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Structure-specific variation in pediatric echocardiographic Z scores across the nutritional spectrum: four body-surface-area nomograms and a BMI-adjusted GAMLSS model

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Structure-specific variation in pediatric echocardiographic Z scores across the nutritional spectrum: four body-surface-area nomograms and a BMI-adjusted GAMLSS model

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Abstract

Purpose

Z score nomograms, the standard for interpreting pediatric echocardiographic dimensions, derive mostly from eutrophic populations. We assessed whether cardiac structure Z scores vary with body mass index Z score (BMIz) across four body-surface-area (BSA)-based nomograms, and whether a BMI-adjusted reference mitigates this bias.

Methods

Retrospective study of 1883 children aged 5–18 years with structurally normal echocardiograms (2016–2021). Eleven cardiac structure diameters were analyzed and Z scores computed with four nomograms (Pediatric Heart Network, Detroit, Toronto, Italy) and, for ten structures, with a fifth based on generalized additive models for location, scale and shape (GAMLSS) adjusted for body size, BMI and age. Associations with BMIz used linear regression in three nested models with false discovery rate correction.

Results

Overall, 25.8% were overweight and 15.8% had obesity. Absolute diameters increased with BMIz in every structure ($p<0.001$), yet Z scores of most left-sided structures were inversely associated with it, most strongly for the four aortic levels (β −0.325 to −0.182, significant in all 45 nomogram-by-model comparisons) and for left ventricular end-diastolic diameter. Ventricular walls showed no consistent pattern; right-sided annuli varied non-monotonically. Under GAMLSS these associations were abolished in the most affected structures, persisted in the sinotubular junction and walls, and reversed in sign in the pulmonary annulus.

Conclusion

Z scores of most left-sided structures were lower in obesity than their absolute dimensions suggest, so interpretation must account for nutritional status and the reference. Persisting bias follows model construction: the three structures whose GAMLSS equations omit BMI are exactly those still biased.

Keywords: echocardiography; Z score nomograms; obesity; body mass index; nutritional status; cardiac structure.

Abbreviations

AAo: ascending aorta

AoV: aortic valve annulus

AR: aortic root (sinuses of Valsalva)

BMI: body mass index

BMIz: body mass index Z score

BSA: body surface area

CS: cardiac structure

GAMLSS: generalized additive model for location, scale and shape

LA: left atrium

LVEDD: left ventricular end-diastolic diameter

MV: mitral valve annulus

PHN: Pediatric Heart Network

PV: pulmonary valve annulus

PW: posterior wall

STJ: sinotubular junction

SW: septal wall

TV: tricuspid valve annulus

What is Known:

Z score nomograms are the standard for interpreting cardiac structure size in children, yet are derived from predominantly eutrophic populations.

BSA-based Z scores carry a systematic BMI-related bias, shown mainly for the aortic root.

What is New:

The bias is structure-specific: Z scores of most left-sided structures are consistently underestimated, whereas ventricular walls and right-sided annuli behave heterogeneously.

Persisting bias tracks model construction: the structures whose BMI-adjusted equations omit BMI are exactly those in which a negative association remained.

Introduction

Assessment of cardiac structure (CS) size is central to diagnosing and managing heart disease throughout life [1-4]. During growth, somatic development drives CS growth in differing proportions, cardiac allometry [4], mainly through the rise in cardiac output required by metabolic demand. Interpreting CS dimensions therefore requires references appropriate for age and body size, most often Z score nomograms, expressing how many standard deviations a measurement lies from the reference mean at the same body surface area (BSA). Most derive from healthy, generally eutrophic populations [2-5]; guidelines acknowledge their limitations in obesity [1,3], yet they remain widely used.

Obesity alters cardiac structure and function mainly through increased blood volume and cardiac output [6,7], modifying CS size and hence Z scores even without hypertension. How much obesity affects each structure remains poorly characterized, and the behavior of nomograms across nutritional categories has received little attention. Given the global rise in childhood obesity, accelerated since the COVID-19 pandemic [8-10], the question is of growing relevance.

We assessed whether echocardiographic Z scores vary with nutritional status, whether associations differ by structure and nomogram, and whether a reference incorporating body mass index (BMI) mitigates them.

Materials and methods

Design and participants

Retrospective, single-center study at the Non-Invasive Pediatric Cardiology Laboratory, Red UC Christus, Santiago, Chile. Children and adolescents aged 5–18 years with an echocardiogram between 2016 and 2021 showing no structural lesion were eligible; a small patent foramen ovale or mild or lesser valvular regurgitation was not considered a structural lesion for this purpose. We excluded participants with surgically corrected congenital heart disease, known or suspected genetic syndromes or connective tissue disease, missing or unreliable weight or height, or no measurable CS; when more than one study was available, only the last was used.

Anthropometry and nutritional status

Weight, height and blood pressure were measured on the examination day by trained staff using SECA® 713 scales and a CRITIKON Dinamap 8100® monitor, following international standards [11]. Nutritional status was classified

from the BMI Z score (BMIz) using WHO 2007 references [12]: risk of undernourishment/thinness if BMIz < −1; eutrophic −1 to +1; overweight +1 to +2; obesity ≥ +2. BSA used the Haycock formula [13].

Echocardiography

Images were acquired by experienced pediatric cardiologists (>300 studies annually) on Vivid E9 and E95 systems (GE Vingmed Ultrasound, Milwaukee, WI, USA), following American Society of Echocardiography guidelines [1,14]. M-mode provided left ventricular end-diastolic diameter (LVEDD) and septal (SW) and posterior (PW) wall thickness; two-dimensional imaging provided the diameters of the left atrium (LA), the mitral (MV), aortic (AoV), tricuspid (TV) and pulmonary (PV) valve annuli, and the aortic root at the sinuses of Valsalva (AR), sinotubular junction (STJ) and ascending aorta (AAo). Structure measurements were obtained from the reports generated by the same operators who acquired the images.

