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ORIGINAL ARTICLE

Effects of a highly intensive clean and jerk exercise on blood pressure and arterial stiffness in experienced non-professional weight lifters

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Abstract

Purpose Weight lifting training has gained much popularity in recent years and is frequently used in non-professional and professional settings. However, little is known about the acute effects of a highly intensive weight lifting exercise (clean and jerk) on the hemodynamic reaction.

Methods 18 non-professional experienced weight lifters were recruited. Hemodynamic parameters were obtained and measured at baseline (T1), after warming up (T2), and after a highly intensive clean and jerk exercise (90–95% of personal best; T3). Further, 15 (T4), and 30 min (T5) post-exercise measurements were conducted. Evaluated parameters were heart rate (HR) (b/min), peripheral and central systolic and diastolic blood pressure (pSysBP, pDiaBP, cSysBP, cDiaBP) (mmHg), pulse wave velocity (PWV) (m/s), and double product (DP).

Results All hemodynamic values increased from T1 up to T3 with significantly higher values measured at T3 compared to T1 and T2. Values of measured parameters at T3 were as follows: HR: 94.4 ± 15.6 b/min, pSysBP: 147.1 ± 15.9 mmHg, pDiaBP: 87.4 ± 12.2 mmHg, cSysBP: 129.3 ± 13.8 mmHg, cDiaBP: 89.9 ± 12.8 mmHg, and: 5.8 ± 0.5 m/s, DP: 14053 ± 3669. Post-exercise (T4, T5), all values returned to baseline levels.

Conclusions Results indicate that a highly intensive weight lifting exercise led to an acute increase of blood pressure and an acute stiffening of the arteries. Yet, increases were moderate and did not reach disproportionately high levels and returned to baseline levels within 15 min post-exercise. Hence, no negative acute effects of a maximum weight lifting exercise on the hemodynamic system are observed.

Keywords Arterial stiffness · Resistance training · Weight lifting · Clean and jerk · Blood pressure · Arterial compliance

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Abbreviations

ANOVA	Analyses of variance
AS	Arterial stiffness
BP	Blood pressure
cDiaBP	Central diastolic blood pressure
cSysBP	Central systolic blood pressure
DP	Double product
mmHg	Millimeter of mercury
m/s	Meter per second
NO	Nitric oxide
pDiaBP	Peripheral diastolic blood pressure
pSysBP	Peripheral systolic blood pressure
PWV	Pulse wave velocity
RT	Resistance training
WL	Weight lifting

Introduction

Hypertension is one of the leading diseases in the western world (ESH/ESC Task Force for the Management of Arterial Hypertension 2013) and there is evidence that the measurement of the central blood pressure and arterial stiffness is better related to future cardiovascular events compared to the conventional blood pressure (BP) measurement (McEniery et al. 2014; Karras et al. 2012).

Several studies have demonstrated that endurance training programs lead to decreased arterial stiffness, central and peripheral BP, an improved arterial compliance and, thus, a better vascular health (Ashor et al. 2014). However, the effects of acute and long-term resistance training (RT) on the vascular system are not fully understood (Braith and Stewart 2006). Cross-sectional studies demonstrated that resistance-trained men have stiffer arteries, higher peripheral BP and central BP compared to their sedentary age-matched peers (Miyachi et al. 2003; Bertovic et al. 1999). Also in high-performance sports, athletes performing strength-dominant sports show a higher arterial stiffness and higher BP values compared to athletes who perform endurance-dominated sports (Saka et al. 2016).

Weight lifting (WL) which is performed in recreational and non-professional leisure settings has gained increased popularity in recent years. Also, in other sport categories (e.g. volleyball and basketball (Holmberg 2013)), WL sessions are increasingly often implemented in regular training schedules, as it was shown to improve athleticism, e.g. vertical jump performance (Arabatzi et al. 2010; Otto et al. 2012). It was further suggested that athletes with experience in WL might also exhibit greater confidence and aggressiveness in their approach to competitive sports and that these athletes possess a more complete understanding of athletic lifestyle (Takano 2013).

However, little is known about the acute effects of a WL regimen on the hemodynamic system. Therefore, this study aims to evaluate the acute effects of WL on peripheral and central BP and arterial stiffness in experienced, non-professional weight lifters.

