

# Acute Effects of Different Strength Training Protocols on Arterial Stiffness in Healthy Subjects

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**Abstract** Strength training plays an important role in cardiovascular prevention and rehabilitation. Studies showed inconsistent chronic and acute effects of strength training on arterial stiffness as an emerging biomarker of vascular health. Using pulse wave analysis, the arterial stiffness can be quantified by calculating the pulse wave velocity (PWV). The present study compared acute effects of three strength training protocols on arterial stiffness. 41 healthy, physically active subjects (age  $23.8 \pm 2.3$  yr, height  $1.78 \pm 0.1$  m, body weight  $72.9 \pm 9.0$  kg, body mass index  $22.9 \pm 2.0$  kg/m<sup>2</sup>) were assigned to three groups: group 1: 30% one repetition maximum (1RM), 3x30 repetitions, group 2: 50% 1RM 3x20 repetitions, group 3: 70% 1RM, 4x10 repetitions. All groups completed a resistance exercise session with five dynamic exercises. Pulse wave velocity (PWV), central diastolic (cDBP) and central systolic blood pressure (cSBP) were measured at rest, 0, 5 and 10 minutes after the training session with a pulse wave analysis system. PWV and cSBP showed an increase after resistance training in group 1 and 2 ( $p < 0.05$ ) but not in group 3 ( $p > 0.05$ ). cDBP decreased in all groups 5 and 10 minutes after training compared to 0 minutes after training. These results indicate that resistance exercise with low, moderate and high intensities reduce cDBP 5 and 10 minutes after a training session, but only those protocols with lower load and more repetitions acutely increase arterial stiffness in healthy subjects.

**Keywords** Pulse wave velocity, Central blood pressure, Arterial stiffness, Strength training, Training protocols

## 1. Introduction

Strength training is highly recommended for prevention and treatment of cardiovascular diseases [1–3]. Relevant positive effects of strength training on cardiovascular risk parameters were detected [4, 5].

A significant predictor of cardiovascular risk is the arterial stiffness [6, 7]. Using pulse wave analysis (PWA), the arterial stiffness can be quantified by calculating the pulse wave velocity (PWV).

The decreasing elasticity from central to peripheral arteries leads to an increase of PWV, systolic blood pressure (SBP) and of pulse pressure (PP) from central to peripheral arteries [7]. However, the central systolic blood pressure (cSBP) and the central pulse pressure (cPP) are better associated with end-organ damage than the SBP and PP measured oscillometrically at the upper arm [6, 8, 9]. Central PWV has proved to be a good predictor for cardiovascular events [10]. According to the recent consensus paper on arterial stiffness of the European Society of Hypertension and Cardiology, a PWV equal or

above 10 m/s is regarded as a manifested end-organ damage.

Studies that have investigated the chronic influence of several weeks of physical training on arterial stiffness have demonstrated the positive effects of endurance training on the PWV and central pulse pressure [11], [12]. With regard to strength training interventions, meta-analysis have shown results with discrepancy. Ashor et al. [11] concluded that strength training has no chronic effect on PWV, but there was a significant heterogeneity between the studies. Intense strength training leads to a chronic increase of the PWV [13, 14], but Rossow et al. [15] could not identify any effect on the PWV with 80% of 1RM with an 8-week strength training. However, resistance exercise seems to have no adverse effect on arterial stiffness if the training is performed with low intensity or in a slow eccentric manner or with lower limbs [13]. In one study, a weight training with low intensity reduced PWV after 10 weeks and improved vascular elasticity [16]. Changes in PWV following chronic resistance training were not related to changes in heart rate or blood pressure after interventions [11].

