



Skeletal muscle mass and device-estimated resting cardiac output in special medical group students: A cross-sectional study in Russia

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Abstract

Background: Special medical group (SMG) students at Russian universities are a heterogeneous population whose health conditions limit their participation in standard physical education. Integrated physiological characterisation remains limited, as earlier work has rarely combined body composition, hemodynamic, and autonomic data within a single protocol.

Aims: The study aimed to identify the body-composition variable most strongly and independently associated with device-estimated resting cardiac output and stroke volume in SMG students, while accounting for fat mass, visceral adipose tissue, phase angle, and body mass index.

Methods: In this cross-sectional study, 277 students aged 18-21 were assessed using the MS FIT diagnostic complex, which combines segmental bioimpedance, pulse-wave analysis, and short-term heart rate variability. Cardiac output and stroke volume were device-derived estimates. Spearman correlations, multiple linear regression, and tertile comparisons were computed in SPSS 26.0.

Result: The full five-predictor models explained 69.2% of the variance in cardiac output and 53.3% in stroke volume. Skeletal muscle mass had the largest standardized coefficient in both models (std. $\beta=0.84$ and 0.64 ; $p<.001$), and fat mass contributed independently to cardiac output (std. $\beta=0.41$; $p<.001$). Visceral adipose tissue and phase angle were not significant; body mass index was significant only for stroke volume.

Conclusion: Among the variables examined, skeletal muscle mass showed the strongest independent association with device-estimated resting cardiac output and stroke volume, with fat mass also contributing to cardiac output. These cross-sectional associations suggest that body mass index alone may be insufficient for describing the resting hemodynamic profile. Potential effects of skeletal muscle mass on resting hemodynamics require confirmation in longitudinal and interventional studies.

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INTRODUCTION

The health status of university students has become an increasingly important concern for specialists in physical education and sports science. Alongside the growing prevalence of sedentary behaviour, psychological stress, irregular sleep patterns, and insufficient physical activity,

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universities worldwide report a rising proportion of students presenting with functional limitations or chronic health conditions. These factors may negatively influence physical performance, exercise tolerance, and long-term cardiovascular health. Recent evidence indicates that cardiometabolic risk factors are already widespread among young adults, including elevated blood pressure and unfavourable body-composition profiles even among students with a normal body mass index (BMI) (Adetunji et al., 2025; Alibrahim et al., 2025; Almurdi, 2024; Rossi et al., 2015; Han & Lean, 2016).

From the perspective of coaching and physical education, these trends raise an important practical question. Traditional approaches to exercise prescription in university settings often rely on easily obtainable indicators such as body mass, BMI, resting heart rate, or performance in standard fitness tests. However, such indicators may not adequately reflect the physiological reserves available for exercise adaptation. Two students with similar body weight or BMI can differ substantially in muscular development, cardiovascular function, and autonomic regulation. Consequently, the identification of physiological markers that support more individualised exercise programming has become an important objective in sports science research.

In the Russian Federation, students whose health status limits participation in standard physical education programmes are assigned to the special medical group (SMG). This category includes students with cardiovascular, respiratory, musculoskeletal, metabolic, and other chronic conditions that require modifications to physical activity. The number of students assigned to SMG classes has increased over recent years, creating additional challenges for physical education instructors. Unlike competitive athletes, SMG students often demonstrate considerable heterogeneity in functional status, making the selection of appropriate training loads particularly difficult. Adaptive physical education approaches are increasingly explored to accommodate students with functional limitations (Berezina & Litvinova, 2026). For this reason, objective assessment methods that identify factors associated with cardiovascular function may have practical value for the individualisation of physical education programmes.

One of the most promising approaches is body-composition analysis using bioelectrical impedance analysis (BIA). Modern BIA systems provide rapid estimates of skeletal muscle mass, fat mass, visceral adipose tissue, body-water compartments, and phase angle (Moonen & Van Zanten, 2021; Siedler et al., 2023; Son et al., 2025). Compared with anthropometric measurements alone, these variables offer a more detailed description of the physiological characteristics relevant to exercise performance and health. Phase angle, for example, is considered an indirect indicator of cellular integrity and nutritional status and has been associated with cardiovascular risk in various populations (de Borja et al., 2022, 2024). Reference values for healthy adults have recently been established, facilitating interpretation in young populations (Akamatsu et al., 2022; Rosa et al., 2025).

Among body-composition variables, skeletal muscle mass may be particularly relevant from a coaching perspective. Skeletal muscle serves not only as the primary effector of movement but also as a major determinant of metabolic demand during both exercise and rest (Srikanthan & Karlamangla, 2014). Recent evidence suggests that resting cardiac output is closely related to the size of the skeletal muscle compartment in healthy young adults (Heinonen, 2025). Dlamini et al. (2025) demonstrated that appendicular skeletal muscle mass was independently associated with blood pressure in more than one thousand young women, even after adjustment for fat mass distribution. Similarly, Cvijetic et al. (2023) reported positive relationships between skeletal muscle indices and parasympathetic markers of heart-rate variability (HRV). More recent work has also examined how body composition relates to autonomic regulation in young adults (Páramo-Lira et al., 2026; Mabe-Castro et al., 2024). Collectively, these findings suggest that muscular development may influence not only physical performance but also cardiovascular and autonomic function.

Cardiac output and stroke volume are particularly relevant outcomes, as a higher stroke volume allows a given cardiac output to be maintained at a lower heart rate (Vincent J-L., 2008) – a pattern commonly seen in trained individuals. Understanding which body-composition characteristics are most strongly associated with resting hemodynamics may therefore help characterise the resting physiological profile of this population.

Despite growing interest in body composition and cardiovascular health, research involving SMG students remains limited. Existing Russian studies have predominantly relied on questionnaires, anthropometric measurements, or traditional fitness testing (Spataeva et al., 2024; Dorofieva et al., 2025). Bioimpedance analysis has been applied to SMG populations, but typically

without simultaneous assessment of cardiovascular and autonomic parameters (Terekhina et al., 2023). Other investigations have examined functional reserves and autonomic regulation in students with health deviations, yet these studies generally used separate measurement systems and focused on isolated physiological domains (Bocharin et al., 2023). As a result, relatively little is known about how body composition, hemodynamics, and autonomic regulation interact within this specific population. More specifically, although skeletal muscle mass has been linked to cardiovascular function in general and athletic populations, no previous study has examined which body-composition variable is most closely associated with device-estimated resting cardiac output in special medical group students – a population defined precisely by health-related exercise limitations, and therefore the group for whom individualised, physiologically grounded exercise guidance is most needed. Clarifying this relationship is directly relevant to coaching and physical education practice, where body composition is readily measurable yet rarely integrated into physiological monitoring.

To address this gap, the present study used an integrated diagnostic complex that combined bioelectrical impedance analysis, photoplethysmography-based hemodynamic assessment, and short-term heart rate variability in a single procedure, thereby reducing between-device variability and enabling evaluation of several physiological systems under identical conditions (Bocharin et al., 2022).

Therefore, the present study aimed to identify the body-composition variable that showed the strongest independent association with device-estimated resting cardiac output and stroke volume among first- to third-year medical students assigned to the special medical group. Skeletal muscle mass, fat mass, visceral adipose tissue, phase angle, and BMI were considered as candidate variables. These five were selected because they represent the principal body-composition parameters routinely generated by segmental bioimpedance systems and have each been previously associated with cardiovascular function. Rather than aiming to determine training targets, the study was intended to provide population-specific evidence for physiological monitoring and to support hypothesis development for future intervention studies in special medical group students. Based on prior evidence, it was hypothesised that skeletal muscle mass would exhibit the strongest independent association with these device-estimated hemodynamic parameters.