Methods

Study participants

18 subjects (16 male, 2 female) participated in this study. To be included in the study, participants had to fulfill the following criteria: active in non-professional WL for at least 2 years, being able to conduct a one-time clean and jerk maneuver with a weight that equals 100% of the body weight in proper form, a regular resistance/WL training load of at least three sessions per week, non-smoker, age 20–30 years, no regular consumption of any drugs, no cardiovascular disease conditions such as heart failure, ischemic cardiopathy, and further diseases such as renal insufficiency or diabetes mellitus. Consumption of caffeine and alcohol was prohibited 24 h prior to the testing. The study and the protocols used were approved by the ethics committee of the German Sport University Cologne. These protocols are in line with the Declaration of Helsinki. Participants gave written informed consent to participate in the study. Characteristics of the study participants are displayed in Table 1.

Study design

All measurements and exercises were conducted in a gym in an extra room to provide a quiet and secluded environment at the same time of the day (between 8 and 11 a.m.). A schematic overview of the study design can be found in Fig. 1. On the day of the test, recruited participants were asked to remain calm in a sitting position for 10 min. In this time, participants filled out a questionnaire about their training history. This information is provided in Table 1. Having sat for 10 min, the first measurement of the hemodynamic parameters was conducted in a seated position (T1). This hemodynamic measurement and the measurements that followed at all other time points took approximately 60 s with marginal deviation. Then participants conducted a submaximal warm-up program for 20 min with a bar (20-kg Eleiko bar, Eleiko, AB, Halmstad, Sweden) without and with additional disks (Eleiko Olympic disks, Eleiko, AB, Halmstad, Sweden). The warm-up routine started with only the bar (20 kg). Participants conducted three sets with five

Table 1 Anthropometric, training and performance characteristics of the participants

	Mean ± standard deviation
Men/women	16/2
Age (years)	24.2 ± 1.9
Height (cm)	179.8 ± 7.9
Weight (kg)	84.4 ± 11.3
Experience weight lifting (years)	2.1 ± 0.9
Regular training (session/week)	4.6 ± 0.9
Regular training volume (h/session)	2.3 ± 1.5
Specific weight lifting training (session/week)	2.4 ± 0.9
Specific weight lifting training volume (h/session)	1.6 ± 0.6
Max. clean and jerk (kg)	106.9 ± 21.5
Clean and jerk weight at T3 (kg)	99.2 ± 19.5

Data are presented as means ± standard deviation

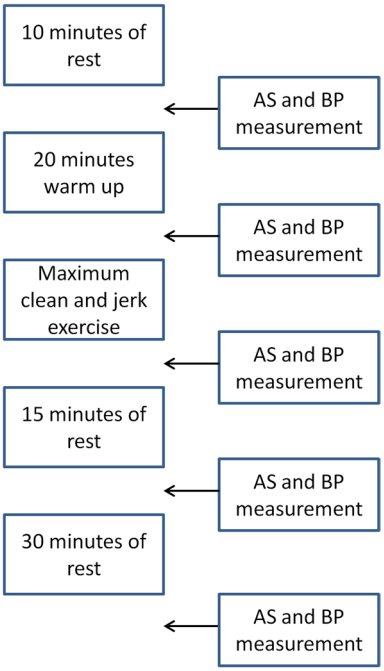

Fig. 1 Schematic overview of the weight lifting (clean and jerk) regimen. *AS* arterial stiffness, *BP* blood pressure

repetitions of hang power cleans, front squats and shoulder press with 1-min rest between the sets. After that, they did three sets with three repetitions of the full range of motion of the clean and jerk exercise. They started with 50% of their planned final clean and jerk weight and increased the weight 10–15% in each set. Between the sets, the pause was set to 2 min.

The second hemodynamic measurement started 15 s after finishing the last exercise of the warm-up routine in a seated position. The final weight was set to 90–95% of the individual's maximum clean and jerk weight which was reported by the participants in the above-mentioned questionnaire. Having completed the final clean and jerk exercise, the hemodynamic measurement started 15 s after the exercise was finished and participants were in a seated position (T3). Following this highly intensive clean and jerk exercise, participants were asked to remain quiet in a sitting position. Hemodynamic measurements were conducted 15 (T4) and 30 (T5) min post-exercise. During these measurements, participants were also in a seated position.