Z score nomograms

Four widely used nomograms were applied: Pediatric Heart Network (PHN) [15], Detroit [16], Toronto, and the Italian nomogram of Cantinotti et al. [17]. Toronto values come from an unpublished data set of The Hospital for Sick Children. Table 1 summarizes the structures analyzed, inclusion and exclusion criteria, nutritional status of the reference participants and methodological robustness of each [18,19]. A methodologically distinct fifth reference was added, the Lauzon-Schnittka (2025) nomogram, derived from more than 20,000 children using generalized additive models for location, scale and shape (GAMLSS) [20], which predicts the median of each dimension from height, weight, age and BMIz, with terms selected per structure and sex. BMIz enters most structures but not the septal wall, posterior wall or sinotubular junction, the last of which also omits age (Online Resource 1). Z scores used the authors' official R calculator; no equation exists for the LA, compared only across BSA-based nomograms. Each CS was classified as normal (Z −2 to +2), dilated (>+2) or small/hypoplastic (<−2).

Statistical analysis

Continuous variables are presented as mean ± standard deviation and compared across the four nutritional groups by one-way ANOVA, categorical variables as n (%) by chi-square test, with global tests only. For categorized Z scores, the proportions in each size category were compared between every pair of nutritional groups within each structure-

by-nomogram combination by chi-square test; significant pairs are marked by brackets in Fig. 1 and listed in Online Resource 2.

Linear regression examined the association between each CS Z score and BMIz under each nomogram, reporting β with its 95% confidence interval. β is standardized: the change in Z score, in standard deviations, per one standard deviation increase in BMIz. Three nested models were fitted: M1 unadjusted; M2 adjusted for age and sex; M3 for age, sex and height. The p values of the regressions and of the pairwise chi-square comparisons were adjusted by the Benjamini-Hochberg false discovery rate procedure within each family, and for the regressions within each model. Complete results are given in Online Resource 3. Significance was set at $p<0.05$; analyses used R 4.5.3.

The authors used Claude (Anthropic) as an AI-assisted tool for language editing and formatting improvements. All scientific content, data analysis, interpretation, and final approval were performed by the authors, who assume full responsibility for the manuscript.

Results

Of 6,019 children assessed, 2,694 (44.8%) had a study without structural lesions; 811 were excluded, leaving 1,883 participants (55.1% male, mean age 10.3 ± 3.2 years) with 16,114 measurements. Only 51.3% (n=966) were eutrophic: 7.2% (n=135) were undernourished, 25.8% (n=485) overweight and 15.8% (n=297) obese. In obesity 70.4% were male and systolic blood pressure was higher (110.9 ± 12.1 versus 103.7 ± 11.1 mmHg; both $p<0.001$; Table 2). Absolute diameters of all eleven structures were larger with excess weight ($p<0.001$; Table 3); the differences below therefore concern Z scores, not dimensions.

Aortic valve and root

Z scores at all four aortic levels fell consistently across nutritional groups under the four BSA-based nomograms ($p<0.001$; Table 3), with significant eutrophic-versus-obesity differences in all four nomograms for the valve annulus and sinuses of Valsalva, in Detroit and Italy for the STJ and in PHN and Italy for the AAo (Fig. 1). Regression showed the study's strongest negative associations, β −0.325 to −0.182 overall and −0.315 to −0.216 for the valve annulus, significant in all 45 comparisons (Fig. 2). Under GAMLSS the association disappeared at three of the four levels (β −0.029 to +0.036), the STJ alone retaining a smaller negative association (β −0.081 to −0.057).

Left ventricular walls

Unlike other left-sided structures, Z scores of both walls did not fall consistently with BMIz and their sign depended on the nomogram: a significant negative association appeared only under Detroit, for SW (β −0.074 to −0.054) and PW (β −0.083 to −0.066), while PHN and Toronto gave small, even weakly positive coefficients. Categorically, the only significant differences were eutrophic versus obesity for SW ($p=0.008$) and PW ($p=0.024$), under Detroit (Fig. 1). Under GAMLSS both walls retained a significant negative association (SW β −0.074 to −0.070; PW β −0.061 to −0.054), almost identical to that under Detroit.

Mitral and right-sided annuli

The MV showed significant negative associations with BMIz under PHN, Toronto and Italy (β −0.144 to −0.084) but not Detroit, no categorical difference under any nomogram, and no significant association under GAMLSS (β +0.044 to +0.062).

Right-sided annuli behaved differently, their most relevant feature being non-monotonicity: under all four BSA-based nomograms mean Z scores peaked in the eutrophic or overweight group and fell in obesity (Table 3). In regression the TV reached significance under PHN and Italy in all three models (β −0.138 to −0.069) and under Toronto in two; the PV showed only sporadic associations. These small coefficients reflect that shape and match the categorical picture: no right-sided combination showed a significant pairwise difference (Fig. 1). Under GAMLSS the PV became monotonically increasing, with mean Z scores of −0.55, −0.32, −0.10 and −0.09, and yielded the largest and only positive coefficient of the study (β +0.128 to +0.155).