METHOD

Research Design

This research employed a cross-sectional observational design based on a retrospective secondary analysis of de-identified routine health-monitoring data, conducted in accordance with the STROBE statement for observational studies. No intervention was administered. Each record corresponded to a single assessment performed at one time point, yielding one set of measurements per student. The study was conducted at Privolzhsky Research Medical University (Nizhny Novgorod, Russia) between January and May 2026.

Participant

In the Russian higher education system, students are assigned to a special medical group (SMG) when chronic health conditions or functional limitations require modification of their physical education programme. Participants were undergraduate medical students of years 1 to 3, assigned by the university medical board to the special medical group – a category formed for students whose health conditions limit their participation in regular physical education classes. Inclusion criteria were: chronological age between 18 and 21 years, current enrolment in the medical curriculum, and formal placement in the special medical group. Records were excluded if the individual had an acute infectious or inflammatory condition on the testing day, carried an implanted electronic device (pacemaker, cardioverter), or had an incomplete measurement protocol. This study is based on a secondary analysis of de-identified data obtained through routine functional health monitoring of special medical group students at Privolzhsky Research Medical University between January and May 2026. Approval for the retrospective analysis of these anonymized records for research purposes was granted by the Local Ethics Committee of Privolzhsky Research Medical University (Protocol No. 7, dated 3 July 2026). No personally identifiable information was accessed or reported. Because the study used only de-identified secondary data, individual informed consent was not required under

the applicable institutional and national regulations. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Population and Sampling

The target population was medical undergraduates in years 1 to 3 who were assigned to the special medical group during the 2025–2026 academic year. All eligible records collected during the study period were screened according to the inclusion and exclusion criteria. From 327 initial records, 30 identical duplicates were removed, and a further 20 records were excluded on a priori quality-control criteria, leaving 277 students for analysis. The sample included both male and female students.

Instrument

All measurements were obtained using a multi-parameter touch-sensor diagnostic complex, MS FIT (MedicalSoft LLC, Moscow, Russia). This device integrates three measurement technologies in a single 60-second session: (1) Tetrapolar bioelectrical impedance analysis (BIA) via hand- and foot-platform electrodes, providing body composition and hydration variables – body weight, BMI, skeletal muscle mass, total and trunk fat mass, visceral adipose tissue (VAT; reported in manufacturer-defined device units), bone mineral mass, total body water, intracellular and extracellular water, phase angle, basal metabolic rate, and device-estimated biological and metabolic age. VAT is reported using the manufacturer's proprietary device scale and is therefore presented in device units rather than square centimetres. (2) Digital pulse wave analysis (DPWA) based on photoplethysmography, yielding systolic and diastolic blood pressure, pulse pressure, resting heart rate, cardiac output (CO), stroke volume (SV), cardiac index (CI), and indices of arterial stiffness. (3) Short-term heart rate variability (HRV) analysis following the Baevsky framework standard in Russian functional diagnostics – SDNN, total spectral power (TP), high-frequency (HF) and low-frequency (LF) power, LF/HF ratio, stress index, and the MxDMn interval difference. The HRV indices were derived from an ultra-short 60–90-second recording. It should be noted that frequency-domain indices (LF, HF, LF/HF, and total power) have been standardised for recordings of at least five minutes ([Shaffer & Ginsberg, 2017](#); [Munoz et al., 2015](#)), and the manufacturer does not provide device-specific validation of these indices for ultra-short recordings; the frequency-domain HRV results were therefore treated as exploratory and interpreted with caution.

The MS FIT system is a certified medical device registered for clinical and wellness use in the Russian Federation. The validity and reliability of bioimpedance analysis for body composition assessment have been demonstrated in previous studies ([Moonen & Van Zanten, 2021](#); [Siedler et al., 2023](#); [Musijowska & Kwilosz, 2024](#)). Cardiac output and stroke volume are device-derived estimates obtained from photoplethysmographic pulse-wave analysis rather than reference-standard measurements. They are produced by combining the pulse-wave signal with entered anthropometric and blood-pressure data, using proprietary algorithms not disclosed by the manufacturer. These estimates were therefore not independently validated against reference methods (e.g., echocardiography or thermodilution) in the present study and should be interpreted as device-derived estimates suitable for within-sample comparison rather than as absolute measurements. This is addressed further in the Limitations. The use of short-term HRV analysis and pulse-wave-derived hemodynamic indices has also been widely reported in physiological and cardiovascular research.

Procedures and Time Frame

Data were collected between January and May 2026. To minimise variability due to circadian rhythms and pre-testing behaviour, all measurements were conducted in the morning, between 9:00 and 12:00. Each participant was instructed to refrain from food intake for at least two hours and from caffeine, nicotine, or vigorous physical activity for at least twelve hours before the assessment. Immediately before measurement, students emptied their bladders and then rested seated for 5 minutes in a quiet, temperature-controlled room maintained at 22 ± 2 °C. Measurement was then performed with the student standing barefoot on the platform, dressed in light indoor clothing, with both hands placed flat on the sensor pads. The full BIA–DPWA–HRV protocol lasted 60 to 90 seconds per student. A single operator supervised all sessions to ensure consistent positioning and contact.

Before analysis, the dataset underwent quality control. Identical duplicate records ($n=30$) were removed. Before analysis, records were screened for obvious technical artefacts. Measurements showing physiologically implausible HRV values ($SDNN < 10$ ms or $TP < 50$ ms²) or biological-age estimates outside the manufacturer's plausible operating range for this population (<10 or >50 years), applied as a prespecified quality-control criterion, were considered invalid and excluded from further analysis. Applying these criteria excluded an additional 20 records.

For descriptive purposes, several established clinical categories were applied to the cleaned dataset. BMI was classified according to WHO categories: underweight (<18.5 kg/m²), normal weight (18.5 – 24.9 kg/m²), overweight (25.0 – 29.9 kg/m²), and obesity (≥ 30 kg/m²). Elevated blood pressure was defined as $SBP \geq 130$ mmHg or $DBP \geq 80$ mmHg, a commonly applied threshold in young-adult cardiovascular research. Resting tachycardia was defined as $HR > 100$ bpm. Two HRV-based descriptive categories were also applied: $SDNN$ below 30 ms, often associated with reduced parasympathetic tone in young adults, and Baevsky stress index above 200, a value treated in the Russian functional-diagnostics tradition as the boundary between balanced and sympathetically dominated regulation. These categories were used for descriptive purposes only and were not entered as variables in the regression or tertile analyses.