Measurement of arterial stiffness parameters

All measurements were performed using a Mobil-O-Graph device (IEM, Stollberg, Germany) with a novel transfer function-like algorithm, using brachial cuff-based waveform recordings. For central systolic pressure, calculation and other measures of arterial stiffness, an integrated transfer function (ARCSolver algorithm) was used (Weber et al. 2011; Wassertheurer et al. 2010). First, the device measures the peripheral BP. After 30 s of pause, the device measures further hemodynamic parameters which are presented in the software output. The parameters that were evaluated were heart rate [HR (b/min)], systolic [pSysBP (mmHg)] and diastolic peripheral BP [pSysBP (mmHg)], systolic [cSysBP (mmHg)] and diastolic central BP [cSysBP (mmHg)] and pulse wave velocity [PWV (m/s)]. PWV was shown to be the most reliable measure of arterial stiffness (O'Rourke et al. 2002; Kim et al. 2007). Compared to the invasive measurement of the central systolic pressure and established radial tonometry with inbuilt generalized transfer function, the ARCSolver algorithm showed good agreement (Weber et al. 2011; Wassertheurer et al. 2010). Further, the double product (DP, also known as rate pressure product) was calculated by multiplication of pSysBP and HR (Fletcher et al. 1979) and serves as valid predictor of myocardial workload and myocardial oxygen consumption during exercise (Picón et al. 2018). All hemodynamic measurements were conducted following the recommendations of the ESH/ESC Task Force for the Management of Arterial Hypertension (ESH/ESC Task Force for the Management of Arterial Hypertension 2013). Hence, participants were at rest in a sitting position in a quiet room with adequate temperature. Cuffs were chosen depending on upper-arm circumference, and the cuffs were at the same level of the heart during all measurements.

Data analyses

All descriptive data are expressed as means ± standard deviation. After checking for normality of distribution using the Shapiro–Wilk test, a one-way repeated measured analysis of variance (ANOVA) was conducted to evaluate the effects of a maximum WL regimen on the different hemodynamic measures when measured before (T1), after the warm-up (T2), immediately after the final weight lift (T3), 15 min after the final weight lift (T4) and 30 min after the final weight lift (T5). If a significant time effect was observed, Bonferroni post hoc analyses were conducted to detect significant differences between time points.

Results

The employed weight for the clean and jerk exercise at T3 was a mean of 99.2 kg (± 19.5) which equaled 92.9% (± 3.1) of the participant's maximum clean and jerk performance and 117.1% (± 15.4) of the participant's body weight. Mean values with standard deviation of the measured hemodynamic parameters are illustrated in Fig. 2 and exact *p* values of the pairwise comparisons of the statistical analyses are provided as supplementary material 1.