Cardiac chambers

LVEDD Z scores fell significantly with BMIz in the three BSA-based nomograms providing this structure, in all models (β −0.219 to −0.154), with significant eutrophic-versus-obesity categorical differences under all three (Fig. 1); GAMLSS abolished it entirely. The LA behaved differently: its Z scores followed no consistent trend, size classification differed only under Detroit (eutrophic versus obesity, $p<0.001$), and its only significant regression association was positive, under Toronto (β +0.083 to +0.098).

Overall, the four BSA-based nomograms produced a consistent negative association between BMIz and the Z scores of the four aortic levels, MV and LVEDD, significant in 63 of 66 comparisons, the exceptions being MV under Detroit. Categorical classification followed the same ordering: significant pairwise differences appeared in 18 combinations, all left-sided and almost all involving the obesity group (Fig. 1). GAMLSS suppressed it where most pronounced: AoV, AR, AAo, LVEDD and MV all fell to an absolute β of 0.062 or less, nonsignificant. Four retained it, with much smaller magnitude: STJ, SW, PW and PV, the last the only positive one (Fig. 3).

Discussion

In this pediatric cohort spanning the whole nutritional spectrum, echocardiographic Z scores varied with nutritional status non-uniformly across structures: both magnitude and direction depended on the structure and the nomogram, and a BMI-adjusted model corrected this bias just as heterogeneously. We propose that this pattern, not previously described, reflects two opposing effects on each structure: normalization by body surface area and obesity-related cardiac remodeling and that their balance explains the ordering of our results.

The first effect is likely methodological. Cardiac output best predicts CS size, and BSA correlates best with it among anthropometric indices [4], but BSA does not distinguish fat from lean mass. At equal BSA, a child with obesity has less lean mass and lower cardiac output demand than the eutrophic reference, since adipose tissue has a lower metabolic rate per unit mass [21]. The nomogram then predicts a larger dimension than metabolic demand justifies, and the Z score declines. This could explain why absolute dimensions rise with excess weight while Z scores fall [22-24].

The second effect is probably biological. Obesity enlarges cardiac structures through volume overload from increased blood volume and preload, and through pressor and neurohormonal activation (sympathetic, renin-angiotensin-aldosterone, hyperinsulinemic) [6,7]. Systolic blood pressure was higher in obesity in our cohort, consistent with a pressor contribution, though we did not measure neurohormonal activation directly. Unlike the first effect, this one is uneven, concentrating in the myocardium and distensible chambers, and minimal in fibroelastic structures [25]. The observed Z score likely reflects this balance. The aortic levels and mitral annulus are fibroelastic, with little remodeling, show the negative association at its strongest; LVEDD sits in between, consistent with a cavity that dilates under volume overload.

At the opposite end sit the ventricular walls, the direct pressor target, where remodeling is greatest. Here the two effects may cancel, so the net sign could depend on small differences between references: a steeper BSA-scaling slope for wall thickness yields a negative net, as with Detroit, while a flatter slope, as with PHN and Toronto, yields a null or slightly positive one. This disagreement may reflect that balance rather than noise, though without measured body composition it remains an interpretation. Wall thickness holds while the cavity falls relative to expectation, a concentric pattern consistent with pressor load. The left atrium may show the same principle in reverse: sensitive to chronic volume overload [26], it was the only left-sided structure with a positive association, though only under one nomogram.

The right side warrants separate consideration: its Z score peaked in an intermediate group before falling in obesity, rather than varying monotonically. Linear regression captures such a shape poorly, since its coefficient tends toward zero when the relationship reverses. Our sporadic coefficients likely reflect that shape rather than a true absence of association, and greater measurement variability in these annuli attenuates them further [27]. This variability may also reflect imaging difficulty: the pulmonary and tricuspid annuli sit within the right ventricle's retrosternal, crescentic geometry, poorly captured by standard 2D planes and more variable between observers than simpler, left-sided structures [28]. The shape fits the two-effect balance if the biological contribution plateaus rather than rising steadily. Why right ventricular loading behaves this way is unclear: pulmonary load determinants such as obstructive sleep apnea [29] vary widely in obesity, but our design cannot test this.

Different nomograms correlate well overall yet differ significantly, particularly those with weaker methodology [30], and these differences widen when stratified by nutritional status. Adult evidence on BSA indexation in obesity does not transfer to children [26]. In pediatrics, Dallaire et al. first showed an association between BMI-for-age and BSA-based Z scores in the proximal aorta [22]; Plante et al. then extended this to a multicenter cohort [23]; and Lauzon-Schnittka et al. developed GAMLSS equations incorporating body size, BMI and age [20]. Our study evaluates existing references rather than building new ones.

Scope and limits of a BMI-adjusted reference model

The GAMLSS nomogram substantially attenuated these associations, with correction essentially complete in the most biased structures. This attenuation is expected wherever BMIz enters the equation and it confirms the reference works as intended, rather than revealing anything new. The informative contrast is with structures whose equations omit

BMIz. The supplementary material shows BMIz included in seven of ten structures with a GAMLSS equation, omitted in three: the sinotubular junction, septal wall and posterior wall. These three are exactly the structures in which a negative association persisted; none of the seven that model BMIz retained one.

For the sinotubular junction, sample size is a plausible explanation: it is the least-measured structure in the derivation cohort, fewer than 500 studies per sex, versus about 10,000 for the aortic annulus, so the authors avoided non-linear effects in sparsely measured variables. Its model reduces to height and weight, the same class as a BSA nomogram, and the authors' own cross-validation shows it does not outperform that model here. That it was the only uncorrected aortic level reflects a modeling choice, not anatomy.