Analysis Plan

Statistical analysis was performed in IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Distribution of continuous variables was checked using the Shapiro–Wilk test, accompanied by visual inspection of histograms and Q-Q plots. Because most variables deviated from normality, descriptive statistics are presented as mean $\pm$ standard deviation ($M \pm SD$), supplemented by median and range; bivariate associations were tested using Spearman's rank correlation coefficient (ρ). To identify independent predictors of the four primary hemodynamic outcomes – cardiac output, stroke volume, systolic blood pressure, and diastolic blood pressure – we used multiple linear regression with the enter method, including skeletal muscle mass, fat mass, visceral adipose tissue, phase angle, and BMI as simultaneous predictors. The enter method was chosen because the objective was to estimate the independent contribution of all pre-specified body-composition variables simultaneously, rather than to build a parsimonious predictive model. For each model, we report unstandardized (β) and standardized (β^*) regression coefficients, standard errors, t-statistics, p-values, R^2 , and adjusted R^2 . Variance inflation factors were calculated to assess multicollinearity, and no problematic multicollinearity was detected (all values were below the conventional threshold of 5). Regression assumptions were examined using model residuals. The normality of residuals was assessed using the Shapiro–Wilk test and Q-Q plots, independence using the Durbin–Watson statistic, and homoscedasticity using the Breusch–Pagan test. Residuals of the cardiac output model were approximately normally distributed (Shapiro–Wilk $p=0.55$) and independent (Durbin–Watson=1.80). Because the Breusch–Pagan test indicated heteroscedasticity ($p=0.005$), the cardiac output model was re-estimated using heteroscedasticity-consistent (HC3) robust standard errors as a sensitivity analysis; under robust standard errors, skeletal muscle mass and fat mass both remained statistically significant ($p < 0.001$), confirming that heteroscedasticity did not alter the conclusions (reported in the Results). Body size, sex, and habitual physical activity were considered as potential confounders. Sex could not be included as a covariate because it was not coded in the de-identified dataset, and habitual physical activity was not measured using a validated instrument. To address the influence of overall body size, a complementary regression was performed using cardiac index as the outcome, allowing the muscle–hemodynamic association to be examined independently of body dimensions. Cardiac index was generated directly by the MS FIT system and was not calculated by the researchers; the manufacturer does not disclose the body-surface-area formula used internally by the device.

For an additional comparison across muscle mass levels, students were stratified into tertiles using the empirical 33rd and 67th percentiles (cut-offs of 24.6 kg and 35.2 kg). Between-tertile differences in hemodynamic and HRV parameters were tested using the Kruskal–Wallis H test, followed by Dunn's post-hoc pairwise comparisons with Bonferroni correction. A sensitivity analysis for the primary cardiac output regression model was conducted after excluding participants with VAT values above the 95th percentile, to verify that extreme visceral adiposity values did not drive the regression estimates. Additionally, influential-case diagnostics based on Cook's distance were

computed for the cardiac output model, and the model was re-estimated after excluding any observation exceeding the conventional cut-off, to confirm the robustness of the estimates.

Effect sizes for the regression models were expressed as Cohen's f^2 , using the conventional thresholds of 0.02, 0.15, and 0.35 for small, medium, and large effects. Categorical proportions reported in the descriptive section of the Results were calculated as simple percentages of the analytic sample ($n=277$) without inferential testing. Throughout, the significance threshold was set at $p<0.05$ (two-tailed).

Limitations of the Methodology

Several methodological limitations should be noted. The cross-sectional design captures associations between body composition and cardiovascular variables at a single point in time; causal inferences are therefore not available. All parameters were obtained from a single integrated device. Any systematic measurement bias would therefore affect both predictors and outcomes; conversely, this approach ensures greater within-device consistency than a multi-device setup. Habitual physical activity was not recorded using a validated questionnaire (e.g., IPAQ) or objective monitoring, which limits the interpretation of skeletal muscle mass as a marker of training status. Participant sex was not included as a covariate because it was not coded in the available dataset; this issue is addressed further in the Limitations section of the Discussion. Finally, the sample was drawn from a single university and a specific subgroup (special medical group students); therefore, generalisation beyond this population should be undertaken with caution. Interaction terms between body-composition predictors were not modelled, as they were beyond the scope of the present analysis.

RESULTS AND DISCUSSION

Results

The final analytic sample consisted of 277 first- to third-year medical students of the special medical group, aged 18 to 21 years. Sample characteristics across anthropometric, body composition, hemodynamic, and autonomic domains are summarised in Table 1. Mean body weight was 65.30 ± 16.63 kg and mean BMI was 22.33 ± 4.96 kg/m². The BMI distribution by WHO categories was uneven: 17.3% of participants were classified as underweight (BMI<18.5), 64.3% as normal weight, 12.6% as overweight, and 5.8% as obese. This profile reflects the heterogeneity of the SMG category, which includes students with diverse health limitations. The full BMI range extended from 14.3 to 53.6 kg/m².

Body composition variables showed comparable heterogeneity. Mean skeletal muscle mass was 30.32 ± 8.53 kg, with values ranging from 14.4 to 54.0 kg, representing almost a fourfold difference between the lowest and highest observed values. Mean fat mass was 15.49 ± 8.15 kg. Phase angle, the bioimpedance marker of cellular integrity (Akamatsu et al., 2022), averaged $8.93\pm1.94^\circ$, which is within the upper range of published reference values for this age group. Visceral adipose tissue values showed a markedly skewed distribution (median = 8.2, IQR = 6.2–16.9, maximum = 192.9 device units), indicating a small subgroup of participants with substantially elevated visceral adiposity. This wide dispersion, particularly the near-fourfold range in skeletal muscle mass, suggests that participants sharing the SMG designation may nonetheless differ markedly in their underlying physiological profile. This skewness was consistent with the general non-normality of most variables in the sample; non-parametric methods were therefore used for the descriptive, correlational, and tertile analyses, while the assumptions of the multivariable regression were evaluated using model residuals. Resting hemodynamics, while within accepted physiological limits on average, were not uniformly favourable. Mean systolic blood pressure was 124.47 ± 12.95 mmHg, mean diastolic 74.44 ± 9.25 mmHg, and mean resting heart rate 87.95 ± 16.44 bpm. Applying a threshold of 130/80 mmHg for elevated blood pressure, 54.5% of participants exceeded at least one of the two cut-offs. Resting tachycardia (HR>100 bpm) was present in 23.1% of the sample. These proportions indicate that a substantial share of the cohort presented with resting cardiovascular values above conventional reference thresholds. Device-estimated cardiac output averaged 5.83 ± 1.11 L/min and stroke volume 69.12 ± 19.34 mL.

Autonomic regulation, assessed through short-term HRV indices, showed similar within-group variability. Mean SDNN was 45.79 ± 14.41 ms, with 14.1% of participants below 30 ms – a level often associated with reduced parasympathetic tone in young adults. The Baevsky stress index

averaged 182.56 ± 126.77 , and 32.5% of the sample exceeded 200, which the Russian functional-diagnostics tradition treats as a marker of centralised, sympathetically driven regulation. Total spectral power averaged $1427.85 \pm 989.25 \text{ ms}^2$, and the LF/HF ratio averaged 1.47 ± 0.63 . Taken together, the widespread distribution of these HRV indices and the sizeable minority with reduced SDNN or elevated stress index suggest that autonomic regulation varied considerably across the cohort rather than clustering around a single typical value. The wide ranges observed across all four domains reinforce the impression of a clinically heterogeneous cohort and underscore the relevance of analysing body-composition correlates of cardiac output rather than relying on group means alone.

Bivariate Spearman correlations between body-composition variables and cardiovascular outcomes are presented in Table 2. The strongest bivariate associations were observed for skeletal muscle mass: $\rho=0.74$ with cardiac output, $\rho=0.66$ with stroke volume, $\rho=0.42$ with pulse pressure, and $\rho=0.40$ with systolic blood pressure (all $p<.001$). Correlation with diastolic blood pressure was essentially zero ($\rho=0.02$), and correlation with resting heart rate was negative ($\rho=-0.23$, $p<.001$), indicating that greater muscle mass was associated with a slower resting heart rate and higher stroke volume.

