

Training type and vascular health in young male athletes: A comparison of brachial and central blood pressure and pulse wave velocity

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Abstract

Background:

Regular exercise influences cardiovascular development in children and adolescents; however, vascular adaptations may differ according to the predominant exercise demands of specific sports. Whether broad sport categories adequately capture these differences remains unclear. This study compared blood pressure and pulse wave velocity (PWV) among young athletes from different sport categories and explored sport-specific vascular profiles.

Methods:

This study pooled data from three cohorts including 532 boys aged 8–17 years. Participants comprised competitive athletes (≥ 3 h/week training) and controls (< 3 h/week organized sports). Sports were categorized into endurance ($n = 72$), power ($n = 43$), and mixed ($n = 389$); the skill group was excluded due to small sample size. Brachial systolic and diastolic blood pressure (bSBP and bDBP), central systolic blood pressure (cSBP), and PWV were assessed oscillometrically. Training load was quantified using a MET-based index. Group differences were analyzed using ANCOVA adjusted for age, body surface area, and training load. Sport-specific vascular profiles were explored using hierarchical cluster analysis.

Results:

After adjustment, significant group effects were observed for bDBP ($p < 0.001$, $\eta^2p = 0.034$) and cSBP ($p = 0.012$, $\eta^2p = 0.021$), but not for bSBP or PWV. Power athletes had higher bDBP and cSBP than controls and endurance athletes; mixed-sport athletes also showed higher bDBP than controls and endurance athletes. Cluster analysis identified three distinct sport-specific vascular profiles. A “low-normal” cluster (e.g., swimming, cross-country skiing) showed lower values across all parameters, whereas a “high-normal” cluster (e.g., wrestling, rowing) exhibited higher values for bSBP, cSBP, and PWV (all $p \leq 0.012$ vs. low-normal). An intermediate cluster (e.g., soccer, basketball) displayed moderate values with relatively higher bDBP.

Discussion:

Sport-specific vascular differences were mainly evident in cSBP and bDBP, whereas adjusted bSBP and PWV did not differ across broad sport categories. Higher cSBP in power athletes suggests that central hemodynamics may be more sensitive than peripheral systolic pressure to sport-specific loading. Cluster-derived profiles crossed conventional sport categories, indicating that broad classifications may obscure heterogeneity between disciplines. These exploratory findings support more differentiated

sport-specific cardiovascular phenotyping but require longitudinal validation accounting for growth and biological maturation.

1. Introduction

Although cardiovascular disease usually becomes clinically apparent in adulthood, several underlying risk factors and vascular phenotypes emerge during childhood and adolescence. Elevated blood pressure in youth contributes to the cumulative cardiovascular risk burden associated with later clinical events, making early life an important period for prevention [1]. Regular physical activity generally promotes cardiovascular health, and longitudinal data link greater activity during adolescence to more favorable endothelial function and arterial structure [2]. Competitive sport, however, represents a substantially more intense and specialized physiological exposure. Its cardiovascular effects may therefore depend not only on training volume, but also on the duration, intensity and static or dynamic characteristics of the exercise stimulus [3].

Brachial blood pressure remains the clinical standard for blood pressure assessment, but it does not fully characterize the load within the central arterial system. Because pressure amplification varies along the arterial tree, individuals with similar brachial systolic pressure may have different central aortic pressures [4]. Central systolic blood pressure more closely approximates the pulsatile load imposed on the proximal aorta and left ventricle and is more strongly associated with cardiovascular outcomes than brachial pressure in adult populations [5]. Aortic pulse wave velocity (PWV) provides complementary information on central arterial mechanics and predicts cardiovascular events and mortality in adults [6]. These relationships cannot be directly extrapolated to healthy youth, in whom central pressure and PWV are strongly influenced by biological measures such as age, height, body size, blood pressure, and pubertal development [7]. Importantly, resting pressure is not determined by the arterial wall alone. Mean arterial pressure reflects the interaction between cardiac output and systemic vascular resistance, whereas systolic and central pressures are additionally shaped by stroke volume, ventricular ejection, proximal aortic impedance, arterial compliance, heart rate, and wave reflection [4]. Training may modify several of these components through changes in autonomic regulation, ventricular filling, peripheral vascular tone, and circulating blood volume. Hematological adaptation may also contribute as endurance training can expand plasma and red-cell volume, such that hematocrit may decrease despite preserved or increased total erythrocyte and hemoglobin mass [8, 9]. Blood volume affects venous return and stroke volume, while hematocrit and erythrocyte properties influence viscosity and microvascular resistance. Between-sport differences may therefore reflect an integrated adaptation of the heart, vascular system, and circulating blood rather than isolated arterial stiffening. Such differences need not denote disease but may represent distinct physiological responses to recurrent training loads.

Training modality provides a plausible basis for divergent hemodynamic profiles. Endurance exercise produces sustained increases in cardiac output and antegrade flow, repeatedly exposing the endothelium to laminar shear stress and generally promoting nitric oxide-mediated vasodilation and arterial compliance [3]. In contrast, power-oriented exercise involves brief high-force contractions,

compression of resistance vessels, sympathetic activation, increased intrathoracic pressure, and often the Valsalva maneuver. These responses can generate exceptionally large transient increases in arterial pressure [10]. Recurrent pressure loading has consequently been proposed to alter central arterial compliance, with some intervention studies reporting reduced compliance after high-intensity resistance training [11, 12].

The terms vascular adaptation and remodeling should nevertheless be distinguished carefully. Central pressure and PWV characterize an integrated hemodynamic and mechanical phenotype but cannot establish cellular or histological remodeling. True structural remodeling may involve layer-specific changes in endothelial signaling, smooth-muscle cell phenotype and cytoskeletal organization, and the relative content and arrangement of elastin, collagen and other extracellular-matrix proteins [13]. However, such changes may be adaptive rather than pathological, and their relevance to healthy growing athletes remains uncertain.

Comparisons among adult endurance, strength, and power athletes indicate heterogenous, region-specific, and body-size-dependent vascular phenotypes rather than one uniform “athlete’s artery” [14]. Evidence in children and adolescents is more limited. Blood pressure and adiposity remain important correlates of higher central PWV and there is evidence of an inverse association between cardiorespiratory fitness and arterial stiffness [15]. More recent data indicate that participation in high-static, low-dynamic sports is associated with a greater prevalence of elevated brachial blood pressure in adolescent athletes [16], while longitudinal observations show that central systolic pressure increases during adolescence with trajectories differing by sex and sport type [17].

Broad categories such as endurance, power, and mixed sport nevertheless simplify multidimensional training exposures. Disciplines within the same category may differ in contraction pattern, intermittent load, body-composition demands, training history, and acute pressor stimuli. Data-driven analysis of integrated vascular profiles may therefore complement conventional sport classifications by identifying systematic discipline-specific adaptation patterns that remain evident after accounting for age, sex, body size, and developmental status, and by clarifying whether these profiles are consistent with physiological vascular remodeling rather than adverse vascular alteration..

Accordingly, the present study aimed to compare brachial systolic and diastolic blood pressure, central systolic blood pressure, and aortic PWV among young male endurance, mixed-sport, and power athletes and lower-training controls, while accounting for age, body surface area, and training exposure. We hypothesized that power athletes would exhibit higher central systolic blood pressure and greater aortic stiffness than endurance and mixed-sport athletes. In addition, an exploratory sport-level clustering analysis was performed to identify disciplines sharing similar multivariable vascular profiles beyond their predefined categories.

2. Methods

This cross-sectional analysis included male participants aged 8 to 17 years from three cohorts conducted at the Institute of Preventive Pediatrics, Technical University of Munich (TUM), between 2018 and 2024. Data from the MuCAYA study [18], the MuCAYA^{plus} study [19], and the LICO study [20] were pooled for analysis, resulting in a total sample of 532 boys. Detailed descriptions of the study designs and recruitment procedures have been published previously.