In the ventricular walls, the omission likely reflects model selection, not sample size: wall measurements are among the largest in the cohort, but adiposity enters the model only through the weight exponent. A model cannot correct a bias it does not include, which is why the residual pattern nearly matches Detroit's, for the walls, both are body-size models. Wall thickness is also most sensitive to the measuring center: its mean Z score varied between institutions by more than one unit, among the widest ranges we studied, so a calibration difference alone could explain what persists.

The pulmonary annulus is the exception: its model includes BMIz, but only linearly, with height as the sole body-size variable. Its Z score still varied with nutritional status, almost entirely between undernutrition and overweight, the positive association arises not from an excess in obesity but from a deficit in the leanest, where the model predicts a larger annulus than measured. A linear term imposes a constant-slope correction, estimated on about half the observations available for the sinuses of Valsalva. This finding most clearly needs independent confirmation.

One general point deserves note: a BMI-incorporating model cannot distinguish the normalization artifact from true remodeling, it removes both. In the aorta, where remodeling is slight, almost everything removed is artifact, so correction is desirable. In wall thickness, where remodeling is the signal of interest, incorporating BMI could mask it. This is not a risk today, but one for future model versions.

Clinical implications and future directions

Recent recommendations discourage extrapolating Z scores beyond the population for which they were built [3,18]; applied literally, this would mean abandoning the tool for half our sample. But switching between nomograms for different structures within one report is neither practical nor validated. Our results establish that the choice of reference is not interchangeable outside the eutrophic range. For aortic dimensions, the mitral annulus and LVEDD, BSA-based

nomograms underestimate the Z score enough to shift size classification in excess weight. For wall thickness, GAMLSS offers no advantage, since it omits BMI for that structure; normalization by lean mass or height has been proposed instead [31,32]. For right-sided annuli, no reference is validated at the upper end of the spectrum. Until then, we recommend reporting nutritional status and absolute dimension alongside the Z score whenever weight status departs from eutrophic. No consensus yet exists on the best normalization method at nutritional extremes [33].

Strengths and limitations

Strengths include the sample size across the whole nutritional spectrum and a design evaluating eleven structures with four nomograms plus a fifth, BMI-adjusted reference, allowing direct comparisons within one population. Limitations include the retrospective, single-center design. We did not measure body composition, so the separation between the two effects is an interpretation, not a demonstration. We did not assess reproducibility, despite evidence of greater measurement variability in obesity [27]. The Toronto nomogram is unpublished, so several methodological details could not be characterized, and comparisons remain exploratory. What persists under GAMLSS could reflect differences between its Canadian derivation cohort and ours rather than model construction. M3 adjusts for age, sex and height, internal to the GAMLSS equations but not the BSA nomograms, so estimates are not strictly comparable across families; M1, adjusting for nothing, identifies the same four structures. We did not assess pubertal stage or physical activity, both relevant covariates of cardiac size in pediatric populations [34,35], so residual confounding cannot be excluded.

In conclusion, echocardiographic Z scores varied with nutritional status in a structure-specific manner: negative, consistent and largest in the four aortic levels, the mitral annulus and LVEDD; null or nomogram-dependent in the ventricular walls and left atrium; and non-monotonic in right-sided annuli, an ordering that reflects the balance of two opposing effects, the bias of BSA normalization and true obesity-related remodeling. A BMI-adjusted GAMLSS model substantially reduced the first bias, though associations persisted in the ventricular walls and sinotubular junction, and reversed in sign in the pulmonary annulus. These findings, from a single-center retrospective cohort without measured body composition, support considering BMI-incorporating nomograms for aortic and chamber dimensions, and caution for wall thickness, pending external validation.

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Statements and Declarations

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Competing interests

The authors have no relevant financial or non-financial interests to disclose.

Author contributions

All authors contributed to the study conception and design. Material preparation and data collection were performed by Rodolfo Amstein, Valentina Metcalfe and Guillermo Larios. Analysis was performed by Álvaro Passi-Solar, Rodolfo Amstein, Claudia Trincado and Guillermo Larios. The first draft of the manuscript was written by Rodolfo Amstein and Guillermo Larios, and all authors commented on previous versions. All authors read and approved the final manuscript.

Ethics approval

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the Pontificia Universidad Católica de Chile (No. 220801012).

Consent to participate

Informed consent was waived by the institutional ethics committee owing to the retrospective nature of the study and the use of anonymized data.

Consent to publish

Not applicable; the manuscript contains no data from identifiable individual participants.

Data availability

The data sets generated and analyzed during this study are not publicly available because they contain potentially identifying clinical information from pediatric participants, but anonymised data and the analysis code are available from the corresponding author on reasonable request and subject to institutional ethics approval.

Tables

Table 1 Characteristics of the Z score nomograms used in this study: inclusion and exclusion criteria, nutritional status of the reference participants and methodological robustness according to the criteria of Cantinotti et al. [18]. The variables on which the median of each dimension is predicted are body surface area for the four BSA-based nomograms and, for the GAMLSS nomogram, a structure-specific set detailed in Online Resource 1. BSA, body surface area; BMIz, body mass index Z score.