Two other variables showed unexpectedly strong bivariate correlations with central hemodynamics. BMI correlated with cardiac output ($\rho=0.66$) and stroke volume ($\rho=0.63$; both $p<.001$), values nearly as high as those for muscle mass. Visceral adipose tissue correlated at $\rho=0.49$ (CO) and $\rho=0.50$ (SV). At the bivariate level, therefore, several body-composition variables appeared to be associated with central hemodynamics, suggesting overlapping information among them rather than several fully independent relationships. Phase angle and fat mass produced weaker associations. Phase angle correlated with CO at $\rho=0.36$ and with SV at $\rho=0.41$ (both $p<.001$); fat mass correlated with CO at $\rho=0.20$ ($p<.001$) but accounted for very little of the variance in either stroke volume ($\rho=0.15$) or systolic pressure ($\rho = 0.01$, ns). Several variables were also moderately correlated with each other. The cross-correlation between BMI and VAT reached $\rho=0.67$, and BMI overlapped with both fat mass ($\rho=0.59$) and phase angle ($\rho=0.59$). Muscle mass correlated with BMI ($\rho=0.41$) and with phase angle ($\rho=0.37$). This pattern raised the standard concern of multicollinearity, which the multivariable analysis (Table 3) addresses directly by partitioning shared variance among the five candidate variables.

Multiple linear regression models tested the independent contribution of each body-composition variable to four hemodynamic outcomes: cardiac output, stroke volume, systolic and diastolic blood pressure (Table 3).

Table 1. Descriptive Characteristics of the Study Sample (n=277)

Variable	M	SD	Min	Max	Median	IQR
Anthropometrics						
Body weight (kg)	65.3	16.63	32.0	122.0	64.0	55.0-75.0
BMI (kg/m^2)	22.33	4.96	14.3	53.6	21.4	19.5-24.2
Body composition						
Fat mass (kg)	15.49	8.15	4.1	53.0	14.5	9.0-19.9
Muscle mass (kg)	30.32	8.53	14.4	54.0	29.3	23.1-37.4
Visceral adipose tissue (device units)	21.47	32.16	4.1	192.9	8.2	6.2-16.9
Phase angle ($^\circ$)	8.93	1.94	3.7	14.1	8.8	7.4-10.2
Hemodynamics						
Systolic blood pressure (mmHg)	124.4	12.95	90.0	166.0	120.0	119.0-131.0
Diastolic blood pressure (mmHg)	74.44	9.25	50.0	110.0	75.0	68.0-80.0
Pulse pressure (mmHg)	50.02	12.14	27.0	89.0	49.0	40.0-57.0
Resting heart rate (bpm)	87.95	16.44	53.6	144.2	86.4	75.8-99.5
Cardiac output (L/min)	5.83	1.11	3.4	9.5	5.9	5.0-6.5
Stroke volume (mL)	69.12	19.34	31.6	176.7	67.4	56.0-78.8

Variable	M	SD	Min	Max	Median	IQR
Cardiac index (L/min/m ²)	3.33	0.3	2.5	4.5	3.3	3.1-3.5
Heart rate variability						
SDNN (ms)	45.79	14.41	13.8	101.0	45.7	35.2-54.3
MxDMn (ms)	212.73	55.78	97.0	419.0	217.0	170.0-247.0
Stress index (y.e.)	182.56	126.77	26.0	869.0	145.0	96.0-228.0
TP (ms ²)	1427.85	989.25	62.0	6905.0	1330.0	747.0-1868.0
LF/HF (y.e.)	1.47	0.63	0.1	3.0	1.3	0.9-2.1
HF (%)	28.09	7.05	15.5	54.0	27.3	22.6-32.7
LF (%)	38.12	11.46	3.6	66.4	36.1	28.9-47.4

Note. M = mean; SD = standard deviation. The Shapiro–Wilk test indicated non-normal distributions for most variables. Non-parametric methods (Spearman correlations, Kruskal–Wallis tests) were therefore used for bivariate and between-group analyses. The multivariable linear regression is a parametric method whose validity was assessed through model residuals rather than the distribution of individual variables

Table 2. Spearman Correlations (ρ) between Body Composition Variables and Hemodynamic Outcomes (n=277)

Predictor	SBP	DBP	PP	HR	CO	SV	CI
Muscle mass	0.4***	0.02	0.42***	-0.23***	0.74***	0.66***	0.33***
Visceral adipose tissue	0.36***	0.34***	0.09	-0.21***	0.49***	0.5***	0.02
Phase angle	0.2**	0.17**	0.03	-0.16**	0.36***	0.41***	-0.06
BMI	0.27***	0.34***	0.04	-0.19**	0.66***	0.63***	0.06
Fat mass	0.01	0.34***	-0.25***	0.05	0.2***	0.15*	-0.11

Note. SBP = systolic blood pressure; DBP = diastolic blood pressure; PP = pulse pressure; HR = resting heart rate; CO = cardiac output; SV = stroke volume; CI = cardiac index. * $p < .05$, ** $p < .01$, *** $p < .001$. Strongest associations are highlighted in bold

For cardiac output, the full five-predictor regression model explained 69.2% of the variance ($R^2 = .692$; adjusted $R^2 = .687$). Within this model, skeletal muscle mass had the largest standardized coefficient ($\beta = 0.109$; $\beta^* = 0.84$; $t = 16.93$; $p < .001$) – more than twice as large as that of the next contributor, fat mass ($\beta = 0.055$; $\beta^* = 0.41$; $t = 5.73$; $p < .001$), which also remained independently significant. Visceral adipose tissue, phase angle, and BMI were not significant after adjustment. The strong bivariate associations between BMI and device-estimated cardiac output ($\rho = 0.66$) and between VAT and cardiac output ($\rho = 0.49$) reported in Table 2 were therefore consistent with substantial shared variance among the predictors rather than with independent contributions to cardiac output. This pattern reflects the statistical attenuation observed after simultaneous adjustment and does not, by itself, identify which specific predictor accounts for that shared variance.

For stroke volume, the full-model R^2 was .533 (adjusted $R^2 = .525$), and skeletal muscle mass again had the largest standardized coefficient ($\beta = 1.454$; $\beta^* = 0.64$; $p < .001$). BMI retained a small but statistically significant association with stroke volume ($\beta = 0.839$; $\beta^* = 0.22$; $p = .036$), while fat mass, VAT, and phase angle did not. The systolic blood pressure model explained considerably less variance ($R^2 = .195$; adjusted $R^2 = .180$), with skeletal muscle mass the only significant variable ($\beta^* = .31$; $p < .001$). The diastolic blood pressure model showed the weakest fit ($R^2 = .156$; adjusted $R^2 = .140$), and BMI was the only significant variable ($\beta^* = .29$; $p = .035$). The comparatively low variance explained for both blood-pressure outcomes indicates that the body-composition variables examined were much more closely associated with cardiac output and stroke volume than with resting blood pressure. The Cohen's f^2 effect size for the cardiac output model was 2.25, well above the threshold

for a large effect. Because the Breusch-Pagan test had indicated heteroscedasticity, the cardiac output model was re-estimated with heteroscedasticity-consistent (HC3) robust standard errors; under this specification, skeletal muscle mass and fat mass both remained statistically significant ($p < .001$), confirming that the results were not affected by non-constant error variance. Influential-case diagnostics identified one observation exceeding a Cook's distance of 1.0 (a participant with extreme obesity, BMI=53.6); re-estimating the model without this case left the results essentially unchanged (skeletal muscle mass $\beta^* = 0.79$ versus 0.84; adjusted $R^2 = 0.70$), confirming that individual influential observations did not drive the findings. A further sensitivity analysis excluding participants with visceral adipose tissue above the 95th percentile also left the pattern unchanged, with skeletal muscle mass remaining the predominant correlate ($\beta = 0.80$) and fat mass the second ($\beta = 0.35$; $R^2 = 0.686$). Together, these checks indicate that the leading role of skeletal muscle mass was stable across model specifications and was not produced by a small number of extreme observations.