There is less knowledge about acute effects of exercise on vascular elasticity. For an individual risk factor oriented control of a strength training, acute cardiovascular responses to exercise are of major relevance. This is

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especially important for target groups that have cardiovascular diseases or that have an increased risk of developing cardiovascular disease due to metabolic disorders (diabetes mellitus, renal failure). Therefore, it is crucial to understand the acute response of arterial stiffness to a strength training. Some studies have demonstrated an increased PWV after a strength training involving all major muscle groups [17–19]. After less than 60 minutes the PWV was back to baseline level [17]. Furthermore intensive resistance exercises of the upper body elevated PWV significantly [20]. Nevertheless, another study showed no significant changes in PWV after strength training [21]. A single-leg resistance exercise appeared to decrease arterial stiffness in the exercised leg while having no effect on central arterial stiffness or arterial stiffness of the non-exercised leg [22]. However, the exercise intensity and the measurement times were very different in all studies. Only one of the existing studies examined the PWV immediately after strength training [23] and none of the studies distinguished between intensity levels. In our own studies, an increase in arterial stiffness during isometric muscle tension was observed [24]. Recent studies have examined the physiological response of vascular stiffness during strength training. Strictly standardized experimental protocols were chosen, which examined the physiological effects. No study compared different training protocols that are applied in training practice.

The purpose of the present study was to examine the acute effects of three practice-oriented strength training protocols with different intensity levels and exercise volumes on arterial stiffness and on central blood pressure parameters.

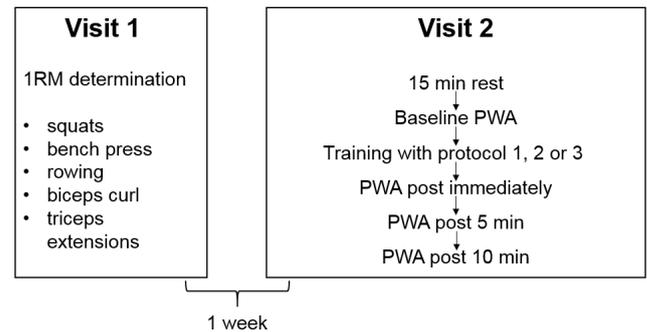
## 2. Methods

41 healthy sport students (5 women, 36 men; age 23,8±2,3 yr; height 1,78±0,1 m; body weight 72,9±9,0 kg; BMI 22,9±2,0 kg/m<sup>2</sup>) were assigned into three groups (Table 1) with different strength exercise protocols (Table 2). All of the participants have been physically active in one or more kind of sport for at least two years. All were normotensive, non-obese and free of known cardiovascular or metabolic disease. No participant took medications likely to affect the heart rate (HR) or the blood pressure (BP). Participants were non-smokers and were instructed to avoid alcohol, caffeine and strenuous exercise 24-h before the testing. All participants gave written informed consent. This study conformed to the principles outlined in the Helsinki Declaration.

The participants visited the laboratory two times. At the first visit, one week before the main experimental session, they underwent a test to determine the dynamic concentric one repetition maximum (1RM) for the five resistance exercises (1RM was defined as the maximum amount of weight, which was lifted with proper form through a full range of motion for a single repetition).

**Table 1.** Subject data (mean ± standard deviation, N=sample size: female/male, BMI=body mass index, \*=significant difference to Group 1, †=significant difference to Group 2, p<0,05)

	Age (yr)	Height (m)	Weight (kg)	BMI (kg/m <sup>2</sup> )
<b>Total</b> N=41:5/36	23.78±2.25	1.78±0.07	72.94±9.08	22.92±2.01
<b>Group 1</b> N=10:1/9	24.50±0.97	1.80±0.07	73.15±9.45	22.39±1.56
<b>Group 2</b> N=15:0/15	22.93±2.55	1.81±0.05	76.80±7.78	23.43±1.82
<b>Group 3</b> N=16:4/12	24.13±2.32	1.74±0.07*†	69.19±8.64	22.77±2.31



**Figure 1.** Flow diagram of the study design (1RM = one repetition maximum, PWA = pulse wave analysis)

At the second visit, participants stayed in a lying position for 15 minutes before measuring the baseline PWA values. The resistance exercise bout consisted of the five different exercises in the following order: squats in the multi-press, bench press in the multi-press, rowing with the barbell, biceps curl with the SZ curl bar (Eleiko®) and lying triceps extensions with the SZ curl bar (Eleiko®). Immediately after the last set, after 5 and after 10 minutes, PWA measurements were accomplished in a supine position. The subjects were requested to exhale regularly in the concentric phase and inhale in the eccentric phase.