Characteristic	**PHN**	**Detroit**	**Toronto**	**Italy**	Lauzon-Schnittka
Origin	Multicenter, USA	Detroit, USA	Toronto, Canada	Massa, Italy	Multicenter, Canada
Author	Lopez	Pettersen	Mertens L (unpublished)	Cantinotti	Lauzon-Schnittka
Year	2017	2008	NR	2017	2025
Age range	0–18 years	0–18 years	0–18 years	0–17 years	0-18 years
BSA range	NR	0.1-2.0	NR	0.12-2.12	NR
Inclusion and exclusion	Clear and relevant	Clear and relevant	NR	Clear and relevant	Clear and relevant
Nutritional status	Excludes obesity (BMI>95th centile)	Excludes obesity	NR	Excludes obesity (BMI>95th centile)	Full BMI range
No. of participants	3566	782	350	1151	>20,000
R^2	>0.99	NR	NR	0.90-0.94	NR
Heteroscedasticity testing	Yes	NR	NR	Yes	Yes (GAMLSS)
Cardiac structures included in this study	LVEDD, SW, PW, LA, AoV, AR, STJ, AAo, MV, TV, PV	LVEDD, SW, PW, LA, AoV, AR, STJ, MV, TV, PV	LVEDD, SW, PW, LA, AoV, AR, STJ, AAo, MV, TV, PV	AoV, AR, STJ, AAo, MV, TV, PV	LVEDD, SW, PW, AoV, AR, STJ, AAo, MV, TV, PV

Table 2 Demographic, anthropometric and blood pressure characteristics of participants by nutritional status. Data are mean ± standard deviation for continuous variables and n (%) for categorical variables. The p value for sex is from a chi-square test; all others are from one-way ANOVA across the four nutritional groups. All p values are global tests across the four groups; no pairwise comparisons were performed. Significant differences ($p<0.05$) are in bold. BMIz, body mass index Z score; BSA, body surface area; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Characteristic	Total n=1883	Undernourished n=135 (7.2%)	Eutrophic n=966 (51.3%)	Overweight n=485 (25.8%)	Obesity n=297 (15.8%)	p value
Age, years	10.29 ± 3.23	10.52 ± 3.59	10.22 ± 3.35	10.54 ± 2.99	9.98 ± 3.01	0.076
Male, n (%)	1038 (55.1%)	67 (49.6%)	497 (51.4%)	265 (54.6%)	209 (70.4%)	**<0.001**
Weight, kg	40.11 ± 17.03	29.43 ± 11.33	35.36 ± 13.64	44.68 ± 15.68	52.91 ± 21.28	**<0.001**
Height, cm	140.59 ± 18.94	138.06 ± 20.30	139.37 ± 19.48	142.93 ± 17.82	141.91 ± 17.85	**0.002**
BSA, m^2	1.24 ± 0.34	1.05 ± 0.28	1.16 ± 0.30	1.33 ± 0.32	1.45 ± 0.38	**<0.001**
BMI, kg/m^2	19.34 ± 4.10	14.77 ± 1.57	17.38 ± 2.17	21.01 ± 2.66	25.06 ± 4.43	**<0.001**
BMIz	0.74 ± 1.28	-1.57 ± 0.68	0.09 ± 0.54	1.48 ± 0.30	2.71 ± 0.69	**<0.001**
SBP, mmHg	105.67 ± 11.77	103.95 ± 12.37	103.74 ± 11.13	106.84 ± 11.57	110.86 ± 12.05	**<0.001**
DBP, mmHg	58.62 ± 7.83	59.24 ± 7.13	58.14 ± 7.85	58.72 ± 7.61	59.71 ± 8.31	**0.019**

Table 3 Distribution of cardiac structure diameters and their Z scores by nutritional status. Data are mean ± standard deviation. For each structure the first row reports the absolute diameter and subsequent rows the Z score under each nomogram. The p value is from one-way ANOVA across the four nutritional groups, adjusted by the Benjamini-Hochberg false discovery rate procedure; significant differences ($p<0.05$) are in bold. The n under each mean is the number of participants contributing to that group. PHN, Pediatric Heart Network; SW, septal wall; PW, posterior wall; LVEDD, left ventricular end-diastolic diameter; LA, left atrium; MV, mitral valve annulus; AoV, aortic valve annulus; AR, aortic root; STJ, sinotubular junction; AAo, ascending aorta; TV, tricuspid valve annulus; PV, pulmonary valve annulus.

