-arm occlusion. Peak RHI after occlusion release paralleled findings in previous publications [15, 16], with maximal RH observed at 2 minutes after occlusion release. RH measured by PPG amplitude also peaked at 2 minutes post-arm occlusion [1], aligning with our data. The progressive recovery of the DPW amplitude by the third minute may reflect metabolite washout during later hyperemia [1]. A contrasting time course was reported by [17]. showing peak RH at 1 minute. This discrepancy may stem from participant age differences (23 vs. 55 years). Forearm occlusion elicited more sustained RH than arm occlusion (Figure 1). This could relate to reduced sympathetic activation due to less discomfort during forearm occlusion [14, 18]. Alternatively, greater muscle mass ischemia during arm occlusion may increase metabolite accumulation, activating metaboreceptor (chemosensitive) afferents and sympathetic responses [18]. PTT responses were nearly identical between occlusion sites (Figure 2). Post-release PTT increases reflect transient stiffness reduction due to hyperemia, as blood flow restriction abolishes this effect. RH-triggered NO release increases shear stress, dilating conduit arteries [19].

Conclusion

Peak RH occurred at 2 minutes post-release for both occlusion sites. Forearm RH persisted longer (up to 5 minutes) than arm RH, although magnitudes were similar. Peak PTT, indicating maximal compliance, occurred at 1-minute post-release for both sites and persisted until 2 minutes for arm RH.

References

1. Selvaraj, N., Jaryal, A. K., Santhosh, J., & Deepak, K. K. (2009). Monitoring of reactive hyperemia using photoplethysmographic pulse amplitude and transit time. Journal of Clinical Monitoring and Computing, 23(6), 315-322.
2. Pyke, K. E., & Tschakovsky, M. E. (2007). Peak vs. total reactive hyperemia: Which determines the magnitude of flow-mediated dilation? Journal of Applied Physiology, 102, 1510–1519.
3. Guyton, A. C., & Hall, J. E. (2006). Textbook of medical physiology (11th ed.). Saunders.
4. Laurent, S., Cockcroft, J., Van Bortel, L., Boutouyrie, P., Giannattasio, C., Hayoz, D., Pannier, B. Vlachopoulos, C., Wilkinson, I., & Struijker-Boudier, H. (2006). Expert consensus document on arterial stiffness: Methodological issues and clinical applications. European Heart Journal, 27(21), 2588-2605.
5. Mitchell, G. F., Parise, H., Benjamin, E. J., Larson, M. G., Keyes, M. J., Vita, J. A., & Vasan, R. S. (2004). Changes in arterial stiffness and wave reflection with advancing age in healthy men and women: The Framingham Heart Study. Hypertension, 43(6), 1239-1245.
6. Tanaka, H., Munakata, M., Kawano, Y., Ohishi, M., Shoji, T., Sugawara, J., & Ozawa, T. (2009). Comparison between carotid–femoral and brachial–ankle pulse wave velocity as measures of arterial stiffness. Journal of Hypertension, 27(10), 2022-2027.