The strength training protocols have practice oriented load characteristics (Table 2) and are recommended in reference books [25, 26].

**Table 2.** Load characteristics of exercise groups (1RM=one repetition maximum, sets= number of sets in the 5 different exercises, set pause=pause between sets in the same exercise, exercise pause=pause between the different exercises, for example between squats and bench press)

Group	Intensity (of 1RM)	Sets	Repetitions per set	Set pause [sec]	Exercise pause [sec]
1	30%	3	30	30	120
2	50%	3	20	90	120
3	70%	4	10	90	120

PWA was performed using Mobil-O-Graph (I.E.M., Stolberg, Germany). This device is a non-invasive, cuff-based, oscillometric measurement device applying a transfer function from the brachial pressure waves that were validated according to European Society of Hypertension recommendations [27]. The cuff was centered to the left

upper arm. Cuff size was chosen according to the circumference of the mid upper arm. The parameters were calculated by the Hypertension Management Software CS V. 4.7. The measuring system was extensively validated against catheter measurement (gold standard) [28] and cardiac magnetic resonance imaging and is considered to be very reliable [29].

Based on the PWA, the following parameters were determined: pulse wave velocity (PWV) [m/s], central systolic blood pressure (cSBP) [mmHg], central diastolic blood pressure (cDBP) [mmHg], central pulse pressure (cPP) [mmHg], peripheral vascular resistance (PVR) [s\*mmHg/ml] and heart rate (HR) [1/min].

The Shapiro-Wilk test was used to test the normal distribution. For normally distributed parameters, the T-test was used to test mean differences and for abnormality distributed parameters, the Wilcoxon test was used ( $p < 0.05$ ). Correlations between anthropometric data and the studied parameters were tested by the Spearman correlation. The Levene test was used to check the homogeneity of variance. The sphericity was checked by the Mauchly test. Effects between repeated measurements and their interaction with the intensity factor were tested using the repeated measures two-factor analysis of variance (3 groups x 4 time points). An ANCOVA Model was used to test the interaction of PWV and SBP with SBP as the covariate. If no homogeneity of variance has been found, the Huynh-Feldt test was used to prove the main effects of mean differences between time points. Differences between repeated measurements and between the intensities were tested using the Bonferroni correction. The significance level was 5%. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) 23.0 (SPSS Inc, Chicago, IL, USA). Figures were created using Grapher 4.0 (Golden Software, Golden, CO, USA).

### 3. Results

Table 3 shows the average values as well as the standard deviations of the studied parameters. No significant differences of PWV among different exercise protocols could be detected ( $p > 0,05$ ). The largest time effects within the groups showed the measurements immediately after training with a significant increase in PWV, cSBP, cPP and HR compared to the resting value. In Figure 2, it is evident that the training protocol of group 3 (high intensity, low repetition number, long set pause) leads to the lowest deflections of the measured parameters. It was the only training protocol without significant changes in the PWV and the cSBP at different time points. The rest values of these parameters were reached after 5 minutes, while in group 1 and 2 the rest values were not reached again after 10 minutes. Group 3 showed also the significantly smallest increase of the HR. The exercise protocol effected only the HR (partial  $\eta^2 = 0,122$ ;  $p = 0,047$ ) and the cSBP (partial  $\eta^2 = 0,161$ ;  $p = 0,035$ ). The other parameters showed no significant

effect between the groups ( $p > 0,05$ ). The factor “measuring time point” influenced all parameters (Table 4). The analysis of variance with covariate cSBP showed a significant effect on the PWV ( $p < 0.05$ ). This means that load dependent increases of the blood pressure also increase the PWV.

### 4. Discussion

Arterial stiffness is an important predictor for cardiovascular risk and can be positively influenced by chronic exercise training. So far, there are few studies that examined the acute effects of resistance training on arterial stiffness and central blood pressure values immediately after a training session, e.g. [23]. No study compared different strength training protocols with different load characteristics so far. This study was designed to investigate the acute changes after strength training in typical practice-oriented strength training protocols with different levels of intensity, repetition numbers and set pauses.