Cardiac structure	Nomogram	Undernourished	Eutrophic	Overweight	Obesity	p value
Aortic valve annulus (AoV), mm (n=1750)		16.15 ± 2.33 n=129	16.55 ± 2.31 n=901	17.24 ± 2.34 n=441	17.42 ± 2.49 n=279	**<0.001**
	PHN	0.81 ± 1.01	0.52 ± 0.89	0.20 ± 0.91	-0.06 ± 0.86	**<0.001**
	Detroit	0.34 ± 0.89	0.19 ± 0.81	0.02 ± 0.86	-0.36 ± 1.08	**<0.001**
	Toronto	0.34 ± 1.15	0.02 ± 1.03	-0.36 ± 1.09	-0.68 ± 1.05	**<0.001**
	Italy	0.16 ± 1.03	-0.14 ± 0.92	-0.50 ± 0.98	-0.79 ± 0.94	**<0.001**
	Lauzon-Schnittka	-0.12 ± 1.01	-0.22 ± 0.89	-0.27 ± 0.94	-0.31 ± 0.91	0.247
Aortic root, sinuses of Valsalva (AR), mm (n=1728)		21.91 ± 3.11 n=128	22.41 ± 3.21 n=892	23.34 ± 3.25 n=431	23.47 ± 3.21 n=277	**<0.001**
	PHN	0.58 ± 0.92	0.23 ± 0.98	-0.11 ± 1.02	-0.42 ± 0.95	**<0.001**
	Detroit	0.30 ± 0.74	0.12 ± 0.77	-0.04 ± 0.82	-0.43 ± 1.06	**<0.001**
	Toronto	0.18 ± 0.74	-0.09 ± 0.79	-0.36 ± 0.84	-0.61 ± 0.80	**<0.001**
	Italy	0.23 ± 0.84	-0.08 ± 0.91	-0.38 ± 0.96	-0.66 ± 0.92	**<0.001**
	Lauzon-Schnittka	0.17 ± 0.82	0.14 ± 0.89	0.18 ± 0.95	0.20 ± 0.89	0.832
Sinotubular junction (STJ), mm (n=1674)		17.72 ± 2.71 n=126	18.27 ± 2.82 n=866	19.14 ± 2.88 n=418	19.11 ± 2.79 n=264	**<0.001**
	PHN	0.36 ± 0.95	0.15 ± 1.04	-0.11 ± 1.00	-0.43 ± 0.97	**<0.001**
	Detroit	0.31 ± 0.70	0.24 ± 0.75	0.16 ± 0.74	-0.18 ± 0.90	**<0.001**
	Toronto	-0.10 ± 0.72	-0.24 ± 0.79	-0.43 ± 0.77	-0.68 ± 0.77	**<0.001**
	Italy	0.61 ± 0.90	0.40 ± 0.99	0.12 ± 0.97	-0.20 ± 0.98	**<0.001**
	Lauzon-Schnittka	0.22 ± 0.91	0.19 ± 0.96	0.11 ± 0.94	-0.07 ± 0.93	**0.002**
Ascending aorta (AAo), mm (n=909)		18.83 ± 2.86 n=67	19.41 ± 2.94 n=470	20.19 ± 3.01 n=237	20.06 ± 3.00 n=135	**<0.001**
	PHN	0.15 ± 0.87	0.12 ± 0.98	-0.12 ± 1.00	-0.53 ± 0.91	**<0.001**
	Toronto	0.48 ± 0.71	0.42 ± 0.80	0.19 ± 0.83	-0.16 ± 0.80	**<0.001**
	Italy	-0.54 ± 0.91	-0.58 ± 1.02	-0.81 ± 1.06	-1.24 ± 1.02	**<0.001**
	Lauzon-Schnittka	-0.64 ± 0.90	-0.46 ± 1.00	-0.44 ± 1.05	-0.58 ± 0.99	0.386

Cardiac structure	Nomogram	Undernourished	Eutrophic	Overweight	Obesity	p value
Mitral valve annulus (MV), mm (n=1150)		22.35 ± 3.15 n=91	23.33 ± 3.27 n=596	24.83 ± 3.45 n=282	24.88 ± 3.71 n=181	**<0.001**
	PHN	-0.07 ± 0.87	-0.12 ± 0.98	-0.23 ± 0.94	-0.41 ± 0.97	**0.004**
	Detroit	-0.63 ± 0.59	-0.58 ± 0.64	-0.51 ± 0.66	-0.67 ± 0.67	0.097
	Toronto	0.05 ± 0.86	0.03 ± 0.98	-0.02 ± 0.96	-0.19 ± 1.01	0.092
	Italy	-0.77 ± 0.96	-0.81 ± 1.10	-0.89 ± 1.08	-1.08 ± 1.13	**0.035**