To determine whether the association between skeletal muscle mass and cardiac output reflected overall body size, an additional regression was performed with device-estimated cardiac index as the outcome and the same five body-composition predictors (Table 4). Skeletal muscle mass had the largest standardized coefficient ($\beta = 0.015$; $\beta^* = 0.42$; $t = 5.15$; $p < .001$), while fat mass, visceral adipose tissue, and BMI were not significant. Phase angle showed a small but statistically significant negative association with cardiac index ($\beta = -0.16$; $p = .031$), the only variable besides muscle mass to reach significance in this model. The bivariate correlation between skeletal muscle mass and cardiac index remained significant ($\rho = 0.33$; $p < .001$), while that between BMI and cardiac index was negligible and non-significant ($\rho = 0.06$; $p = .29$). This contrast may suggest that the muscle-cardiac-output association was less dependent on overall body size than the BMI association, although the present analysis cannot establish this directly. The model explained less variance than the model for absolute cardiac output ($R^2 = 0.157$; adjusted $R^2 = 0.141$), as expected when the body-size component shared by all predictors is removed through indexing.

Table 3. Multiple Linear Regression Models Predicting Hemodynamic Outcomes (n=277)

Outcome / Predictor	β	95% CI	β^*	SE	t	p
Cardiac output ($R^2 = .692$, adj. $R^2 = .687$)						
Muscle mass	0.109	[0.096, 0.121]	0.84	0.006	16.93	< .001
Fat mass	0.055	[0.036, 0.074]	0.41	0.01	5.73	< .001
VAT	0.001	[-0.002, 0.004]	0.02	0.002	0.46	.649
Phase angle	-0.015	[-0.064, 0.034]	-0.03	0.025	-0.59	.553
BMI	0.000	[-0.036, 0.037]	0.00	0.019	0.02	.983
Stroke volume ($R^2 = .533$, adj. $R^2 = .525$)						
Muscle mass	1.454	[1.183, 1.725]	0.64	0.138	10.57	< .001
BMI	0.839	[0.053, 1.624]	0.22	0.399	2.1	.036
Fat mass	0.206	[-0.201, 0.613]	0.09	0.207	1.0	.320
VAT	0.01	[-0.056, 0.077]	0.02	0.034	0.31	.760
Phase angle	-0.283	[-1.334, 0.767]	-0.03	0.533	-0.53	.596
Systolic blood pressure ($R^2 = .195$, adj. $R^2 = .180$)						
Muscle mass	0.472	[0.234, 0.710]	0.31	0.121	3.9	< .001
VAT	0.048	[-0.011, 0.106]	0.12	0.03	1.61	.109
BMI	0.487	[-0.203, 1.177]	0.19	0.351	1.39	.166
Phase angle	-0.459	[-1.383, 0.464]	-0.07	0.469	-0.98	.328
Fat mass	-0.068	[-0.426, 0.290]	-0.04	0.182	-0.37	.709
Diastolic blood pressure ($R^2 = .156$, adj. $R^2 = .140$)						
BMI	0.544	[0.039, 1.048]	0.29	0.256	2.12	.035
Fat mass	0.097	[-0.165, 0.359]	0.08	0.133	0.73	.468
Muscle mass	-0.087	[-0.261, 0.087]	-0.08	0.088	-0.99	.325
Phase angle	0.226	[-0.450, 0.901]	0.05	0.343	0.66	.511
VAT	0.009	[-0.034, 0.052]	0.03	0.022	0.42	.675

Note. β = unstandardized regression coefficient; β^* = standardized coefficient; SE = standard error.

Table 4. Multiple Linear Regression Predicting Device-Estimated Cardiac Index (n=277)

Predictor	β	95% CI	β^*	SE	t	p
Muscle mass	0.015	[0.009, 0.02]	0.42	0.003	5.15	<.001
Fat mass	0.002	[-0.007, 0.01]	0.04	0.004	0.36	.718
VAT	0.001	[-0.001, 0.002]	0.08	0.001	1.04	.298
Phase angle	-0.024	[-0.046, -0.002]	-0.16	0.011	-2.17	.031
BMI	-0.008	[-0.024, 0.009]	-0.13	0.008	-0.91	.364

Note. $R^2=.157$; adjusted $R^2=.141$. β = unstandardized coefficient; β^* = standardized coefficient; SE = standard error. Cardiac index was generated directly by the MS FIT system.

The association between skeletal muscle mass and cardiac output is illustrated in Figure 1. The figure shows a clear positive gradient: cardiac output increases steadily across the range of muscle mass, and the concentration of points around the fitted line reflects the strength of this association ($\rho=0.74$). Figure 2 summarises the multivariable model and visually conveys its central finding: only skeletal muscle mass and fat mass have confidence intervals that exclude zero, whereas visceral adipose tissue, phase angle, and BMI cluster around the no-effect line. The much greater distance of the muscle-mass coefficient from zero, relative to all other predictors, is the key pattern of the analysis.

For the exploratory tertile comparison, participants were stratified into low (≤ 24.6 kg, $n=94$), medium (24.7-35.1 kg, $n=91$), and high (≥ 35.2 kg, $n=92$) muscle-mass categories. Cardiac output, stroke volume, and resting heart rate all showed a clear gradient across tertiles (Table 5). Cardiac output rose from 5.00 L/min in the low tertile to 6.86 L/min in the high tertile (Kruskal-Wallis H, $p<.001$) – an absolute difference of 1.86 L/min, or 37% relative to the low tertile mean. Stroke volume showed an even steeper gradient (56.77 mL – 65.62 mL – 85.20 mL, $p<.001$), corresponding to a 50% gap between the extreme tertiles. Resting heart rate moved in the opposite direction (91.29 – 89.10 – 83.39 bpm, $p=.001$), with an absolute decrease of 7.9 bpm between the low and high tertiles. Descriptively, therefore, the higher muscle-mass tertiles were characterised by a higher stroke volume together with a lower resting heart rate, which is consistent with the negative muscle–heart-rate correlation reported above. Systolic blood pressure also rose across tertiles (119.27 – 122.58 – 131.64 mmHg, $p<.001$), although the magnitude of this gradient was smaller than for the output indices. Diastolic blood pressure showed no significant tertile effect ($p = .433$).