The blood pressure is regulated via baroreceptors in the aortic arch and carotid sinus, which are stimulated by the stretching of the vascular wall. Here, the brain stem is constantly informed about the pressure conditions in the arterial vascular system and controls the activation and inhibition of the sympathetic and parasympathetic nervous system. A feedback control model describes the regulation of blood pressure. The control variable is the mean arterial blood pressure, which is regulated by cardiac output and PVR [30].

Heffernan et al. [18] noted that central arterial stiffness was correlated with baroreflex-sensitivity (BRS) after resistance exercise bout, and changes in central stiffness from rest to recovery were correlated with changes in BRS from rest to recovery. This supports a relationship between BRS and arterial function following acute resistance exercise. Consequently the acute increase of the PWV can be explained with a reduction in BRS following resistance exercise [18]. This could explain the increase of PWV in all three training protocols. The lower increase of PWV at 70% could be explained by a lower impact of this protocol on the BRS.

Furthermore, the blood circuit is adapted to physical stress by local chemoreceptors, which are activated by hypoxia, acidosis and hypercapnia, which increases the blood flow and vasodilation in the working muscles [30]. A potential mechanism explaining acute reductions of the cDBP and the peripheral PWV after the bout of exercise is the sheer stress-induced release of nitric oxide (NO) from the endothelium [22]. Assuming that arterial stiffness is dependent on the concentration of NO [31-33], an increased concentration of NO can possibly be found in the vascular system after 70%, because no significant increase in PWV and in cSBP can be shown in this protocol. Guzel et al. [34] has shown that high intensity resistance exercises caused increases in NO and low intensity resistance exercises did not affect NO levels. In a study with chronic heart failure

patients, Meyer *et al.* [35] could not detect any differences between blood pressure increase after strength training with 60% and 80% exercise intensity. However, the PWR after 80% was significantly lower than at 60%. As well as in the present study, the blood pressure increase at higher exercise intensities appeared to be reduced by a decrease in PWR. The increased NO release of the endothelium could play an important role with regard to the arterial stiffness.

The slight changes of PWV and cSBP after 70% could be due to the lower number of repetitions and the related lower time under tension in this protocol. This protocol increased the heart rate significantly less. Therefore, a smaller total stress on the cardiovascular system can be concluded, which leads to a lower cardiac output and therefore a smaller increase in blood pressure parameters.

After an acute bout of exercise, Collier *et al.* [36] found that central PWV decreased by 8% after an aerobic exercise and remained at this level for 60 minutes, whereas a whole body resistance exercise increased the central PWV by 9.8% from pre to 40 and 60 minutes after exercise. A comparison of this study with the present one is not sensible, because the measuring time points are different. Heffernan *et al.* [18] showed 20 minutes after a full-body strength training with 10 repetition maximum (approximately 70% of 1RM) in a comparable collective, a significant increase of PWV of  $4.8 \pm 0.30$  to  $5.8 \pm 0.31$  m/s. Similar results demonstrated Yoon *et al.* [19] after a full-body strength training with 65% of 1RM. 20 minutes after the exercise, they found a significant increase of PWV compared to the control group, without a significant change of the central blood pressure parameters. However, there was only a PWV increase of approximately 0.3 m/s. In contrast to the studies above, a higher PWV 10 minutes after exercise was observed in this study only at the 30% intensity level. After a full-body strength training with 65% of 1RM (five exercises, three sets, 10 repetitions, 120 s set pause) there were no significant differences between PWV and central blood pressure values after the exercise compared to the control group [21]. This study used three of five exercises for lower extremities. In contrast to that, the present strength training protocol consisted of only one exercise for the lower limbs and this one was the first exercise in the protocol, followed by four upper body exercises.

Li *et al.* [23] showed that strength training with 70% of 1RM for the upper limb increased arterial stiffness immediately after training. However, a strength training with the same intensity for the lower limb and a full body workout decreased the vascular stiffness.