Participants from the MuCAYA (n = 320) and MuCAYA^{plus} cohorts (n = 189) were competitive young athletes. Inclusion criteria comprised regular participation in a primary sports discipline, club affiliation, active participation in competitive sport, and a weekly training duration of at least three hours. The control group (n = 23) consisted of boys from the LICO study who engaged in organized sports at a recreational level with a maximum of three hours of per week. Exclusion criteria for all cohorts included acute infections, current injuries, chronic diseases, and missing written informed consent. Across all studies, identical measurement methods, equipment, and standard operating procedures were used.

The young athletes from the MuCAYA and MuCAYA^{plus} studies participated in more than 25 different sports disciplines, with sample sizes ranging from 1 to 216 participants per discipline. Because statistical analysis at the individual sport level was not methodologically feasible due to the highly unequal sample sizes and the large number of disciplines, sports were categorized according to the predominant exercise component into endurance (n = 72), power (n = 43), mixed (n = 389), and skill (n = 5), following the classification of exercise and sports of the European Society of Cardiology [21]. The mixed category was heterogeneous and largely dominated by soccer (see Tab. S1).

All studies were conducted as single-center investigations at the Institute of Preventive Pediatrics, TUM. Ethical approvals were obtained for each study from the Ethics Committee of the Technical University of Munich. Prior to participation, all participants and their legal guardians received written and verbal study information, and written informed consent was obtained from both in accordance with age-appropriate ethical standards.

2.1. Assessment of anthropometry

Body weight was assessed by bioelectrical impedance analysis using the InBody 270 body composition analyzer (InBody Europe, Eschborn, Germany); the study participants wore light clothing and were barefoot. Height was measured with a stadiometer in an upright, standing to the nearest 0.1 cm. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. BSA was calculated using the formula of Dubois and Dubois [22]. Standard deviation scores (SDS) for height and BMI were calculated on German reference data [23].

2.2. Blood pressure and pulse wave analysis

Brachial systolic (bSBP) and diastolic blood pressure (bDBP), as well as central arterial stiffness, were assessed using a noninvasive oscillometric device (Mobil-O-Graph, IEM, Stolberg, Germany) based on a single measurement. All recordings were obtained from the left upper arm with participants in a supine

position following a 10-minute rest period. Appropriate cuff sizes were selected according to individual upper arm circumference. Central systolic blood pressure (cSBP) and aortic pulse wave velocity (PWV) were derived indirectly from brachial pulse waveforms using the ARCSolver algorithm (Austrian Institute of Technology, Vienna, Austria) [24]. SDS for bSBP and bDBP were calculated based on German reference data [23], whereas SDS values for cSBP and PWV were determined according to reference values provided by Elmenhorst, Weberruss [25].

2.3. Assessment of sports activities

Habitual physical activity was assessed using the MoMo Physical Activity Questionnaire for children and adolescents [26]. All participants, including those in the control group, completed the questionnaire. While participants in the MuCAYA and MuCAYA^{plus} cohorts were engaged in competitive sports, control participants were allowed to participate in organized sports at a recreational level with a maximum of three hours per week.

Participants provided information on the weekly time spent in their sports disciplines (min/week), the number of months during the previous year in which this training volume was maintained, and the perceived exercise intensity categorized as low, moderate, or high. Weekly training duration was calculated by adjusting the reported weekly minutes by the proportion of active months (months/12). Each sport was assigned a specific metabolic equivalent (MET) value according to established compendia [27, 28]. The MET-hours index, hereinafter referred to as training load, was then derived by multiplying the corresponding MET value by the adjusted training duration and converting minutes to hours by division by 60.

2.4. Statistical analysis

Only athletes from the control, endurance, power, and mixed category were included in the statistical analyses. Athletes in the skill category were excluded because the limited sample size ($n = 5$) precluded reliable statistical inference.

Statistical analyses were performed using IBM SPSS Statistics version 31.0.0.0 (IBM Corp., Armonk, NY, USA). Statistical significance was defined as a two-sided $p < 0.05$. Data are presented as mean $\pm$ standard deviation unless otherwise stated.

Baseline participant characteristics were compared between groups (control, endurance, mixed, and power) using one-way analysis of variance (ANOVA) with Games-Howell post-hoc test due to unequal group sizes. One-sample t-tests compared SDS against the reference mean (SDS = 0).

Differences in bSBP, bDBP, cSBP, and PWV between groups were assessed using analysis of covariance (ANCOVA). Age and BSA were selected a priori as covariates because both are established determinants of blood pressure and arterial stiffness during growth. Training load was additionally included to account for differences in cumulative exercise load between sports categories. Potential covariates such as BMI, resting heart rate, and training duration were not included as they were considered either highly colinear

with the selected covariates or conceptually related to the exposure of interest. Adjusted marginal means were compared using Sidak-corrected pairwise comparisons.

Assumptions of ANCOVAs, including normality of residuals, homogeneity of regression slopes, homogeneity of variances, linearity between continuous covariates and dependent variables, and absence of influential observations, were assessed using graphical inspection, the Shapiro–Wilk test, Levene's test, and Cook's distance, respectively. Multicollinearity among covariates was evaluated using variance inflation factors (VIF), with all values below 3 indicating no evidence of problematic multicollinearity. Effect sizes are reported as partial eta squared (η^2p). Given that more than 50% of athletes in the mixed category were soccer players, a sensitivity analysis was conducted using the ANCOVA model described above after excluding soccer players from this category. As this analysis yielded no meaningful changes in the results, soccer players were retained in the mixed category for all subsequent ANCOVAs and analyses.

To explore similarities in sport-specific vascular profiles, a hierarchical cluster analysis was performed at the sport level. Only sport disciplines represented by at least 10 participants were included to improve the stability of sport-specific estimates. For each vascular parameter (bSBP, bDBP, cSBP, and PWV), estimated marginal means adjusted for age, BSA, and training load were obtained from the ANCOVA models. The adjusted means were standardized (z-scores) prior to clustering to account for differences in measurement scales. Hierarchical agglomerative clustering was performed using Ward's minimum variance method with squared Euclidean distance as the similarity measure. To support the selection of the optimal number of clusters, several complementary validation approaches were applied, including visual inspection of the dendrogram, the agglomeration schedule, the elbow method (within-cluster sum of squares), the average silhouette method, and the gap statistic. Because these methods yielded partly divergent recommendations, the final number of clusters was determined by jointly considering the statistical criteria, cluster size balance, and the interpretability of the resulting sport-specific vascular profiles. This analysis was considered exploratory and intended to identify groups of sports with similar multivariable vascular characteristics rather than predefined physiological classifications.

Missing data were not imputed, and analyses were performed using all available observations for each respective analysis. The amount of missing data was low (< 2% for all variables). Variable-specific numbers of available and missing observations are provided in Supplementary Table S2.

3. Results

3.1. Participant characteristics and group comparisons

Participant characteristics and post-hoc group comparisons are presented in Table 1. Control participants were significantly younger and smaller than all athlete groups, whereas power athletes were older than endurance and mixed athletes. Body height, body weight, and BSA were significantly lower in controls than in all athlete groups, while BMI was highest in power athletes. Resting heart rate was

higher in controls than in endurance and mixed athletes. Among the vascular parameters, power athletes exhibited higher bSBP than endurance athletes, higher bDBP than controls and endurance athletes, and higher cSBP than all other groups. Mixed athletes also showed higher bDBP than endurance athletes and higher cSBP than controls. PWV differed only between controls and power athletes. Training duration and training load showed the largest group differences, with controls displaying the lowest values and endurance athletes the highest.