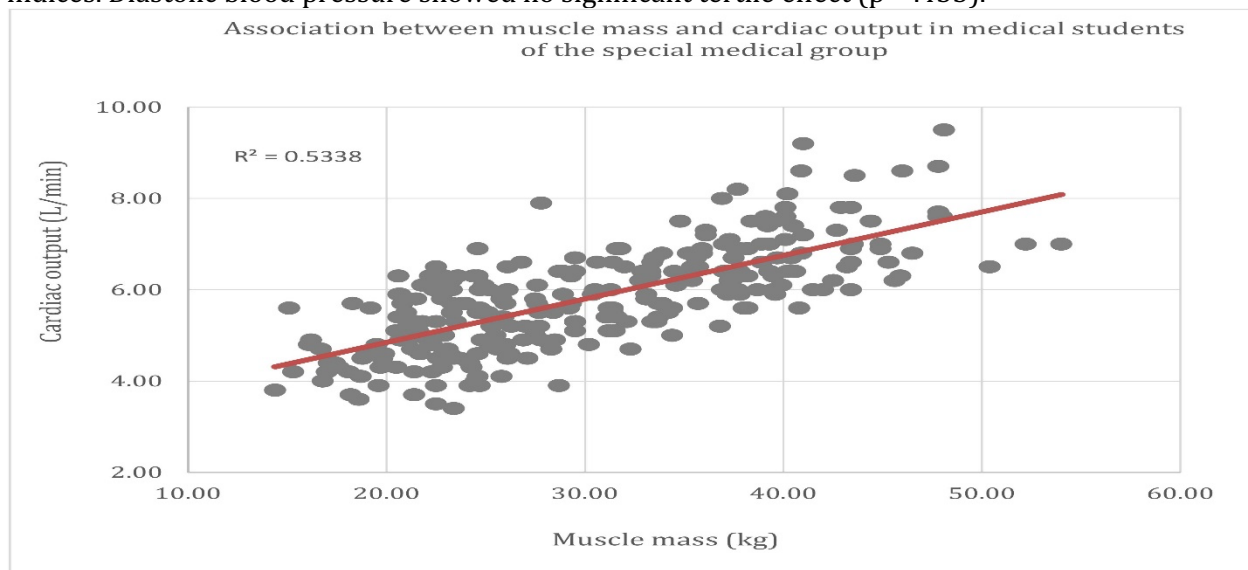


Figure 1. Relationship between skeletal muscle mass and cardiac output in special medical group students ($n=277$). Each point represents one participant; the solid line indicates the fitted least-squares linear regression. Spearman's correlation coefficient: $\rho=0.74$, $p<.001$

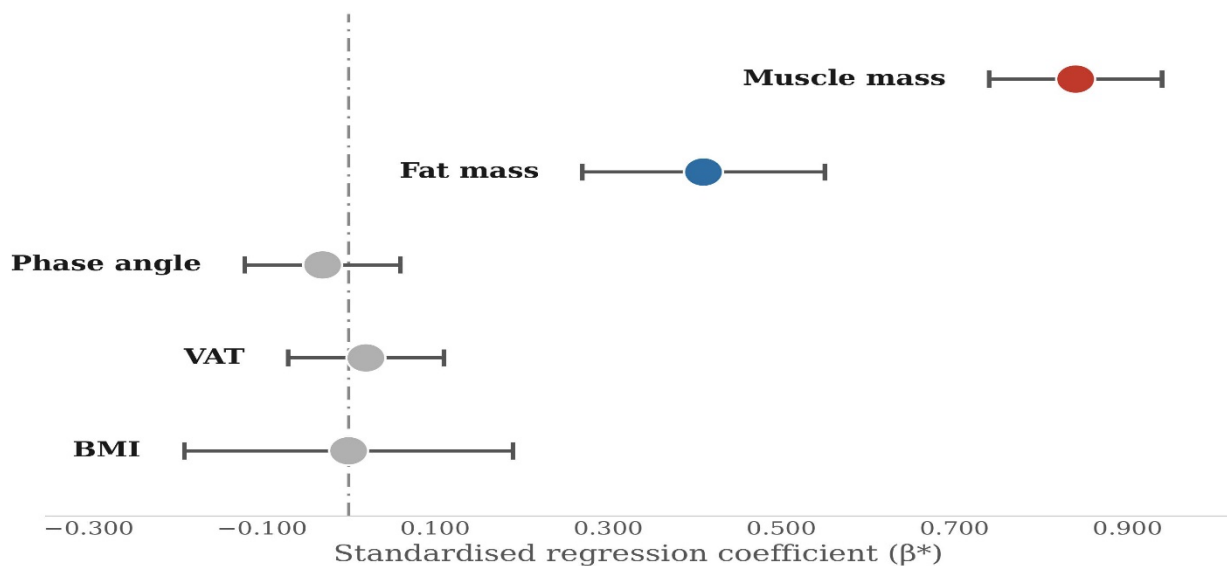


Figure 2. Forest plot of standardized regression coefficients (β^*) with 95% confidence intervals for the five body-composition predictors in the cardiac output regression model ($n=277$). Coloured markers denote statistically significant predictors (red – skeletal muscle mass; blue – fat mass; both $p<.001$); grey markers denote non-significant predictors (phase angle, visceral adipose tissue, and body mass index). The vertical dashed line at $\beta^*=0$ indicates no effect; predictors whose confidence intervals cross this line are not statistically significant

HRV indices trended in the direction expected from a parasympathetic-favouring profile at higher muscle mass – SDNN rose from 43.13 ms to 48.11 ms, and the Baevsky stress index fell from 192.88 to 170.74 – but neither contrast reached significance ($p=.072$ and $.081$ respectively). These autonomic trends were therefore in the same direction as the hemodynamic findings but should be regarded as non-significant.

Table 5. Cardiovascular and Autonomic Parameters Across Tertiles of Skeletal Muscle Mass

Parameter	Low ≤ 24.6 kg ($n=94$)	Medium 24.7–35.1 kg ($n=91$)	High ≥ 35.2 kg ($n=92$)	p
SBP (mmHg)	119.27 $\pm$ 12.39 ^a	122.58 $\pm$ 10.72 ^a	131.64 $\pm$ 12.43 ^b	<.001
DBP (mmHg)	74.70 $\pm$ 9.58	73.75 $\pm$ 8.74	74.87 $\pm$ 9.46	.433
Resting HR (bpm)	91.29 $\pm$ 15.15 ^a	89.10 $\pm$ 16.68 ^{ab}	83.39 $\pm$ 16.62 ^b	.001
Cardiac output (L/min)	5.0 $\pm$ 0.83 ^a	5.65 $\pm$ 0.75 ^b	6.86 $\pm$ 0.82 ^c	<.001
Stroke volume (mL)	56.77 $\pm$ 11.32 ^a	65.62 $\pm$ 14.05 ^b	85.20 $\pm$ 19.44 ^c	<.001
SDNN (ms)	43.13 $\pm$ 12.80	46.18 $\pm$ 14.63	48.11 $\pm$ 15.42	.072
Stress index	192.88 $\pm$ 112.37	183.84 $\pm$ 132.40	170.74 $\pm$ 135.08	.081

Note. Values are mean $\pm$ SD. Differences across tertiles were tested with the Kruskal–Wallis H test; where the omnibus test was significant, Dunn's pairwise comparisons with Bonferroni correction were applied. Within a row, tertiles sharing the same superscript letter did not differ significantly, whereas tertiles with different letters differed significantly ($p<.05$). Parameters showing no superscript letters (DBP, SDNN, stress index) had no significant overall difference across tertiles and were not subjected to pairwise comparison

Discussion

The principal finding of the present study was the strong association between skeletal muscle mass and device-estimated cardiac output. The full regression model, including all five body-composition predictors, explained a substantial proportion of the variance in cardiac output ($R^2=0.692$; adjusted $R^2=0.687$), and within this model skeletal muscle mass had the largest standardized coefficient. When fat mass, visceral fat, phase angle, and BMI were entered alongside it in the same regression, skeletal muscle mass showed the strongest independent association –

standardised $\beta=0.84$, more than twice the size of the next contributor, fat mass, which also remained independently significant ($\beta^*=0.41$). Stroke volume followed a similar pattern ($R^2=0.533$; std. $\beta=0.64$). Visceral fat and phase angle were not significant after adjustment, and BMI retained only a small independent association with stroke volume. The Cohen's f^2 of 2.25 substantially exceeds the conventional threshold for a very large effect. The magnitude of the observed effect indicates that the association is unlikely to be explained solely by sample size.