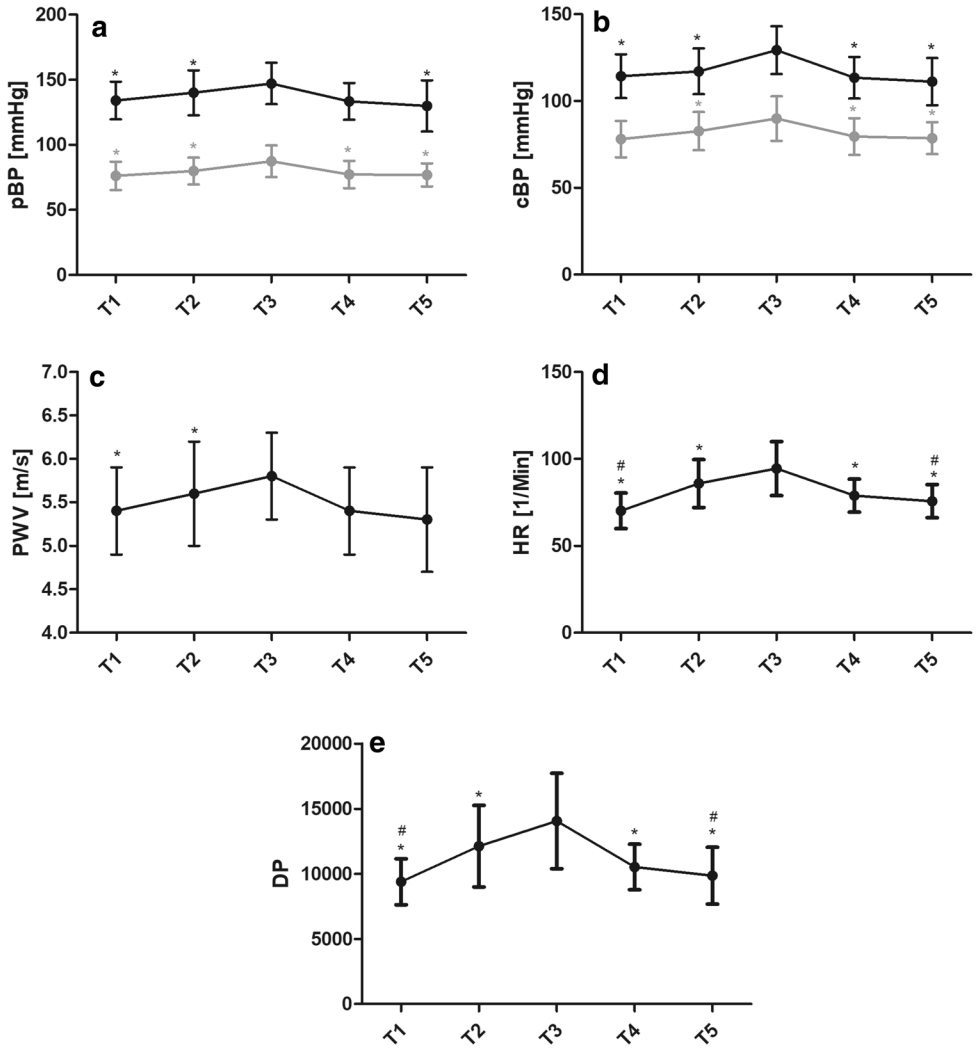

Fig. 2 **a** Peripheral blood pressure (pBP) measured throughout the training regimen. Black lines indicate systolic peripheral blood pressure and gray lines indicate diastolic peripheral blood pressure. Data are presented as means with ± standard deviation. *$p < 0.05$ vs. T3. **b** Central blood pressure (cBP) measured throughout the training regimen. Black lines indicate systolic central blood pressure and gray lines indicate diastolic central blood pressure. Data are presented as means with ± standard deviation. *$p < 0.05$ vs. T3. **c** Pulse wave velocity (PWV) measured throughout the training regimen. Data are presented as means with ± standard deviation. *$p < 0.05$ vs. T3. **d** Heart rate (HR) measured throughout the training regimen. Data are presented as means with ± standard deviation. *$p < 0.05$ vs. T3, #$p < 0.05$ vs. T2. **e** Double product (DP) measured throughout the training regimen. Data are presented as means with ± standard deviation. *$p < 0.05$ vs. T3, #$p < 0.05$ vs. T2

Measured HR (b/min) values were as follows: T1: 70.1 (± 10.1); T2: 85.8 (± 13.8); T3: 94.4 (± 15.6); T4: 78.8 (± 9.5); and T5: 75.6 (± 9.5). Statistical analyses revealed a time effect for HR ($p \leq 0.001$) and post hoc analyses showed that HR measured at T3 differed significantly from all other time points. Further, HR measured at T2 differed significantly from T1 and T5. No further differences were detected between any other time points. Statistical analyses revealed a time effect for pSysBP ($p = 0.003$) and post hoc analyses showed that pSysBP measured at T3 differed significantly from T1, T2 and T5, respectively. The other pairwise comparisons revealed no further significant differences. Measured pSysBP [mmHg] values were as follows: T1: 134.1 (± 14.5); T2: 140.0 (± 17.2); T3: 147.1 (± 15.9); T4: 133.4 (± 14.1); and T5: 129.9 (± 19.6). Statistical analyses revealed a time effect for pDiaBP ($p < 0.001$) and post hoc analyses showed that pDiaBP measured at T3 differed significantly from all other time points. No further significant differences between time points were observed. pDiaBP [mmHg] values were T1: 76.2 (± 10.9); T2: 79.9 (± 10.4); T3: 87.4 (± 12.2); T4: 77.3 (± 10.5); and T5: 76.9 (± 8.9). Statistical analyses revealed a time effect for cSysBP ($p < 0.001$) and post hoc analyses showed that cSysBP measured at T3 differed significantly from all other

time points. Pairwise comparisons of the other time points did not reveal any further significant differences. Measured cSysBP [mmHg] values were as follows: T1: 114.3 (± 12.6); T2: 117.1 (± 13.2); T3: 129.3 (± 13.8); T4: 113.4 (± 12.0); and T5: 111.2 (± 13.6). Statistical analyses revealed a time effect for cDiaBP ($p = 0.005$) and post hoc analyses showed that cDiaBP measured at T3 differed significantly from all other time points, while no further significant difference was detected between other time points. Exact cDiaBP [mmHg] values were T1: 78.1 (± 10.5); T2: 82.7 (± 11.0); T3: 89.9 (± 12.8); T4: 79.5 (± 10.6); and T5: 78.6 (± 9.2). Statistical analyses revealed a time effect for PWV ($p = 0.005$) and post hoc analyses showed that PWV measured at T3 differed significantly from T1 and T2, respectively. No further significant differences between time points were observed. Measured PWV [m/s] values for each time point were as follows: T1: 5.4 (± 0.5); T2: 5.6 (± 0.6); T3: 5.8 (± 0.5); T4: 5.4 (± 0.4); and T5: 5.3 (± 0.6). Measured DP values were as follows: T1: 9394 (± 1770); T2: 12,122 (± 3130); T3: 14,053 (± 3669); T4: 10,532 (± 1750); and T5: 9861 (± 2185). Statistical analyses revealed a time effect for DP ($p \leq 0.001$) and post hoc analyses showed that DP measured at T3 differed significantly from all other time points. Further, T2 differed significantly from T1 and T5. No further difference was detected between other time points.