Table 1
Participant characteristics

Age (years)	Control (n = 23)	Endurance (n = 72)	Mixed (n = 389)	Power (n = 43)	p	Significant group differences
	11.5 ± 2.0	13.4 ± 1.9	13.9 ± 1.9	14.6 ± 1.5	< 0.001	Control < Endurance, Power, Mixed; Endurance < Power; Mixed < Power
Height (cm)	153.3 ± 12.4	165.8 ± 14.4	167.2 ± 14.9	167.5 ± 12.2	< 0.001	Control < Endurance, Power, Mixed
Weight (kg)	43.1 ± 10.8	53.4 ± 12.2	59.1 ± 14.7	59.1 ± 15.1	< 0.001	Control < Endurance, Power, Mixed
BMI (kg/m ²)	18.1 ± 2.6	19.1 ± 2.5	19.2 ± 2.5	20.7 ± 3.0	< 0.001	Control < Power; Endurance < Power; Mixed < Power
BSA (m ²)	1.35 ± 0.22	1.56 ± 0.27	1.59 ± 0.28	1.65 ± 0.26	< 0.001	Control < Endurance, Power, Mixed
Training duration (min/week)	139.9 ± 42.6	583.7 ± 264.3	483.5 ± 196.7	454.1 ± 191.8	< 0.001	Control < Endurance, Mixed
Training exposure (MET _h -Index)	22.6 ± 9.2	101.9 ± 52.2	76.1 ± 29.2	85.9 ± 41.3	< 0.001	Power < Endurance
Resting heart rate (bpm)	68.9 ± 8.4	63.2 ± 9.0	63.6 ± 9.7	65.6 ± 9.0	0.043	Power < Control, Endurance; Mixed > Endurance
Brachial SBP (mmHg)	111.7 ± 13.2	112.9 ± 11.0	115.1 ± 9.4	119.1 ± 10.5	0.005	Power > Control, Endurance, Mixed; Mixed > Control
Brachial DBP (mmHg)	59.8 ± 6.4	60.6 ± 5.8	63.5 ± 5.9	65.0 ± 7.9	< 0.001	Power > Control
Central SBP (mmHg)	96.6 ± 8.5	100.7 ± 11.6	103.1 ± 10.8	108.7 ± 13.1	< 0.001	Power > Control, Endurance, Mixed; Mixed > Control
<i>Data are presented as mean ± SD. Group sample sizes shown in the column headings represent the total number of participants assigned to each group. Due to occasional missing values, the number of participants included in individual analyses may vary slightly. Analyses were performed using all available data.</i>						
<i>BMI, body mass index; BSA, body surface area; DBP, diastolic blood pressure; MET, metabolic equivalent of tasks, PWV, pulse wave velocity; SBP, systolic blood pressure.</i>						

Age (years)	Control (n = 23)	Endurance (n = 72)	Mixed (n = 389)	Power (n = 43)	p	Significant group differences
	11.5 ± 2.0	13.4 ± 1.9	13.9 ± 1.9	14.6 ± 1.5	< 0.001	Control < Endurance, Power, Mixed; Endurance < Power; Mixed < Power
PWV (m/s)	4.59 ± 0.40	4.76 ± 0.52	4.80 ± 0.45	5.01 ± 0.57	0.004	Power > Control, Mixed, Endurance; Power > Control; Mixed > Control
<p><i>Data are presented as mean ± SD. Group sample sizes shown in the column headings represent the total number of participants assigned to each group. Due to occasional missing values, the number of participants included in individual analyses may vary slightly. Analyses were performed using all available data.</i></p>						
<p><i>BMI, body mass index; BSA, body surface area; DBP, diastolic blood pressure; MET, metabolic equivalent of tasks, PWV, pulse wave velocity; SBP, systolic blood pressure.</i></p>						

Table 2
Standard deviation scores of vascular and anthropometric parameters

Brachial SBP SDS	Control (n = 23)	Endurance (n = 72)	Mixed (n = 389)	Power (n = 43)	p	Significant group differences
	0.36 ± 1.30	-0.03 ± 1.07	0.13 ± 0.98	0.43 ± 0.95	0.089	
Brachial DBP SDS	-0.77 ± 0.93	-0.90 ± 0.85	-0.55 ± 0.86	-0.39 ± 1.14	0.006	Mixed > Endurance
Age-adjusted PWV SDS	0.42 ± 1.65	0.19 ± 1.70	0.18 ± 1.36	0.58 ± 1.82	0.340	
Age-adjusted central SBP SDS	0.12 ± 1.08	0.00 ± 1.38	0.18 ± 1.28	0.71 ± 1.76	0.046	
Height-adjusted PWV SDS	-0.14 ± 1.49	-0.34 ± 1.52	-0.21 ± 1.40	0.48 ± 1.67	0.020	
Height-adjusted central SBP SDS	-0.33 ± 0.94	-0.42 ± 1.20	-0.17 ± 1.29	0.61 ± 1.59	< 0.001	Power > Control, Power > Endurance, Power > Mixed
Height SDS	0.38 ± 0.82	0.49 ± 1.03	0.33 ± 1.10	-0.18 ± 1.12	0.011	Endurance > Power, Mixed > Power
BMI SDS	-0.03 ± 0.94	-0.06 ± 0.89	-0.10 ± 0.78	0.27 ± 0.78	0.035	Power > Mixed
<i>Data are presented as mean ± SD. Group sample sizes shown in the column headings represent the total number of participants assigned to each group. Due to occasional missing values, the number of participants included in individual analyses may vary slightly. Analyses were performed using all available data.</i>						
<i>BMI, body mass index; DBP, diastolic blood pressure; PWV, pulse wave velocity; SBP, systolic blood pressure.</i>						

3.2. SDS analyses

The cohort was taller than the reference population (height SDS = 0.31, $p < 0.001$), whereas BMI SDS did not differ significantly from zero (-0.06 , $p = 0.073$). bSBP SDS was slightly higher than the reference mean (0.14 , $p = 0.001$), while bDBP SDS was lower (-0.59 , $p < 0.001$). For height-adjusted vascular parameters, PWV SDS (-0.17 , $p = 0.008$) and cSBP SDS (-0.15 , $p = 0.012$) were significantly below the reference mean. In contrast, age-adjusted SDS were positive for both PWV SDS (0.22 , $p < 0.001$) and cSBP SDS (0.19 , $p = 0.001$).

Group comparisons revealed significant differences for bDBP SDS ($p = 0.006$), age-adjusted cSBP SDS ($p = 0.046$), height-adjusted PWV SDS ($p = 0.020$), and height-adjusted cSBP SDS ($p < 0.001$), whereas

bSBP SDS and age-adjusted PWV SDS did not differ significantly between groups. Mixed-sport athletes showed higher bDBP SDS than endurance athletes ($p = 0.011$). For height-adjusted vascular parameters, power athletes exhibited higher cSBP SDS than endurance ($p = 0.002$) and mixed athletes ($p = 0.016$) as well as the control group ($p = 0.021$). Height SDS also differed between groups ($p = 0.011$), with endurance ($p = 0.009$) and mixed-sport athletes ($p = 0.030$) showing higher values than power athletes, whereas BMI SDS was higher in power than in mixed-sport athletes ($p = 0.021$). Detailed results are shown in Table 3.