The four exercises for the upper extremities were the last four exercises in our training protocol. This could lead to an

increased PWV immediately after training. Similarly at Lefferts *et al.* [37] an increased PWV of 1 m/s for the upper extremity was found 10 minutes after a strength training. Subsequent studies should therefore consider the influence of exercise selection (upper or lower limb) as well.

In the same study [37] significant changes of the cSBD and the cDBP 10, 20 and 30 minutes after exercise were shown. The changes 10 minutes after the training could be confirmed by our study.

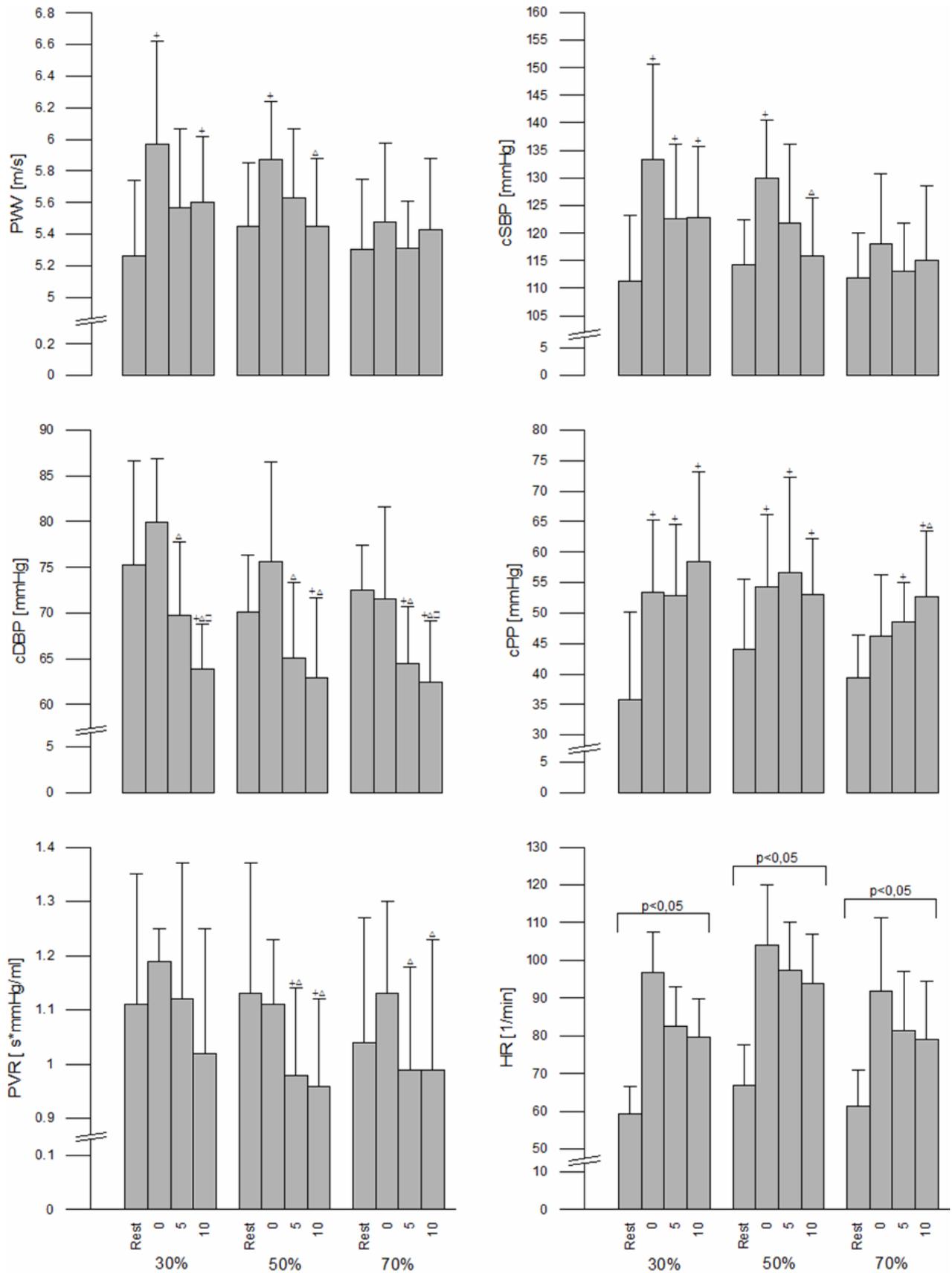
A similar reaction of PWV was found even after intensive sprint loads [38]. In the present study, the load was intensive as well, because the subjects completed the full number of repetitions in every set, if necessary with support from another person.