Several physiological mechanisms have been proposed that could, hypothetically, contribute to the observed relationship. However, the cross-sectional design of the present study does not allow any of them to be tested. A greater skeletal muscle mass may correspond to a larger peripheral vascular bed, which could hypothetically be accompanied by a higher stroke volume rather than a higher resting heart rate. This is consistent with earlier physiological work showing that fat-free mass is strongly related to resting stroke volume and blood volume (Hunt et al., 1998; Middlemiss et al., 2016). This mechanistic account remains speculative and is offered only as a hypothesis for future work. The present findings are descriptively consistent with this interpretation: muscle mass correlated negatively with resting heart rate ($\rho = -0.23$) and positively with stroke volume ($\rho = 0.66$). Heinonen (2025) made a similar argument at the review level, concluding that skeletal muscle is an underrated determinant of resting cardiac output in healthy young adults. The present data extend this observation to a population characterised by documented health limitations and restrictions on participation in standard physical education.

The present findings are broadly consistent with recent evidence. Dlamini et al. (2025), in a sample of 1,162 young African women, found that appendicular muscle mass predicted both systolic and diastolic blood pressure, regardless of body fat. Hostrup and Onslev (2022) reviewed how pharmacological stimulation of muscle mass through β_2 -adrenergic pathways produces measurable hemodynamic effects at rest, well before fitness markers change. Although the present participants were not pharmacologically manipulated, a broadly comparable pattern was observed in the present data: a larger skeletal muscle compartment was associated with higher cardiac output, statistically linked mainly to elevated stroke volume. The observed relationship is biologically plausible and is consistent with current understanding of cardiovascular adaptation (Chantler et al., 2008).

The behaviour of phase angle in the regression models deserves comment. On its own, it correlated reasonably with cardiac output ($\rho \approx 0.40$), and the literature treating phase angle as a global cardiovascular risk marker is solid (Akamatsu et al., 2022; de Borja et al., 2022, 2024). After skeletal muscle mass was entered into the regression model, the independent contribution of phase angle diminished substantially. This attenuation likely reflects the fact that phase angle indexes cell-membrane integrity and intracellular mass, and intracellular mass in young adults is composed largely of skeletal muscle. The two variables therefore share a considerable proportion of their explained variance. In older or clinical cohorts, these two indicators tend to diverge, as membrane integrity typically deteriorates more rapidly than skeletal muscle quantity, particularly in sarcopenia and chronic illness (de Borja et al., 2024). In the present cohort of 18- to 21-year-olds, however, these variables were too closely associated to serve as independent predictors. Recent studies in adolescents and young adults similarly report that phase angle is strongly determined by body composition (Ferreira et al., 2024; Jirků & Křížová, 2025). In the cardiac-index model, phase angle showed a small but significant negative association ($\beta = -0.16$), which should be interpreted with caution given the exploratory nature of the analysis.

A similar pattern was observed for visceral adipose tissue. Although VAT has been linked to adverse cardiometabolic outcomes in young adults (Miazgowski et al., 2021; Yuri et al., 2026), its contribution became negligible after adjustment for the other body-composition variables. This may partly reflect the distribution of visceral adiposity in the present cohort, in which most values were low and only a small subgroup showed elevated levels. Consequently, VAT explained little unique variance in the hemodynamic models.

The tertile analysis quantified the associations observed in the regression model. Students in the highest muscle-mass tertile (≥ 35.2 kg) had a mean cardiac output of 6.86 L/min, compared with 5.00 L/min in the lowest tertile (≤ 24.6 kg) – a 37% gap. An even larger difference was observed for stroke volume: 85.2 mL vs 56.8 mL, a 50% difference. These represent considerable differences among participants aged 18 to 21 years. The observed pattern suggests that differences in skeletal muscle mass within this cohort were associated with a substantial difference in central hemodynamic

parameters. HRV indices (SDNN, Baevsky stress index) moved in the same direction but stopped just short of significance ($p = .072$ and $.081$). Although none of the HRV comparisons reached the predefined significance threshold, the direction of the observed changes was consistent across indicators. Therefore, the findings cannot be considered conclusive but may warrant further investigation in larger cohorts.

Skeletal muscle mass may therefore be relevant when characterising the resting physiological profile of SMG students. Resistance training is generally recognised as the modality that adds muscle most efficiently. The 2023 AHA scientific statement (Paluch et al., 2024), the umbrella review by Bernárdez-Vázquez et al. (2022), and the systematic review by Morris et al. (2024) all converge on the same point: resistance training has been proposed as a valuable component of cardiovascular conditioning, particularly in low-fitness adults. Meta-analyses of randomized trials indicate that resistance training can favourably affect blood pressure and body composition (Cornelissen & Smart, 2013; de Oude et al., 2025; Hu et al., 2025), and resistance exercise has been shown to improve vascular function and central hemodynamics in adults with elevated blood pressure (Banks et al., 2023). Russian SMG curricula have historically emphasised moderate aerobic exercise (Podberezko, 2023). Our cross-sectional findings cannot demonstrate that resistance training improves hemodynamics; whether it does so in this population would need to be tested directly in prospective intervention studies. Cvijetic et al. (2023), in a study of young-to-middle-aged men, demonstrated positive associations between skeletal muscle indices and parasympathetic HRV markers, suggesting that the pathway from muscle mass to cardiovascular health may operate not only through mechanical coupling but also through improved autonomic balance. However, this, too, remains hypothetical.

The magnitude of the observed effect also warrants discussion. Standardised coefficients above 0.8 are uncommon in cross-sectional cardiovascular research. However, because directly comparable studies using the same device-estimated outcomes in this population are lacking, we do not claim that the present magnitude exceeds that of previous work. The relatively large coefficient may be partly explained by methodological factors: the present sample is unusually homogeneous (a single university, a narrow age range, similar daily exposure), and the integrated measurement protocol minimises cross-device noise that typically attenuates such associations (Bocharin et al., 2022). It may also partly reflect the fact that both predictors and outcomes were obtained from the same device. Biological factors may also contribute; at this age, the association between skeletal muscle mass and cardiac output may be tighter than in older cohorts, before other determinants (atherosclerosis, autonomic decline, medication use) begin to contribute additional variance. Distinguishing between these explanations requires longitudinal investigation, which cross-sectional data cannot provide.

One important consideration concerns body size. Because both skeletal muscle mass and absolute cardiac output scale with overall body dimensions, part of the observed association could in principle reflect differences in body size rather than a specific effect of muscle tissue. To address this, we repeated the analysis using device-estimated cardiac index as the outcome. Skeletal muscle mass had the largest standardized coefficient for cardiac index ($\beta^* = 0.42$; $p < 0.001$), whereas BMI – which had correlated strongly with unindexed cardiac output – was no longer significant, and its bivariate correlation with cardiac index was negligible ($\rho = 0.06$). These results indicate that the association between skeletal muscle mass and cardiac output persisted after cardiac output was indexed to body surface area; because sex was unavailable as a covariate and the device uses proprietary algorithms, this should be interpreted as an association that survives body-size indexing rather than as proof of a body-size-independent physiological effect. The lower overall variance explained for cardiac index compared with absolute cardiac output is expected, since indexing removes the substantial body-size component shared by all predictors.