Table 3

Adjusted marginal means derived from ANCOVA models adjusted for age, body surface area, and training exposure

Brachial SBP (mmHg)	Control	Endurance	Mixed	Power	F	p	Partial η^2
	114.3	114.2	114.8	117.8	1.70	0.166	0.010
Brachial DBP (mmHg)	59.4	61.1	63.4	64.9	6.06	< 0.001	0.034
Central SBP (mmHg)	100.0	102.4	102.7	107.0	3.69	0.012	0.021
PWV (m/s)	4.76	4.83	4.79	4.94	2.07	0.103	0.012
<i>Group sample sizes shown in the column headings represent the total number of participants assigned to each group. Due to occasional missing values, the number of participants included in individual analyses may vary slightly. Analyses were performed using all available data.</i>							
<i>DBP, diastolic blood pressure; PWV, pulse wave velocity; SBP, systolic blood pressure.</i>							

When vascular SDS were additionally categorized according to their position within the reference distribution (< 90th percentile = normal, 90th to < 95th percentile = high-normal, and $\geq$ 95th percentile = high), most participants were within the normal range. For bSBP, 87.4% of participants were within the normal range, 6.5% within the high-normal range, and 6.1% within the high range, while 97.5% of participants had normal bDBP SDS.

For age-adjusted PWV, 15.0% of participants had values $\geq$ 95th percentile, with the highest proportion observed in power athletes (23.3%), followed by endurance athletes (18.1%), controls (14.3%), and mixed-sport athletes (13.6%). A similar pattern was observed for age-adjusted cSBP, with 13.0% of the total cohort showing values $\geq$ 95th percentile and the highest proportion again observed in power athletes (20.9%), compared with 14.3% in controls, 11.1% in endurance athletes, and 12.4% in mixed-sport athletes.

For height-adjusted parameters, values $\geq$ 95th percentile were observed in 7.9% of the total cohort for PWV and 8.3% for cSBP. Power athletes showed the highest proportions for both parameters, with 25.6% exhibiting height-adjusted PWV values and 18.6% height-adjusted cSBP values $\geq$ 95th percentile.

Corresponding proportions were considerably lower in controls (9.5% and 4.8%), endurance athletes (8.3% and 4.2%), and mixed-sport athletes (5.7% and 8.1%), respectively.

3.3. Adjusted group differences (ANCOVA)

Adjusted marginal means of ANCOVA are presented in Table 3 and Fig. 1. After adjustment for age, BSA, and weekly training load, a significant overall group effect was observed for bDBP ($F = 6.06$, $p < 0.001$, $\eta^2p = 0.034$) and cSBP ($F = 3.69$, $p = 0.012$, $\eta^2p = 0.021$). In contrast, no significant group effects were found for bSBP ($F = 1.70$, $p = 0.166$, $\eta^2p = 0.010$) or PWV ($F = 2.07$, $p = 0.103$, $\eta^2p = 0.012$).

For bDBP, Sidak-adjusted pairwise comparisons revealed significantly higher adjusted values in power athletes than in controls ($p = 0.010$) and endurance athletes ($p = 0.010$). Likewise, mixed-sport athletes exhibited significantly higher adjusted bDBP than controls ($p = 0.045$) and endurance athletes ($p = 0.039$), while no significant differences were observed between power and mixed-sport athletes.

For cSBP, power athletes showed significantly higher adjusted values than controls ($p = 0.037$), endurance athletes ($p = 0.049$), and mixed-sport athletes ($p = 0.019$), whereas no other pairwise comparisons reached statistical significance.

3.4. Cluster analysis

A hierarchical agglomerative cluster analysis at the sport level was performed to identify sport disciplines with similar multivariable vascular profiles. The analysis included age-, BSA-, and training load-adjusted, z-standardized mean values of bSBP, bDBP, cSBP, and PWV. Ward's method with squared Euclidean distance was used.

The agglomeration schedule indicated a marked increase in within-cluster heterogeneity when merging from three to two clusters. The elbow method likewise suggested a three-cluster solution, whereas the average silhouette method favored two clusters and the gap statistic indicated no clear evidence of an underlying cluster structure. Considering these findings together with balanced cluster sizes and the interpretability of the resulting vascular profiles, a three-cluster solution was selected for subsequent analyses.

The three clusters comprised 2, 4, and 4 sports each: Cluster 1 (soccer, basketball), Cluster 2 (ice hockey, swimming, cross-country skiing, volleyball), and Cluster 3 (field hockey, handball, wrestling, rowing). ANOVAs revealed significant cluster differences for bSBP ($F(2, 7) = 80.73$, $p < 0.001$, $R^2 = 0.958$), cSBP ($F(2, 7) = 32.10$, $p < 0.001$, $R^2 = 0.902$), and PWV ($F(2, 7) = 8.46$, $p = 0.014$, $R^2 = 0.707$), but not for bDBP ($F(2, 7) = 3.64$, $p = 0.082$, $R^2 = 0.510$). Estimated marginal means of the standardized vascular parameters for each cluster are displayed in Table 4.

Table 4

Estimated marginal means (mean $\pm$ SD) of standardized vascular parameters for the three sport clusters

Brachial SBP (z)	Cluster 1 (soccer, basketball)	Cluster 2 (ice hockey, swimming, cross-country skiing, volleyball)	Cluster 3 (field hockey, handball, rowing, wrestling)
	-0.041 $\pm$ 0.231	-1.028 $\pm$ 0.232	1.048 $\pm$ 0.232
Brachial DBP (z)	0.233 $\pm$ 0.793	-0.804 $\pm$ 0.794	0.688 $\pm$ 0.794
Central SBP (z)	-0.240 $\pm$ 0.355	-0.938 $\pm$ 0.356	1.058 $\pm$ 0.356
PWV (z)	-0.396 $\pm$ 0.614	-0.765 $\pm$ 0.614	0.963 $\pm$ 0.614
<i>Group sample sizes shown in the column headings represent the total number of participants assigned to each group. Due to occasional missing values, the number of participants included in individual analyses may vary slightly. Analyses were performed using all available data.</i>			
<i>DBP, diastolic blood pressure; PWV, pulse wave velocity; SBP, systolic blood pressure.</i>			

Post-hoc comparisons with Sidak correction showed that Cluster 2 (lowest values) and Cluster 3 (highest values) differed significantly on all three significant parameters (all $p \leq 0.012$). Cluster 1 was intermediate and did not differ significantly from Cluster 2 or 3 on most parameters. Cluster 2 was characterized by consistently low z-values across all four vascular parameters (e.g., bSBP = -1.028 $\pm$ 0.232, PWV = -0.765 $\pm$ 0.614), whereas Cluster 3 exhibited the highest values (bSBP = 1.048 $\pm$ 0.232, PWV = 0.963 $\pm$ 0.614). Cluster 1 showed an intermediate profile with slightly elevated bDBP (0.233 $\pm$ 0.793) and lower PWV values (-0.396 $\pm$ 0.614) compared with Cluster 3.

These results indicate three distinct vascular profiles: a 'low-normal' profile (Cluster 2), a 'high-normal' profile (Cluster 3), and an intermediate profile (Cluster 1), characterized primarily by a relatively higher bDBP at moderate PWV (see Fig. 2).

4. Discussion

The present cross-sectional study examined whether resting vascular parameters differed according to the predominant physiological demands of competitive sport in boys aged 8–17 years. Four principal findings emerged. First, after adjustment for age and BSA, and weekly training load, power athletes exhibited higher cSBP than controls, endurance athletes, and mixed-sport athletes. The adjusted differences ranged from 4.3 to 7.0 mmHg, although the overall effect size was small. Second, bDBP was higher in power and mixed-sport athletes than in controls and endurance athletes. Third, no significant group effects remained for bSBP or estimated PWV after covariate adjustment. Finally, the exploratory sport-level cluster analysis identified three multivariable vascular profiles that crossed the predefined endurance, mixed, and power categories. The hypothesis was therefore supported for cSBP but not for PWV in the primary adjusted analysis. Rather than demonstrating greater aortic stiffness in power

athletes, the findings indicated a modest sport-category-associated central pressure phenotype whose underlying mechanism remains unsolved.