Discussion

To the best of our knowledge, this is the first study presenting the hemodynamic effects of an acute highly intensive Olympic WL exercise (clean and jerk) in WL-experienced non-professional participants. Due to trending sports such as CrossFit® that use WL elements, WL has become more and more popular since the last half of the twentieth century. WL is a whole body strength training and WL movements are far more complex and much more intensive compared to conventional resistance training (Häkkinen et al. 1987). Hence, results from studies examining the effects of conventional RT on arterial stiffness might be different.

In the present study, BP, HR, DP, and arterial stiffness increased from baseline values (T1) throughout the warm-up phase (T2) and reached highest values after the clean and jerk lift (T3). This is observed for PWV, pSysBP, pDiaBP, cSysBP, cDiaBP, HR, and DP with significantly higher values measured at T3 in all these parameters compared to T1 and T2. This increase in aortic and peripheral BP and arterial stiffness was observed in previous studies following RT programs (DeVan et al. 2005; Kingsley et al. 2016; Yoon et al. 2010).

Systolic BP values measured immediately after the highly intensive clean and jerk lift are not dangerously high with a mean pSysBP of 147.1 mmHg (± 15.9) and mean cSysBP of 129.3 mmHg (± 13.8). These mild increases are in line with the moderate increases of a mean HR of 94.4 b/min (± 15.6) and a mean DP of 14,053 (± 3669). Also, PWV values do not increase much with a mean of 5.8 m/s (± 0.5). Of note is the fact that the measurements started 15 s after finishing the exercise and lasted for 60 s. Hence, the measured parameters were recorded approximately 75 s after finishing the exercise. Therefore, this recovery time of the cardiac and hemodynamic system needs to be considered. A further explanation for this moderate increase might be that the study participants only conducted one very intensive clean and jerk lift. It was previously shown by Nitzsche et al. that acute resistance training regimens with higher volumes and lower intensity lead to a higher vascular response. This response was marked by a greater increase of central BP and a stronger stiffening of the arteries. In the group that performed the low-intensity and high-volume RT, cSysBP increased to 133.4 mmHg (± 17.2) and PWV increased to 5.97 m/s (± 0.65). In contrast, in the high-intensity and low-volume group, cSysBP increased to only 118.1 mmHg (± 12.7) and PWV increased to 5.48 m/s (± 0.5) (Nitzsche et al. 2016). This study group used the same device to measure hemodynamic values as the present study (Mobil-O-Graph, IEM, Stollberg, Germany). However, in the present study, participants conducted a warm-up that included several repetitions with lower weight. Yet, the comparison of the values of the above-mentioned study indicates that the acute increase of the maximum clean and jerk exercise is most likely lower than a low-intensity and high-volume RT. The very high intensity of the exercise probably led to very high increases of NO, as it was shown that a high-intensity RT goes along with higher NO levels compared to low-intensity RT leading to a more moderate rise in BP. Higher blood flow and sheer stress to the endothelium during intensive exercise activate the endothelial NO synthase and in turn the release of NO leading to vasodilatation (Güzel et al. 2007). This might be one reason why the increases in BP are in a moderate range compared to results of other studies that used more repetitions and lower weights (Nitzsche et al. 2016).

Central and peripheral diastolic BP values also increased and were significantly higher at T3 compared to T1 and T2. The reasons for this increase in diastolic BP remain elusive as studies show that the diastolic BP usually remains unaffected or slightly decreases after an acute RT (Carpio-Rivera et al. 2016; Wickwire et al. 2009). It can only be speculated that the high absolute muscle activation primarily in the lower body might lead to a higher restriction in the arterial vascular bed and thus to a higher diastolic BP. It was previously observed that an acute lower body resistance training led to significantly higher central and peripheral diastolic BP increases compared to an upper body resistance training (Tomschi et al. 2018). However, it was also shown that a

lower body RT did lead to altered peripheral diastolic BP responses compared to an upper body RT (Li et al. 2015). In the present study, 15 (T4) and 30 min (T5) after the exercise, pDiaBP and cDiaBP approach the pre-values with significantly lower values compared to T3. This decrease after the acute exercise was also observed in the aforementioned study (Tomschi et al. 2018). This allows the conclusion that no negative time-delayed effects occur after the maximum clean and jerk lift.