	Lauzon-Schnittka	-0.14 ± 0.74	-0.11 ± 0.79	-0.02 ± 0.80	-0.04 ± 0.83	0.370
Left ventricular end-diastolic diameter (LVEDD), mm (n=1878)		39.30 ± 4.45 n=134	41.10 ± 4.97 n=964	42.99 ± 5.19 n=483	43.65 ± 5.26 n=297	**<0.001**
	PHN	0.00 ± 0.88	0.00 ± 0.89	-0.22 ± 0.90	-0.46 ± 0.90	**<0.001**
	Detroit	-0.23 ± 0.71	-0.13 ± 0.76	-0.18 ± 0.84	-0.65 ± 1.48	**<0.001**
	Toronto	-0.05 ± 1.04	-0.02 ± 1.07	-0.25 ± 1.11	-0.51 ± 1.12	**<0.001**
	Lauzon-Schnittka	-0.11 ± 1.01	-0.05 ± 1.03	-0.02 ± 1.11	-0.07 ± 1.08	0.840
Septal wall (SW), mm (n=1875)		5.92 ± 1.04 n=134	6.21 ± 1.22 n=962	6.57 ± 1.15 n=483	6.87 ± 1.26 n=296	**<0.001**
	PHN	0.08 ± 1.03	0.11 ± 1.05	0.12 ± 0.99	0.19 ± 0.95	0.695
	Detroit	-0.12 ± 0.70	-0.12 ± 0.74	-0.16 ± 0.73	-0.24 ± 0.78	0.173
	Toronto	-0.25 ± 1.21	-0.22 ± 1.27	-0.20 ± 1.19	-0.11 ± 1.14	0.631
	Lauzon-Schnittka	0.49 ± 0.96	0.42 ± 0.96	0.34 ± 0.92	0.27 ± 0.85	0.059
Posterior wall (PW), mm (n=1875)		5.81 ± 1.08 n=133	6.13 ± 1.20 n=961	6.57 ± 1.22 n=484	6.82 ± 1.37 n=297	**<0.001**
	PHN	0.05 ± 0.98	0.14 ± 1.01	0.23 ± 1.02	0.24 ± 0.96	0.136
	Detroit	0.23 ± 0.81	0.26 ± 0.81	0.25 ± 0.83	0.08 ± 0.88	**0.025**
	Toronto	-0.04 ± 1.04	0.02 ± 1.04	0.07 ± 1.04	0.06 ± 0.96	0.689
	Lauzon-Schnittka	0.45 ± 1.02	0.43 ± 1.02	0.40 ± 1.02	0.30 ± 0.91	0.289
Left atrium (LA), mm (n=1500)		24.46 ± 3.52 n=93	26.17 ± 3.41 n=796	28.17 ± 3.61 n=381	29.03 ± 4.45 n=230	**<0.001**
	PHN	1.63 ± 0.94	1.69 ± 0.78	1.75 ± 0.72	1.71 ± 0.73	0.575
	Detroit	0.76 ± 0.87	0.95 ± 0.74	1.11 ± 0.74	0.87 ± 1.09	**<0.001**
	Toronto	-0.19 ± 1.03	-0.03 ± 0.86	0.13 ± 0.81	0.12 ± 0.86	**0.001**
Tricuspid valve annulus (TV), mm (n=1037)		23.06 ± 3.44 n=83	24.34 ± 3.75 n=544	25.93 ± 3.88 n=244	25.29 ± 3.67 n=166	**<0.001**
	PHN	-0.29 ± 0.88	-0.19 ± 1.00	-0.25 ± 0.95	-0.57 ± 0.89	**<0.001**
	Detroit	-0.71 ± 0.59	-0.56 ± 0.63	-0.46 ± 0.62	-0.68 ± 0.61	**0.001**
	Toronto	-0.14 ± 0.98	0.02 ± 1.06	0.04 ± 1.04	-0.30 ± 1.00	**0.004**
	Italy	-0.48 ± 0.80	-0.38 ± 0.88	-0.40 ± 0.85	-0.69 ± 0.82	**0.001**
	Lauzon-Schnittka	-0.09 ± 0.86	-0.01 ± 0.93	0.08 ± 0.93	-0.24 ± 0.88	**0.009**
Pulmonary valve annulus (PV), mm (n=738)		18.41 ± 3.22 n=57	19.67 ± 3.57 n=372	21.48 ± 3.37 n=185	21.66 ± 3.76 n=124	**<0.001**
	PHN	-0.22 ± 1.04	-0.10 ± 1.10	-0.03 ± 1.06	-0.23 ± 1.02	0.407
	Detroit	-0.12 ± 0.84	-0.02 ± 0.92	0.08 ± 0.88	-0.20 ± 1.04	0.087
	Toronto	0.05 ± 1.20	0.14 ± 1.27	0.17 ± 1.22	-0.08 ± 1.18	0.370