Implications

The present findings may have practical relevance for the physiological monitoring of students assigned to special medical groups. Although aerobic exercise traditionally forms the basis of many SMG programmes, our results suggest that skeletal muscle mass may be considered as one component of a multidimensional approach to physiological monitoring in this population. The potential effects of resistance-oriented exercise on resting hemodynamics were not tested in the

present study. They should be examined in prospective intervention studies rather than inferred from these cross-sectional data. Because SMG students have heterogeneous medical restrictions, any exercise guidance arising from future work should be individualized and medically appropriate for the specific student, rather than framed as a general modification to SMG physical education programmes. The findings also highlight several limitations of BMI as a stand-alone screening tool. In the present sample, students with similar BMI values often exhibited markedly different device-estimated cardiac output and stroke volume. This observation suggests that body composition assessment may provide information not obtainable from anthropometric indices alone. Where appropriate equipment is available, segmental bioimpedance analysis could be incorporated into routine monitoring procedures to support a more individualised approach to physical education planning.

Research Contribution

Several aspects of the present study contribute to the existing literature. Research involving students assigned to special medical groups remains limited, particularly when objective physiological measurements are used. Most previous studies in this population have relied on questionnaires, fitness testing, or isolated functional indicators (Spataeva et al., 2024; Terekhina et al., 2023). The principal contribution of the present study is the integration of body-composition assessment, device-estimated hemodynamic parameters, and heart rate variability into a single measurement protocol in a sample of patients with a special medical condition, together with a comparison of the relative contributions of several body-composition variables under identical testing conditions. To our knowledge, comparable data have not previously been reported for Russian SMG students. Rather than providing a definitive reference value, and given that a single proprietary device generated the outcomes, that sex was unavailable, and that the sample came from one university, the study is best regarded as offering preliminary, population-specific evidence to inform independent validation and prospective research.

Limitations

Several limitations should be considered. First, participants' sex was not coded in the de-identified dataset, preventing sex-specific analyses. Because skeletal muscle mass and cardiac output are both influenced by sexual dimorphism, the associations reflect the pooled sample and require confirmation in sex-stratified analyses. Second, and most importantly, all predictors and outcomes were obtained from the same integrated device, whose cardiac output algorithm incorporates undisclosed anthropometric and blood-pressure inputs; part of the muscle-cardiac output association may therefore reflect shared device inputs (mathematical coupling) rather than a purely physiological relationship. The device-derived cardiac output and stroke volume were not validated against reference methods such as echocardiography or impedance cardiography (Schierbauer et al., 2022), so the large standardized coefficient should not be interpreted as a physiologically validated effect. Third, the visceral adipose tissue variable was highly skewed, which may have influenced its multivariable estimates; the mild heteroscedasticity in the cardiac output model was addressed with HC3 robust standard errors, and the findings were unchanged. Fourth, habitual physical activity was not assessed with a validated instrument, so training status could not be separated from muscle mass. In addition, the HRV indices were derived from an ultra-short 60–90-second recording; frequency-domain indices in particular are conventionally based on recordings of at least five minutes and were not validated for this device at this length (Nussinovitch et al., 2011; Munoz et al., 2015; Shaffer & Ginsberg, 2017), and are therefore reported as exploratory. Fifth, the cross-sectional design precludes causal inference, and reverse causation cannot be excluded. Finally, the single-centre sample of special medical group students limits generalisability to other populations.

Suggestions

Future studies should attempt to address several limitations of the present work. First and most importantly, the cardiac output and stroke volume estimates produced by the MS FIT device should be validated against reference-standard cardiovascular measurements, such as echocardiography or impedance cardiography, before device-estimated values are used to draw physiological conclusions. Subsequently, replication in larger multi-centre cohorts with sex-stratified

analyses and validated assessment of physical activity would improve the generalisability of the findings. Longitudinal designs and resistance-training interventions are particularly important because the cross-sectional nature of the present study does not allow conclusions regarding causality, and are needed to determine whether changes in resting hemodynamics actually accompany changes in skeletal muscle mass. Future studies should also define primary outcomes in advance, account for potential confounders such as sex, body size, habitual physical activity, and medical diagnoses, and distinguish genuine biological associations from associations that may partly reflect device algorithms. Additional work is also required to determine whether the relationship between skeletal muscle mass and cardiovascular parameters differs across specific categories of special medical group students. Finally, further investigation of autonomic regulation variables may be warranted, as several HRV indicators demonstrated potentially meaningful trends that did not reach statistical significance in the current sample.

CONCLUSION

The present study examined associations between body composition and resting hemodynamic parameters in 277 medical students assigned to the special medical group. Among the body-composition variables examined, skeletal muscle mass showed the strongest independent association with device-estimated cardiac output and stroke volume. At the same time, fat mass also contributed independently to cardiac output. Visceral adiposity, phase angle, and BMI showed little or no independent contribution to cardiac output after adjustment, and the R^2 values refer to the full five-predictor models rather than to skeletal muscle mass alone. Students in the highest muscle-mass tertile demonstrated markedly higher cardiac output and stroke volume, together with lower resting heart rate, compared with those in the lowest tertile. These findings suggest that skeletal muscle mass is closely associated with the resting hemodynamic profile in this population. From a practical perspective, the results support greater attention to body composition assessment in the physiological monitoring of students assigned to the special medical group. Segmental bioelectrical impedance analysis may provide additional information beyond BMI alone when evaluating the resting hemodynamic profile. Whether muscle-preserving or muscle-building exercise strategies improve resting hemodynamics in this population remains to be determined and warrants future longitudinal and interventional research.

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AUTHOR CONTRIBUTION STATEMENT

IB and MG conceived and designed the study. IB, YK, ERy, and AZ carried out data collection at the study site. IB performed the statistical analysis and prepared the figures. IB and MG wrote the first draft of the manuscript. ER, NB, SM, and AE contributed to the interpretation of the results and critically revised the manuscript for important intellectual content. All authors read and approved the final version of the manuscript and agree to be accountable for all aspects of the work.

AI DISCLOSURE STATEMENT

Generative artificial intelligence tools were used exclusively for language editing and improvement of English grammar and style. No AI tools were used for data collection, statistical analysis, interpretation of results, or generation of scientific conclusions. All scientific content remains the responsibility of the authors. The authors reviewed, revised, and approved the final manuscript and take full responsibility for its contents. No personal or identifiable data were processed using AI tools.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests. The authors have no financial, commercial, consultancy, advisory, or personal relationships with MedicalSoft LLC, the manufacturer of the MS FIT diagnostic complex used in this study. No funding, compensation, or other support was received from the manufacturer in connection with this work.

ETHICAL APPROVAL

This study was based on a retrospective secondary analysis of de-identified routine health-monitoring data and was approved by the Local Ethics Committee of Privolzhsky Research Medical University (Nizhny Novgorod, Russia; Protocol No. 7, dated 3 July 2026). The study was conducted in accordance with the Declaration of Helsinki. Because the analysis used only pre-existing, de-identified secondary records and no personally identifiable information was accessed, the Committee waived the requirement for individual informed consent under the applicable institutional and national regulations. The study did not involve animals.

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