The increase of the measured hemodynamic parameters seems to be only transient as they decrease to baseline levels 15 (T4) and 30 (T5) minutes after the exercise. This decrease to baseline levels after finishing physical exercises was also observed in other studies (Tomschi et al. 2018; Müller et al. 2015; Ketelhut et al. 2016). Some studies also report that BP levels decrease even under baseline values which is observed in normotensive and hypertensive subjects (Senna et al. 2018; Kenney and Seals 1993). This "post exercise hypotension" phenomenon (Chen and Bonham 2010) is probably caused by several complex mechanisms including the sympathetic nervous system, baroreceptors, the release of nitric oxide, the decline of HR, and other yet-to-be explored mechanisms. In the present study, the participants' HR and DP also decrease along with BP values but do not fall below baseline values. Besides, BP and arterial stiffness values measured up to 30 min post-exercise do not significantly fall below the baseline values. The above-mentioned study of Müller et al. showed that central and peripheral systolic BP and arterial stiffness decreased under the values measured before the sub-maximal 3000 m run (Müller et al. 2015). Furthermore, a marathon run led to significant post-exercise decreases in peripheral and central systolic BP but no change in arterial stiffness (Vlachopoulos et al. 2010). Simão et al. reported that resistance training acutely resulted in hypotensive effects as the systolic BP was decreased up to 40 min after a 6RM session, a conventional 12 repetition session, and a circuit RT session (Simão et al. 2005).

The participants' baseline pSysBP values are slightly elevated in the present study, even though the measurement took place when participants were in a calm mode and had sat for 10 min. Cross-sectional studies report that strength -trained athletes possess a slightly elevated blood pressure and have a higher prevalence of hypertension than other athletes (e.g. endurance-trained athletes) (Berge et al. 2015).

This study has some limitations that are of note. Volume changes due to sweating were not evaluated which might have influenced BP. Further, subjects were allowed to drink after the maximum clean and jerk exercise (T3). The measurement method used in this study does not allow an analysis during dynamic work. The measurement time of BP and arterial stiffness took about 60 s and a measurement during the weight lifting exercise was, therefore, not possible. Hence, it is important to note that the extent of the values of the examined parameters does not reflect the exact value during dynamic work. Measurements with a catheter would strongly affect performance, safety and motivation of the participants. Therefore, the employed non-invasive method is a suitable way to quantify BP and arterial stiffness immediately after the physical exercise.

Conclusion

The results of the present study show that a WL regimen with a warm-up phase and a high-intensity clean and jerk exercise (90–95% of participant's personal best) does not lead to disproportionately high acute increases in HR, DP, peripheral and central BP or a strong stiffening of the arteries in experienced non-professional weight lifters. Besides, no delayed negative effects are observed up to 30 min following the maximum clean and jerk exercise as all measured hemodynamic parameters return to baseline levels 15 min post-exercise. Hence, no negative acute effects of such weight lifting can be reported. Yet, future studies should aim to evaluate the long-term effects of weight lifting interventions on the cardiovascular and hemodynamic system.

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Author contributions The contributions of the authors were as follows: FT and ER had the original idea for the study. FT, HO, ER, EI contributed to conception or design of the study and analysis or interpretation of the data of the present study. FT and ER drafted the manuscript and HO, EI, WB and MG critically revised the manuscript for important intellectual content.

Compliance with ethical standards

Conflict of interest The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

References

Arabatzi F, Kellis E, Saèz-Saez De Villarreal E (2010) Vertical jump biomechanics after plyometric, weight lifting, and combined (weight lifting + plyometric) training. J Strength Cond Res 24(9):2440–2448. https://doi.org/10.1519/JSC.0b013e3181e274ab

Ashor AW, Lara J, Siervo M, Celis-Morales C, Mathers JC (2014) Effects of exercise modalities on arterial stiffness and wave reflection: a systematic review and meta-analysis of randomized controlled trials. PloS One 9(10):e110034. https://doi.org/10.1371/journal.pone.0110034