This is the first study that examined various exercise protocols. The five exercises are often used in practice and consist of free exercises with barbells and leaded exercises at a multi-press. Li *et al.* [23] used only leaded exercises at sequence devices. An analysis with regard to the differentiation between individual exercises can provide additional information about the hemodynamic effects of strength training contents. At this point, a differentiation between exercises for a small and a large percentage of the whole body muscle mass is important, because a different percentage results in a different strain of the cardiorespiratory system [25, 39, 40]. In addition, aspects of the movement frequency need to be considered. Moreover, it can be assumed, that the order of exercises is important. For reasons of standardization, the exercises have been completed in the same order in all three groups in this study. The last exercise was triceps extensions. This may have influenced the acute measurement at the end of the training. Future studies should implement a randomization of the order. Another factor influencing acute changes of PWV is the breathing during exercise execution. Mak *et al.* [41] showed that the PWV after a strength training with Vasalva-manuevers increased significantly more than after a strength training with continuous breathing.

The measurement method used in our paper does not allow an analysis during dynamic work. The measurement time of the analysis would be too long for practical training protocols. However, we should retain that the extent of the values of the examined parameters do not reflect the exact value during dynamic work, because the measuring time was about 60 seconds. Measurements with a catheter would significantly affect the performance and the motivation of volunteers. Therefore, the employed non-invasive method is a suitable way to quantify the arterial stiffness immediately after physical strain.

**Table 3.** Results (mean ± standard deviation (95% confidence interval)) of measurements in rest, immediately afterload (0), 5 minutes afterload (5) and 10 minutes afterload (10) (PWV=pulse wave velocity in m/s, cSBP=central systolic blood pressure in mm/Hg, cDBP=central diastolic blood pressure in mm/Hg, cPP=central pulse pressure in mm/Hg, PVR=peripheral vascular resistance in s\*mmHg/ml, HR=heart rate in 1/min)