The higher cSBP in power athletes is directionally consistent with evidence that the static and dynamic components of exercise may influence blood pressure development in youth. Participation in high-static sports has recently been associated with higher odds of an elevated blood pressure screening result in a large cohort of adolescent athletes, whereas high-dynamic sport participation was associated with lower odds [16]. Longitudinal observations have also shown that cSBP increases during adolescence, with its trajectory varying according to sex and sport category [17]. In young adults, endurance athletes have demonstrated lower aortic pressure and PWV than athletes performing high-intensity resistance training [29]. Pediatric longitudinal data similarly suggest lower PWV among participants in predominantly dynamic compared with predominantly static sports [30]. However, the absence of an adjusted PWV difference in the primary ANCOVA may partly reflect the limited granularity of the applied sport classification, which may have obscured discipline-specific vascular adaptations. Accordingly, this finding should not be interpreted as evidence of uniform PWV responses across sports, populations, training programs, or measurement methods.

The distinction between cSBP and PWV is important. Power athletes demonstrated a higher central pressure, but not significantly greater estimated aortic stiffness in the fully adjusted ANCOVA. Central pressure is determined by more than the material properties of the arterial wall, as it reflects the interaction of ventricular ejection, stroke volume, proximal aortic impedance and compliance, heart rate, systemic vascular tone, pressure-wave reflection, and peripheral pressure amplification [4]. Consequently, a higher cSBP in the absence of a clear PWV difference may arise from altered ventricular-arterial coupling, wave reflection characteristics, or resting hemodynamic regulation rather than structural stiffening of the aorta.

The acute cardiovascular demands of power-oriented exercise provide one plausible explanation. High-force muscular contractions compress resistance vessels and are accompanied by sympathetic activation, increased intrathoracic pressure, and often the Valsalva maneuver. The resulting combination of increased cardiac output and incompletely reduced, or transiently elevated, systemic vascular resistance can generate pronounced increases in systolic and diastolic pressure [10]. Repeated exposure to these short pressure peaks may influence resting arterial tone, wave reflection, and proximal arterial mechanics [11]. Recent observations in resistance-trained young men are consistent with a central-peripheral dissociation: higher carotid systolic pressure and lower central arterial compliance were present despite comparable brachial blood pressure [31]. Nevertheless, resistance training does not produce a uniformly less favorable vascular phenotype. Moderate-intensity resistance-training interventions have also reported reductions in carotid–femoral PWV [32], while the available pediatric evidence does not indicate a consistent adverse effect on resting blood pressure [33]. Thus, resistance training does not appear to induce a uniform chronic increase in systolic blood pressure in youth. Rather, vascular and hemodynamic responses may depend on training intensity and volume, contraction pattern,

recovery periods, training history, baseline cardiovascular phenotype, and the relative contribution of endurance and resistance components.

The current data also requires precise terminology. Blood pressure and PWV characterize hemodynamic and mechanical phenotypes but do not per se demonstrate cellular or histological remodeling. Although experimental resistance-training models suggest alterations in smooth-muscle and extracellular-matrix architecture [13], these findings cannot be directly extrapolated to healthy growing athletes. Higher cSBP should therefore be interpreted as a sport-associated hemodynamic phenotype rather than evidence of adverse structural remodeling.

The lack of adjusted differences in bSBP and PWV further supports this cautious interpretation. Although power athletes had the highest unadjusted values, these differences were substantially attenuated after accounting for age, BSA, and training load. Growth and body size therefore appear to explain an important proportion of the crude between-group variation. PWV is additionally pressure dependent. Greater distending pressure can increase wave velocity without a permanent change in arterial wall composition. Conversely, the absence of a significant group effect does not establish identical arterial properties, particularly given the small effect size, unequal group sizes, and indirect PWV estimation. Overall, however, the primary ANCOVA provides no sufficient evidence that power athletes had greater aortic stiffness than the other groups.

The SDS analyses further highlight the importance of the reference framework, as pediatric cSBP and PWV reference values vary with both age and height. The cohort was modestly taller than the reference population (height SDS = 0.31), whereas BMI SDS did not differ significantly from zero. Age-adjusted PWV and cSBP SDS were positive (0.22 and 0.19), but height-adjusted values were below the reference mean (-0.17 and -0.15). This reversal is consistent with greater stature contributing to the apparently elevated age-standardized values and supports considering height-based references in this cohort. It does not establish that height explains the entire difference, nor should the slightly negative height-adjusted means be interpreted as evidence of a protective training effect.

Importantly, the significant overall group difference in height-adjusted PWV SDS ($p = 0.020$) contrasts with the non-significant age-adjusted PWV SDS and primary ANCOVA results. Reference-standardized comparisons and ANCOVA address different questions and do not account for identical covariates. Without reported significant pairwise PWV SDS contrasts, this secondary finding cannot establish greater arterial stiffness specifically in power athletes. Instead, it indicates that conclusions regarding PWV depend partly on the analytical framework.

The bDBP findings provide a complementary but less sport-specific signal. Both power and mixed-sport athletes showed higher adjusted bDBP than controls and endurance athletes, whereas power and mixed-sport athletes did not differ from each other. The higher bDBP SDS in mixed-sport compared with endurance athletes ($p = 0.011$) partly supports this pattern. Nevertheless, cohort-average bDBP SDS was below the reference mean (-0.59), and 97.5% of participants had values below the 90th percentile. Thus, higher bDBP between athlete groups should not be equated with elevated diastolic pressure relative to

the reference population. Lower bDBP in endurance athletes could reflect lower resting peripheral resistance, enhanced endothelial vasodilator capacity, or differences in autonomic regulation. Conversely, intermittent and static exercise components may be associated with greater sympathetic vascular tone or less pronounced resting vasodilation. Nevertheless, bDBP is sensitive to acute physiological conditions, and neither systemic vascular resistance nor autonomic function was measured. The finding should therefore be interpreted as hypothesis-generating rather than as evidence of a specific vascular mechanism.

The clearer group differentiation for cSBP than for bSBP is clinically relevant. Central pressure more closely approximates the pulsatile load experienced by the proximal aorta and left ventricle, whereas brachial pressure is additionally affected by pressure amplification between the central and peripheral arteries [4–7]. This amplification varies considerably during growth according to height, heart rate, arterial geometry, vascular tone, and wave reflection. In the present study, power athletes differed from all comparison groups in cSBP, while no overall group effect was detected for bSBP. Thus, individuals or sport groups with broadly similar peripheral systolic pressure may nevertheless differ in central pressure exposure.

This cSBP contrast was also evident in the height-adjusted SDS analyses: power athletes had higher values than endurance athletes ($p = 0.002$), mixed-sport athletes ($p = 0.016$), and controls ($p = 0.021$). Notably, their height SDS was lower than that of endurance and mixed-sport athletes, indicating lower stature relative to age-specific norms, not necessarily shorter absolute height. Their higher central pressure therefore cannot simply be attributed to being taller for age. Higher BMI SDS than in mixed-sport athletes suggests that body-composition differences may warrant consideration, although BMI cannot distinguish lean from fat mass.

The potential clinical value of cSBP lies primarily in refining cardiovascular phenotyping rather than diagnosing disease. In specialized sports-cardiology assessments, central pressure could complement brachial measurements when an athlete participates in a sport with substantial static or high-force components, or when repeated brachial measurements are high-normal, elevated, or inconsistent with the broader clinical profile. A comparatively high cSBP might justify closer longitudinal observation and integration with ambulatory blood pressure, cardiac structure and function, and direct vascular measurements. However, the present study does not establish that routine cSBP screening improves outcome prediction in young athletes. Pediatric outcome-based thresholds for central hypertension remain unavailable, and the observed group differences do not imply that individual values were abnormal. Reporting cSBP SDS, percentile distributions, and the proportion of participants exceeding device-specific reference thresholds would strengthen clinical interpretation.