Berge HM, Isern CB, Berge E (2015) Blood pressure and hypertension in athletes: a systematic review. Br J Sports Med 49(11):716–723. https://doi.org/10.1136/bjsports-2014-093976

Bertovic DA, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA (1999) Muscular strength training is associated with

low arterial compliance and high pulse pressure. Hypertension 33(6):1385–1391. https://doi.org/10.1161/01.HYP.33.6.1385
Braith RW, Stewart KJ (2006) Resistance exercise training: its role in the prevention of cardiovascular disease. Circulation 113(22):2642–2650. https://doi.org/10.1161/CIRCULATIONAHA.105.584060
Carpio-Rivera E, Moncada-Jiménez J, Salazar-Rojas W, Solera-Herrera A (2016) Acute effects of exercise on blood pressure: a meta-analytic investigation. Arq Bras Cardiol 106(5):422–433. https://doi.org/10.5935/abc.20160064
Chen C-Y, Bonham AC (2010) Postexercise hypotension: central mechanisms. Exerc Sport Sci Rev 38(3):122–127. https://doi.org/10.1097/JES.0b013e3181e372b5
DeVan AE, Anton MM, Cook JN, Neidre DB, Cortez-Cooper MY, Tanaka H (2005) Acute effects of resistance exercise on arterial compliance. J Appl Physio (Bethesda Md 1985) 98(6):2287–2291. https://doi.org/10.1152/japplphysiol.00002.2005
ESH/ESC Task Force for the Management of Arterial Hypertension (2013) 2013 Practice guidelines for the management of arterial hypertension of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC): ESH/ESC Task Force for the Management of Arterial Hypertension. J Hypertens 31(10):1925–1938. https://doi.org/10.1097/HJH.0b013e328364ca4c
Fletcher GF, Cantwell JD, Watt EW (1979) Oxygen consumption and hemodynamic response of exercises used in training of patients with recent myocardial infarction. Circulation 60(1):140–144
Güzel NA, Hazar S, Erbas D (2007) Effects of different resistance exercise protocols on nitric oxide, lipid peroxidation and creatine kinase activity in sedentary males. J Sports Sci Med 6(4):417–422
Häkkinen K, Komi PV, Alén M, Kauhanen H (1987) EMG, muscle fibre and force production characteristics during a 1 year training period in elite weight-lifters. Eur J Appl Physiol Occup Physiol 56(4):419–427
Holmberg PM (2013) Weightlifting to improve volleyball performance. Strength Cond J 35(2):79–88. https://doi.org/10.1519/SSC.0b013e3182889f47
Karras A, Haymann J-P, Bozec E, Metzger M, Jacquot C, Maruani G, Houillier P, Froissart M, Stengel B, Guardiola P, Laurent S, Boutouyrie P, Briet M (2012) Large artery stiffening and remodeling are independently associated with all-cause mortality and cardiovascular events in chronic kidney disease. Hypertension (Dallas Tex 1979) 60(6):1451–1457. https://doi.org/10.1161/HYPERTENSIONAHA.112.197210
Kenney MJ, Seals DR (1993) Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertension (Dallas Tex 1979) 22(5):653–664
Ketelhut S, Milatz F, Heise W, Ketelhut RG (2016) Influence of a high-intensity interval training session on peripheral and central blood pressure at rest and during stress testing in healthy individuals. VASA 45(5):373–377. https://doi.org/10.1024/0301-1526/a000560
Kim EJ, Park CG, Park JS, Suh SY, Choi CU, Kim JW, Kim SH, Lim HE, Rha SW, Seo HS, Oh DJ (2007) Relationship between blood pressure parameters and pulse wave velocity in normotensive and hypertensive subjects: invasive study. J Hum Hypertens 21(2):141–148. https://doi.org/10.1038/sj.jhh.1002120
Kingsley JD, Mayo X, Tai YL, Fennell C (2016) Arterial stiffness and autonomic modulation after free-weight resistance exercises in resistance trained individuals. J Strength Cond Res 30(12):3373–3380. https://doi.org/10.1519/JSC.0000000000001461
Li Y, Bopp M, Botta F, Nussbaumer M, Schäfer J, Roth R, Schmidt-Trucksäss A, Hanssen H (2015) Lower body vs. upper body resistance training and arterial stiffness in young men. Int J Sports Med 36(12):960–967. https://doi.org/10.1055/s-0035-1549921