	Group 1				Group 2				Group 3			
	Rest	0	5	10	Rest	0	5	10	Rest	0	5	10
<b>PWV</b>	5,26 ±0,48 (4,90-5,62)	5,97 ±0,65 (5,48-6,46)	5,57 ±0,50 (5,12-5,95)	5,60 ±0,42 (5,28-5,92)	5,45 ±0,40 (5,23-5,67)	5,87 ±0,37 (5,66-6,07)	5,63 ±0,44 (5,38-5,87)	5,45 ±0,43 (5,21-5,69)	5,30 ±0,45 (5,06-5,54)	5,48 ±0,50 (5,21-5,75)	5,31 ±0,30 (5,15-5,47)	5,43 ±0,45 (5,19-5,67)
<b>cSBP</b>	111,3 ±11,9 (102,3-120,3)	133,4 ±17,2 (120,4-146,4)	122,6 ±13,6 (112,3-132,9)	122,8 ±13,0 (113,0-132,6)	114,3 ±8,1 (109,8-118,8)	129,9 ±10,5 (124,1-135,8)	121,8 ±14,4 (113,8-129,8)	115,8 ±10,6 (109,9-121,7)	111,9 ±8,2 (107,5-116,3)	118,1 ±12,7 (111,3-124,8)	113,1 ±8,7 (108,5-117,8)	115,0 ±13,5 (107,8-122,2)
<b>cDBP</b>	75,3 ±11,4 (66,7-83,9)	80,0 ±6,9 (74,8-85,2)	69,7 ±8,1 (63,6-75,8)	63,9 ±4,9 (60,2-67,6)	70,1 ±6,3 (66,7-73,6)	75,6 ±10,9 (69,5-81,7)	65,1 ±8,3 (60,5-69,7)	62,9 ±8,8 (58,0-67,8)	72,5 ±4,9 (69,9-75,1)	71,6 ±10,0 (66,3-76,9)	64,5 ±6,2 (61,2-67,8)	62,5 ±6,7 (58,9-66,1)
<b>cPP</b>	35,8 ±14,3 (25,0-46,6)	53,4 ±11,9 (44,5-62,3)	52,8 ±11,7 (44,0-61,6)	58,5 ±14,7 (47,4-69,6)	44,1 ±11,5 (37,8-50,5)	54,3 ±11,9 (47,7-60,9)	56,6 ±15,6 (47,9-65,3)	53,0 ±9,2 (47,9-58,1)	39,4 ±7,0 (35,7-43,2)	46,2 ±10,0 (40,9-51,5)	48,5 ±6,5 (45,1-51,9)	52,7 ±10,8 (46,9-58,4)
<b>PVR</b>	1,11 ±0,24 (0,93-1,29)	1,19 ±0,06 (1,15-1,23)	1,12 ±0,25 (0,94-1,31)	1,02 ±0,23 (0,85-1,20)	1,13 ±0,24 (1,00-1,26)	1,11 ±0,12 (1,04-1,18)	0,98 ±0,16 (0,91-1,06)	0,96 ±0,16 (0,87-1,04)	1,04 ±0,23 (0,91-1,16)	1,13 ±0,17 (1,04-1,23)	0,99 ±0,19 (0,89-1,09)	0,99 ±0,24 (0,86-1,12)
<b>HR</b>	59,3 ±7,3 (53,8-64,8)	96,9 ±10,6 (88,9-104,9)	82,7 ±10,4 (74,9-90,5)	79,7 ±10,0 (72,2-87,2)	66,9 ±10,7 (61,0-72,9)	104,1 ±16,0 (95,2-112,9)	97,5 ±12,6 (90,6-104,5)	94,0 ±13,0 (86,8-101,2)	61,4 ±9,6 (56,3-66,6)	92,0 ±19,4 (81,7-102,3)	81,5 ±15,5 (73,2-89,8)	79,2 ±15,2 (71,0-87,3)



**Figure 2.** Changes of the parameters in the different training protocols (+=significant changes from rest, Δ=significant changes to 0 after load, □=significant changes to 5 after load, horizontal bracket indicates significant differences between time points)

**Table 4.** Effect sizes and p values of ANOVA

	Eta <sup>2</sup> of factor group	Eta <sup>2</sup> of factor measuring time point	Eta <sup>2</sup> of group x measuring time point
PWV	0,114 p=0,101	0,185 p=0,000	0,073 p=0,204
cSBP	0,161 p=0,035	0,260 p=0,000	0,087 p=0,101
cDBP	0,087 p=0,176	0,460 p=0,000	0,055 p=0,358
cPP	0,089 p=0,169	0,372 p=0,000	0,075 p=0,171
PVR	0,055 p=0,343	0,135 p=0,001	0,037 p=0,624
HR	0,187 p=0,020	0,840 p=0,000	0,122 p=0,047

## 5. Conclusions

The present investigation allows conclusions about physiological effects of different dynamic strength training protocols of active young healthy subjects. The performed protocols showed a physiological adaption of the cardiovascular system. Resistance exercises with low, moderate and high intensity let to a modest increase of PWV, cSBP, cPP and HR. Only those protocols with lower load and more repetitions acutely increased arterial stiffness (PWV, cSBP, cPP) in healthy subjects significantly. The cDBP showed 5 and 10 minutes after a training session a reduction compared to 0 minutes after training in all of the training protocols. For practical conclusions in specific target groups, like older healthy adults or patients with cardiovascular diseases, the protocols need to be tested in these groups.

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