The exploratory cluster analysis revealed vascular profiles that crossed conventional endurance, mixed, and power categories. Field hockey, handball, rowing, and wrestling formed the higher-value cluster, whereas ice hockey, swimming, cross-country skiing, and volleyball showed lower values; soccer and

basketball displayed an intermediate profile. This suggests that classification by a single predominant exercise component may not fully capture the cardiovascular demands of individual sports.

Cross-category similarities may reflect differences in static and dynamic loading, muscle-group involvement, repeated sprinting, recovery patterns, strength conditioning, and athlete characteristics such as body composition and maturation. For example, rowing combines endurance demands with substantial force production, while wrestling involves high static loads and intermittent dynamic activity. However, the present analysis cannot determine which characteristics underly the observed profiles. The clusters should therefore be interpreted as hypothesis-generating patterns of multidimensional training load rather than as a new physiological classification.

The clinical implications remain deliberately limited. The findings do not justify restricting participation in power-oriented sports or defining the higher cSBP profile as pathological. Effect sizes were small, PWV did not differ significantly between categories, and no hypertension-mediated organ damage was assessed. Standardized brachial blood pressure remains the primary clinical measure, and elevated values require confirmation through repeated measurements and, where appropriate, ambulatory monitoring [34]. Central pressure may provide complementary information but should not currently be used as a stand-alone diagnostic, eligibility, or return-to-play criterion. The higher reference-percentile proportions in power athletes strengthen the rationale for repeated, individualized assessment, but not for changing participation recommendations on the basis of these cross-sectional findings alone. Longitudinal studies are needed to determine whether the observed differences persist, diminish, or increase across puberty and whether they are associated with subsequent cardiac or vascular outcomes.

Limitations

The study benefits from a large sample of male youth athletes, standardized single-center assessments, simultaneous evaluation of peripheral and central hemodynamics, adjustment for major growth-related factors and training load, and a sensitivity analysis demonstrating that the findings were not materially altered by the predominance of soccer within the mixed-sport category. Several limitations nevertheless require consideration.

First, the cross-sectional design precludes causal inference. The observed differences may reflect training adaptation but also pre-existing characteristics that influence selection into particular sports. Longitudinal assessment beginning before sport specialization is required to distinguish adaptation from selection.

Second, the groups differed substantially in age, anthropometry, and likely biological maturation. Adjustment for chronological age and BSA cannot fully account for pubertal stage, growth velocity, hormonal status, lean mass, or fat mass [35]. Likewise, height-based SDS account for stature relative to the selected vascular reference curves but do not provide simultaneous adjustment for age, biological maturation, body composition, and training load. Limited overlap in age and body size between controls

and some athlete groups may also have introduced model-based extrapolation. Furthermore, BSA and training load may be both confounders and components of the sport-associated phenotype. Their inclusion improves comparability but may also attenuate meaningful pathways linking sport participation to hemodynamic outcomes.

Third, athletes and controls originated from different cohorts assessed between 2018 and 2024. Although identical equipment and standardized procedures were used at the same center, residual cohort effects related to recruitment, examination year, personnel, or unmeasured procedural differences cannot be excluded. The small control group and highly unequal athlete-group sizes also reduced the precision of some pairwise comparisons; non-significant findings involving controls should not be interpreted as evidence of equivalence.

Fourth, vascular parameters were based on a single oscillometric recording. Blood pressure is subject to within-person variability and may be influenced by recent training, hydration, sleep, emotional stress, caffeine intake, and time of day. Repeated measurements and ambulatory blood pressure monitoring would have provided more reliable estimates. Accordingly, classification at or above the 95th reference percentile does not establish a persistent abnormality.

Fifth, cSBP and PWV were derived from the same brachial waveform and algorithm. Although the Mobil-O-Graph PWV estimate has been validated against invasive measurements in pediatric and young populations [36], estimated PWV is not interchangeable with directly measured carotid–femoral PWV [37]. Shared waveform inputs and correlated measurement error also mean that cSBP and PWV are not fully independent outcomes. Direct PWV, wave-separation analysis, and measures of aortic impedance would permit a more detailed mechanistic interpretation.

Sixth, important hemodynamic and biological covariates were unavailable. These included pubertal stage, detailed body composition, stroke volume, cardiac output, systemic vascular resistance, autonomic function, plasma and red-cell volume, hemoglobin and hematocrit, dietary sodium, family history, sleep, and medication or supplement use. Their absence prevents determination of whether the cSBP differences were primarily related to ventricular ejection, volume status, vascular tone, wave reflection, or arterial wall properties.

Seventh, training exposure was self-reported, and the MET-hour index principally reflects metabolic expenditure. It does not capture contraction intensity, Valsalva use, short pressure peaks, work-to-rest patterns, or cumulative years of training. Categorizing entire disciplines as endurance, mixed, or power also simplifies heterogeneous real-world exposures.

Finally, the cluster analysis was exploratory and included only ten sports represented by at least ten participants. Validation methods yielded partly divergent recommendations, and the gap statistic provided no clear evidence of an underlying cluster structure. The three-cluster solution was selected partly on interpretability and requires external replication. Moreover, because the same vascular parameters were used both to construct and compare the clusters, subsequent group tests are

descriptive rather than independent confirmation of cluster validity. The labels “low-normal” and “high-normal” describe relative positions within this sample and should not be interpreted as clinical categories.

Only boys from a single German center were included, limiting generalizability to female athletes and other populations. In addition, the absence of endothelial, structural, cardiac target-organ, or histological measures precludes conclusions regarding vascular health or tissue-level remodeling.

5. Conclusion

After adjustment for age, BSA, and training load, power athletes exhibited higher cSBP than controls, endurance athletes, and mixed-sport athletes, while power and mixed-sport athletes showed higher bDBP than controls and endurance athletes. In contrast, no significant group differences remained for bSBP or estimated PWV. The findings therefore support a modest sport-associated central pressure phenotype but do not provide evidence of greater aortic stiffness in power athletes.

The exploratory sport-level clusters further suggest that vascular profiles are not fully explained by broad endurance, mixed, and power classifications. Central blood pressure may complement conventional brachial assessment by identifying differences in central hemodynamic load that are not apparent peripherally, but its pediatric prognostic and diagnostic relevance remains uncertain. Longitudinal multimodal studies incorporating biological maturation, body composition, cardiac output and stroke volume, blood-volume and hematological measures, repeated blood pressure assessment, and direct carotid–femoral PWV are needed to clarify the mechanisms, persistence, and clinical significance of these sport-specific hemodynamic profiles.

Abbreviations

ANCOVA, analysis of covariance

ANOVA, analysis of variance

BMI, body mass index

BSA, body surface area

bDBP, brachial diastolic blood pressure

bSBP, brachial systolic blood pressure

cSBP, central systolic blood pressure

LICO, LICO study

MET, metabolic equivalent of task

MuCAYA, MuCAYA study

MuCAYApplus, MuCAYApplus study

PWV, pulse wave velocity

SDS, standard deviation score

TUM, Technical University of Munich

VIF, variance inflation factor

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Ethics Committee of the Technical University of Munich. Ethical approval was obtained for the MuCAYA study (approval nos. 301/18 S and 131/19 S-SR), the MuCAYApplus study (approval no. 2023-426-S-DFG-SB), and the LICO study (approval no. 140/21 S-EB).

Consent for publication

Not applicable.

Competing interests

The authors have no conflict of interest to declare.

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Author Contribution

Conceptualization, methodology, and writing – original draft: LB, JHWriting – review and editing: MD, TE, SF, SH, FM, RO, TS

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Availability of data and materials

The datasets presented in this article are not readily available due to privacy and ethical restrictions. Requests to access the datasets should be directed to the corresponding author.