McEniery CM, Cockcroft JR, Roman MJ, Franklin SS, Wilkinson IB (2014) Central blood pressure: current evidence and clinical importance. Eur Heart J 35(26):1719–1725. https://doi.org/10.1093/eurheartj/eht565
Miyachi M, Donato AJ, Yamamoto K, Takahashi K, Gates PE, Moreau KL, Tanaka H (2003) Greater age-related reductions in central arterial compliance in resistance-trained men. Hypertension (Dallas Tex 1979) 41(1):130–135
Müller J, Wilms M, Oberhoffer R (2015) Acute effects of submaximal endurance training on arterial stiffness in healthy middle- and long-distance runners. J Clin Hypertens 17(5):371–374. https://doi.org/10.1111/jch.12530
Nitzsche N, Weigert M, Baumgärtel L, Auerbach T, Schuffenhauer D, Nitzsche R, Schulz H (2016) Acute effects of different strength training protocols on arterial stiffness in healthy subjects. Int J Sports Sci 6(5):195–202. https://doi.org/10.5923/j.sports.20160605.05
O'Rourke MF, Staessen JA, Vlachopoulos C, Duprez D, Plante GE (2002) Clinical applications of arterial stiffness; definitions and reference values. Am J Hypertens 15(5):426–444
Otto WH, Coburn JW, Brown LE, Spiering BA (2012) Effects of weightlifting vs. kettlebell training on vertical jump, strength, and body composition. J Strength Cond Res 26(5):1199–1202. https://doi.org/10.1519/JSC.0b013e31824f233e
Picón MM, Chulvi IM, Cortell J-MT, Tortosa J, Alkhadar Y, Sanchís J, Laurentino G (2018) Acute cardiovascular responses after a single bout of blood flow restriction training. Int J Exerc Sci 11(2):20–31
Saka T, Sekir U, Dogan A, Akkurt S, Karakus M, Celebi MM, Sarli B, Oguzhan A (2016) Arterial stiffness differences between aerobically and resistance trained turkish elite athletes. Br J Sports Med 50(Suppl 1):A40.2–A40. https://doi.org/10.1136/bjsports-2016-097120.69
Senna GW, Scudese E, Silva-Grigoletto MED, Alias A, Fuqua JD, Brandão PP, Dantas EHM (2018) Post resistance exercise hypotension on distinct types of somatotype characteristics. J Hum Sport Exerc. https://doi.org/10.14198/jhse.2018.131.05
Simão R, Fleck SJ, Polito M, Monteiro W, Farinatti P (2005) Effects of resistance training intensity, volume, and session format on the postexercise hypotensive response. J Strength Cond Res 19(4):853–858. https://doi.org/10.1519/R-16494.1
Takano RK (2013) Weightlifting in the development of the high school athlete. Strength Cond J 35(6):66–72. https://doi.org/10.1519/SSC.0000000000000012
Tomschi F, Köster P, Predel H-G, Lay D, Bloch W, Grau M (2018) Acute effects of lower and upper body-resistance training on arterial stiffness, peripheral, and central blood pressure in young normotensive women. Sport Sci Health 14(2):357–363. https://doi.org/10.1007/s11332-018-0440-7
Vlachopoulos C, Kardara D, Anastasakis A, Baou K, Terentes-Printzios D, Tousoulis D, Stefanadis C (2010) Arterial stiffness and wave reflections in marathon runners. Am J Hypertens 23(9):974–979. https://doi.org/10.1038/ajh.2010.99
Wassertheurer S, Kropf J, Weber T, van der Giet M, Baulmann J, Ammer M, Hametner B, Mayer CC, Eber B, Magometschnigg D (2010) A new oscillometric method for pulse wave analysis: comparison with a common tonometric method. J Hum Hypertens 24(8):498–504. https://doi.org/10.1038/jhh.2010.27
Weber T, Wassertheurer S, Rammer M, Maurer E, Hametner B, Mayer CC, Kropf J, Eber B (2011) Validation of a brachial cuff-based method for estimating central systolic blood pressure. Hypertension (Dallas Tex 1979) 58(5):825–832. https://doi.org/10.1161/HYPERTENSIONAHA.111.176313
Wickwire PJ, McLester JR, Green JM, Crews TR (2009) Acute heart rate, blood pressure, and RPE responses during super slow vs. traditional machine resistance training protocols using small muscle

group exercises. J Strength Cond Res 23(1):72–79. https://doi.org/10.1519/JSC.0b013e3181854b15

Yoon ES, Jung SJ, Cheun SK, Oh YS, Kim SH, Jae SY (2010) Effects of acute resistance exercise on arterial stiffness in young men. Korean Circ J 40(1):16–22. https://doi.org/10.4070/kcj.2010.40.1.16