7. Vaitkevicius, P. V., Fleg, J. L., Engel, J. H., O'Connor, F. C., Wright, J. G., Lakatta, L. E., & Lakatta, E. G. (1993). Effects of age and aerobic capacity on arterial stiffness in healthy adults. Circulation, 88(4), 1456-1462.
8. Nohria, A., Gerhard-Herman, M., Creager, M. A., & Hurley, S. (2006). Role of nitric oxide in the regulation of digital pulse volume amplitude in humans. Journal of Applied Physiology, 101(2), 545-548.
9. Pyke, K. E., & Tschakovsky, M. E. (2005). The relationship between shear stress and flow-mediated dilatation: Implications for the assessment of endothelial function. Journal of Physiology, 568(2), 357-369.
10. Naka, K. K., Tweddel, A. C., Parthimos, D., Henderson, A., Goodfellow, J., & Frenneaux, M. P. (2006). Flow-mediated changes in pulse wave velocity: A new clinical measure of endothelial function. European Heart Journal, 27(3), 302-309.
11. McCrea, C. E., Skulas-Ray, A. C., Chow, M., & West, S. G. (2012). Test–retest reliability of pulse amplitude tonometry measures of vascular endothelial function: Implications for clinical trial design. Vascular Medicine, 17(1), 29-36.
12. Adnan, S. K., Neda, F. S., & Al-Gailani, B. T. (2023). Vascular stiffness in cold pressor test hyper-reactors. Polish Journal of Medical Physics and Engineering, 29(3), 151-154.
13. Bonetti, P. O., Pumper, G. M., Higano, S. T., Holmes, D. R. Jr., Kuvin, J. T., & Lerman, A. (2004). Noninvasive identification of patients with early coronary atherosclerosis by assessment of digital reactive hyperemia. Journal of the American College of Cardiology, 44(12), 2137-2141.
14. Faizi, A. K., Kornmo, D. W., & Agewall, S. (2009). Evaluation of endothelial function using finger plethysmography. Clinical Physiology and Functional Imaging, 29, 372-375.
15. Zahedi, E., Jaafar, R., Mohd Ali, M. A., Mohamed, A. L., & Maskon, O. (2008). Finger photoplethysmogram pulse amplitude changes induced by flow-mediated dilation. Physiological Measurement, 29, 625-637.
16. Khare, P., Talwar, A., Chandran, D., Guleria, R., Jaryal, A. K., Kumar, G., Trivedi, A., & Deepak, K. K. (2016). Impaired systemic vascular reactivity & raised high-sensitivity C-reactive protein levels in chronic obstructive pulmonary disease. Indian Journal of Medical Research, 143, 205-212.
17. Kuznetsova, T., Szczesny, G., Thijs, L., Jozeau, D., D'hooge, J., & Staessen, J. A. (2011). Assessment of peripheral vascular function with photoplethysmographic pulse amplitude. ARTRES, 5, 58-64.
18. Bade, G., Chandran, D. S., Jaryal, A. K., Talwar, A., & Deepak, K. K. (2019). Contribution of systemic vascular reactivity to variability in pulse volume amplitude response during reactive hyperemia. European Journal of Applied Physiology, 119(3), 753-760.
19. Jackson, R., Lima, N. S., Sherman, S. R., & Clifford, P. S. (2023). Reduction in peripheral arterial stiffness after reactive hyperemia is dependent on increases in blood flow. Physiological Reports, 11, e15894.

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مؤشر احتقان الدم التفاعلي وتصلب الشرايين بعد انسداد الذراع والساعد: دراسة مقارنة

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احتقان الدم التفاعلي، زمن عبور النبض، تصلب الشرايين، صحة الأوعية الدموية، وموقع الانسداد.

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الخلاصة:

احتقان الدم التفاعلي (RH) هو زيادة مؤقتة في تدفق الدم بعد نقص التروية، ويصاحبه انخفاض في تصلب الشرايين، ويُستخدم كمؤشر على صحة الأوعية الدموية. يُقيَّم احتقان الشرايين، وهو عامل خطر رئيسي لأمراض القلب والأوعية الدموية، عادةً باستخدام زمن عبور النبض (PTT)، وهي طريقة غير جراحية. تبحث هذه الدراسة في تأثير موقع الانسداد (الذراع مقابل الساعد) على مقدار احتقان الدم التفاعلي وعلاقته بتصلب الأوعية الدموية. شارك في الدراسة عشرة ذكور أصحاء (23.1 ± 5.0 سنة). تم قياس زمن عبور النبض (PTT) وسعة النبض الرقمية (DPWA) قبل وبعد تحريض احتقان الدم التفاعلي عند خط الأساس، وعلى فترات دقيقة واحدة لمدة خمس دقائق بعد التحريض. أدى إغلاق الذراع إلى زيادة في مؤشر احتقان الدم التفاعلي (RHI) بعد الانسداد بنسبة 23% بعد دقيقتين (قيمة الاحتمال < 0.03). أدى انسداد الساعد إلى زيادة في RHI بنسبة 36% و27% و27% و18% عند الدقائق 2 و3 و4 و5 على التوالي، مقارنةً بالدقيقة الواحدة. لم تُعثر على فروق جوهرية في RHI بين انسداد الذراع والساعد، حيث استمر RH لمدة تصل إلى 5 دقائق بعد انسداد الساعد. زاد PTT بشكل ملحوظ بنسبة 6% و4% عند الدقيقة 1 والدقيقة 2 بعد فك انسداد الذراع. بعد انسداد الساعد، كان PTT أعلى بشكل ملحوظ (5%) من خط الأساس فقط عند الدقيقة 1 بعد فك الانسداد. حدث ذروة RHI في الدقيقة الثانية بعد فك الانسداد، مع استمرار RH في الساعد لفترة أطول (حتى 5 دقائق)، على الرغم من أن القيم كانت متشابهة. حدث ذروة PTT، مما يشير إلى أقصى انخفاض في تصلب الأوعية الدموية، في الدقيقة الأولى بعد فك الانسداد وقد يمتد إلى الدقيقة الثانية.