References

1. Jacobs DR Jr., et al. Childhood Cardiovascular Risk Factors and Adult Cardiovascular Events. *N Engl J Med*. 2022;386(20):1877–88.
2. Pahkala K, et al. Association of physical activity with vascular endothelial function and intima-media thickness. *Circulation*. 2011;124(18):1956–63.
3. Green DJ, et al. Vascular Adaptation to Exercise in Humans: Role of Hemodynamic Stimuli. *Physiol Rev*. 2017;97(2):495–528.
4. McEniery CM, et al. Central blood pressure: current evidence and clinical importance. *Eur Heart J*. 2014;35(26):1719–25.
5. Vlachopoulos C, et al. Prediction of cardiovascular events and all-cause mortality with central haemodynamics: a systematic review and meta-analysis. *Eur Heart J*. 2010;31(15):1865–71.
6. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. *J Am Coll Cardiol*. 2010;55(13):1318–27.
7. Elmenhorst J, et al. Percentiles for central blood pressure and pulse wave velocity in children and adolescents recorded with an oscillometric device. *Atherosclerosis*. 2015;238(1):9–16.
8. Mairbäurl H. Red blood cells in sports: effects of exercise and training on oxygen supply by red blood cells. *Front Physiol*. 2013;4:332.
9. Montero D, Lundby C. Regulation of Red Blood Cell Volume with Exercise Training. *Compr Physiol*. 2018;9(1):149–64.
10. MacDougall JD, et al. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol* (1985). 1985;58(3):785–90.
11. Miyachi M, et al. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation*. 2004;110(18):2858–63.
12. Souza RR, et al. Resistance training improves aortic structure in Wistar rats. *Braz J Phys Ther*. 2017;21(4):244–50.

13. Jaminon A et al. The Role of Vascular Smooth Muscle Cells in Arterial Remodeling: Focus on Calcification-Related Processes. *Int J Mol Sci*, 2019. 20(22).
14. Naylor LH, et al. Is there an athlete's artery? A comparison of brachial and femoral artery structure and function in male strength, power and endurance athletes. *J Sci Med Sport*. 2021;24(7):635–40.
15. Lona G, et al. Association of blood pressure, obesity and physical activity with arterial stiffness in children: a systematic review and meta-analysis. *Pediatr Res*. 2022;91(3):502–12.
16. Musselman JR, Price DE, Nelson VR. Participation in High-Static, Low-Dynamic Demand Sports Increases Prevalence of Elevated Blood Pressure in Adolescent Athletes. *Med Sci Sports Exerc*. 2026;58(3):405–9.
17. Willinger L, et al. Longitudinal development of central systolic blood pressure in young athletes. *J Sci Med Sport*. 2026;29(4):328–34.
18. Baumgartner L, et al. Influence of Vigorous Physical Activity on Structure and Function of the Cardiovascular System in Young Athletes - The MuCAYA-Study. *Front Cardiovasc Med*. 2019;6:148.
19. Haferanke J, et al. The MuCAYA(plus) Study-Influence of Physical Activity and Metabolic Parameters on the Structure and Function of the Cardiovascular System in Young Athletes. *CJC Open*. 2024;6(12):1549–57.
20. Harada M, et al. Echocardiographic Assessment of Cardiac Function and Reserve Under Exercise in Post-COVID-19 Children: A Prospective Cross-sectional Study. *CJC Pediatr Congenit Heart Dis*. 2024;3(5):203–13.
21. Pelliccia A et al. 2020 ESC Guidelines on sports cardiology and exercise in patients with cardiovascular disease: The Task Force on sports cardiology and exercise in patients with cardiovascular disease of the European Society of Cardiology (ESC). *European Heart Journal*, 2021. 42(1): pp. 17–96.
22. Dubois D, Dubois EF. A formula to estimate the approximate surface-area if height and weight be known. *Arch Intern Med*. 1989;17:863–71.
23. Neuhauser H, et al. Referenzperzentile für anthropometrische Maßzahlen und Blutdruck aus der Studie zur Gesundheit von Kindern und Jugendlichen in Deutschland (KiGGS). Berlin: Robert Koch-Institut; 2013.
24. Wassertheurer S, et al. A new oscillometric method for pulse wave analysis: comparison with a common tonometric method. *J Hum Hypertens*. 2010;24(8):498–504.
25. Elmenhorst J, et al. Comparison of Two Measurement Devices for Pulse Wave Velocity in Children: Which Tool Is Useful to Detect Vascular Alterations Caused by Overweight? *Front Pediatr*. 2019;7:334.
26. Woll A, et al. The German national cohort study on the development of motor performance, physical activity and health in children and adolescents: the MoMo 2.0-Study protocol. *BMJ Open*. 2025;15(4):e094895.
27. Ridley K, Ainsworth BE, Olds TS. Development of a Compendium of Energy Expenditures for Youth. *Int J Behav Nutr Phy*, 2008. 5(45).

28. Ainsworth BE, et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. *Med Sci Sport Exer.* 2011;43(8):1575–781.
29. Said Ouamer D, et al. Arterial stiffness assessed by pulse wave velocity in young athletes practicing endurance or intense resistance training. *Sci Sports.* 2023;38(3):266–73.
30. Vugrinec Mamić M, et al. Regular Dynamic Exercise Has a Beneficial Effect on Arterial Stiffness Already in Childhood and Adolescence. *Clin Pediatr (Phila).* 2025;64(7):944–54.
31. Kawano H, et al. Higher central arterial wall viscosity and stiffness in resistance-trained young men. *Physiol Rep.* 2025;13(17):e70526.
32. Zhang Y, et al. Effects of Moderate-Intensity Resistance Training on Vascular Endothelial Function and Arterial Stiffness in Young Healthy Men. *J Strength Cond Res.* 2025;39(8):e960–6.
33. Guillem CM et al. The Effects of Resistance Training on Blood Pressure in Preadolescents and Adolescents: A Systematic Review and Meta-Analysis. *Int J Environ Res Public Health*, 2020. 17(21).
34. de Simone G, et al. Hypertension in children and adolescents. *Eur Heart J.* 2022;43(35):3290–301.
35. Malina RM, et al. Biological maturation of youth athletes: assessment and implications. *Br J Sports Med.* 2015;49(13):852–9.
36. Walser M, et al. Oscillometric pulse wave velocity estimated via the Mobil-O-Graph shows excellent accuracy in children, adolescents and young adults: an invasive validation study. *J Hypertens.* 2023;41(4):597–607.
37. Spronck B, et al. 2024 Recommendations for Validation of Noninvasive Arterial Pulse Wave Velocity Measurement Devices. *Hypertension.* 2024;81(1):183–92.

Figures

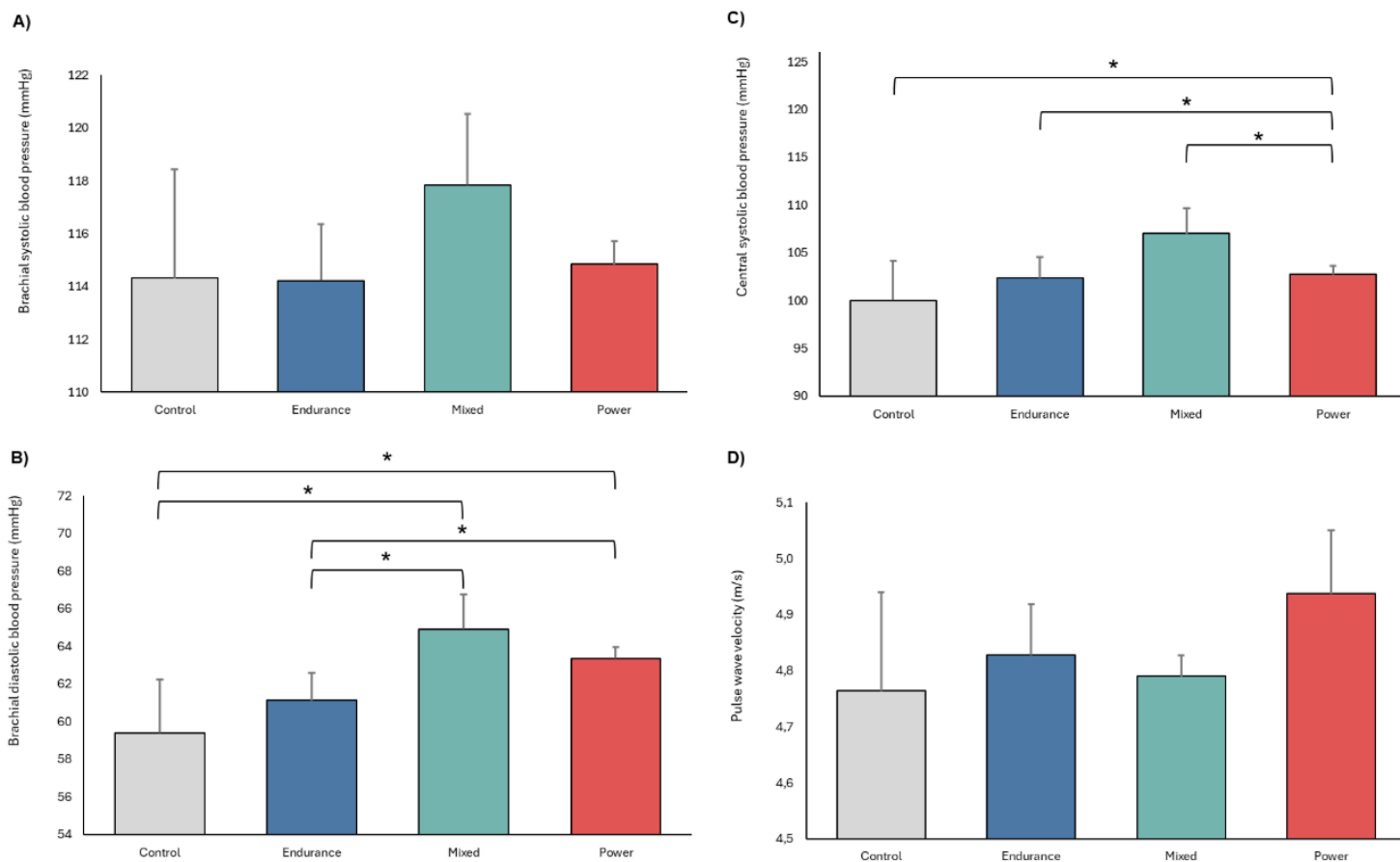


Figure 1

Adjusted marginal group means for brachial systolic (A) and diastolic blood pressure (B), central systolic blood pressure (C) and pulse wave velocity (D) for controls (n = 23), endurance (n = 72), mixed (n = 389), and power athletes (n = 43).

Figure legend: Error bars indicate the 95% confidence intervals of the adjusted marginal means. * p < 0.05.

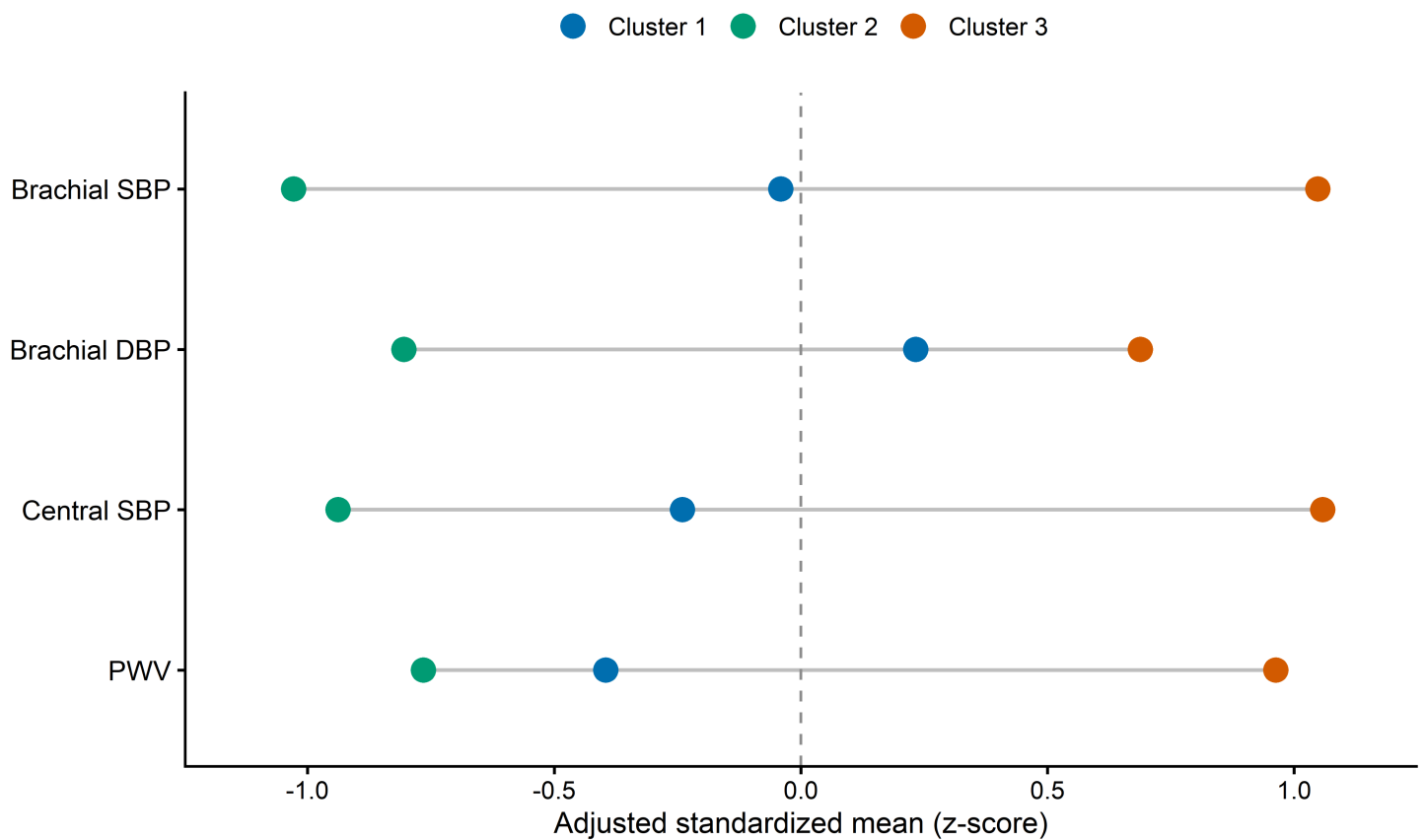


Figure 2

Cluster-specific vascular profiles based on adjusted standardized mean values.

Figure legend: Cluster profiles are based on age-, body surface area-, and training exposure-adjusted estimated marginal means for brachial systolic blood pressure (bSBP), brachial diastolic blood pressure (bDBP), central systolic blood pressure (cSBP), and pulse wave velocity (PWV). Adjusted values were standardized (z-scores) prior to hierarchical cluster analysis. Points represent the cluster-specific mean standardized values, and grey lines connect the three cluster means for each vascular parameter to facilitate comparison. The dashed vertical line indicates the overall standardized mean ($z = 0$).