Cardiac structure	Nomogram	Undernourished	Eutrophic	Overweight	Obesity	p value
	Italy	0.05 ± 1.19	0.14 ± 1.26	0.18 ± 1.21	-0.07 ± 1.17	0.370
	Lauzon-Schnittka	-0.55 ± 0.84	-0.32 ± 0.90	-0.10 ± 0.88	-0.09 ± 0.85	**<0.001**

Figures

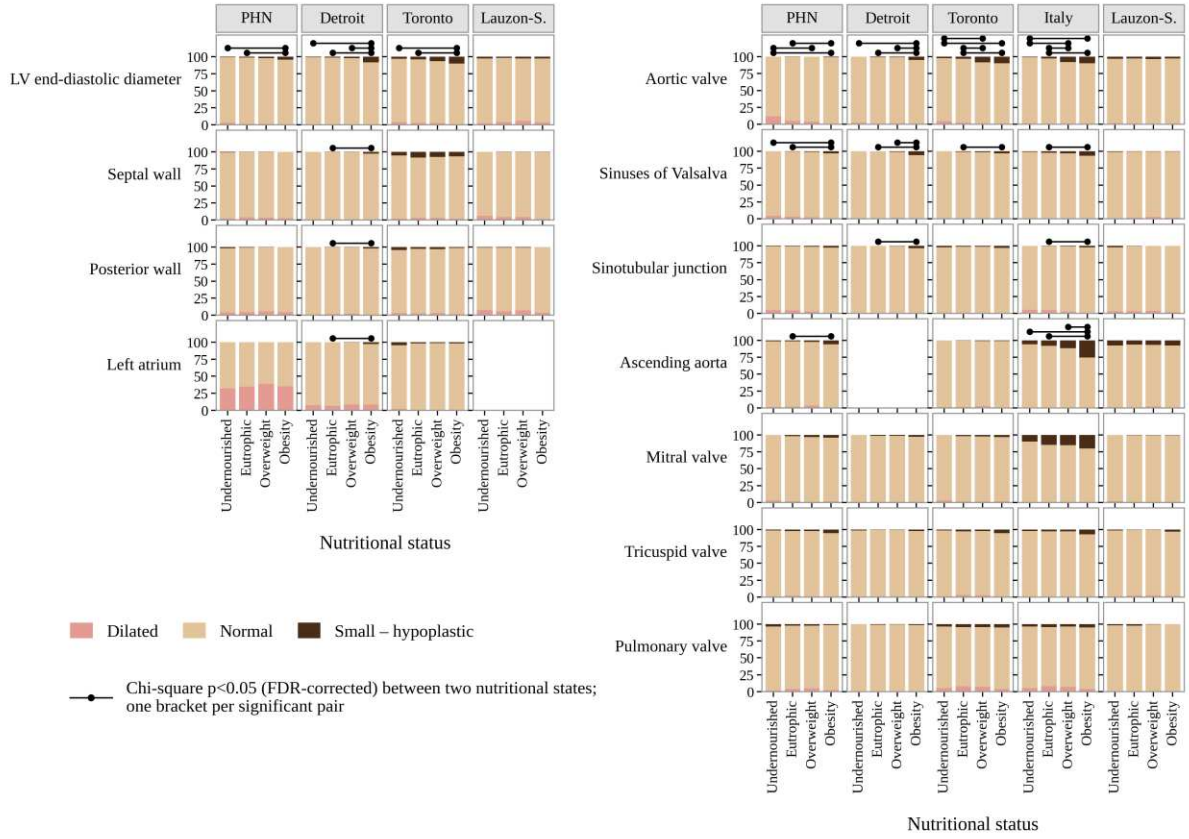


Fig. 1 Cardiac structure size classification (normal, dilated or small/hypoplastic) by nutritional status under each Z score nomogram. Bars show the proportion (%) of children classified as dilated, normal or small/hypoplastic within each nutritional category (undernourished, eutrophic, overweight, obesity) for each of the 49 structure-by-nomogram combinations. Brackets mark each pairwise comparison between two nutritional groups reaching significance by chi-square test after Benjamini-Hochberg false discovery rate correction ($p<0.05$), across 18 panels. The two blocks group structures by nomogram availability: the left block contains left ventricular end-diastolic diameter, septal wall and posterior wall, available in four nomograms (PHN, Detroit, Toronto, Lauzon-Schnittka), together with the left atrium, available in three (PHN, Detroit, Toronto) and without a GAMLSS equation; the right block contains the aortic, mitral, tricuspid and pulmonary valve annuli, sinuses of Valsalva and sinotubular junction, available in all five, together with the ascending aorta, available in four (PHN, Toronto, Italy, Lauzon-Schnittka). PHN, Pediatric Heart Network; GAMLSS, generalized additive model for location, scale and shape

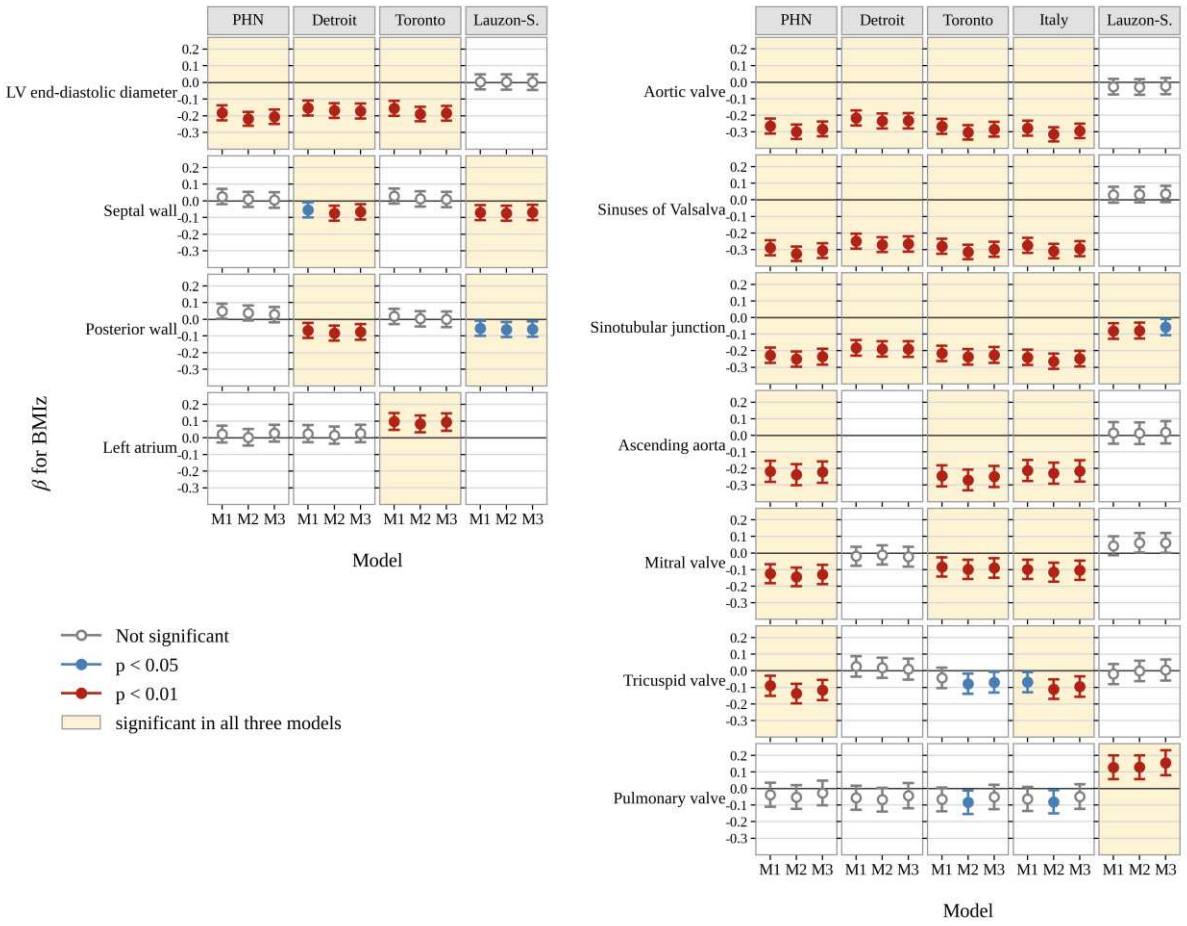


Fig. 2 Linear regression of the association between BMIz and each cardiac structure Z score, across nomograms and the three regression models. Each panel corresponds to a cardiac structure (rows) and a nomogram (columns). Points are standardized β coefficients for BMIz and vertical bars are 95% confidence intervals, shown for three models: M1 unadjusted, M2 adjusted for age and sex, M3 adjusted for age, sex and height. Marker color encodes statistical significance after Benjamini-Hochberg false discovery rate correction: grey open symbol, not significant; blue, $p<0.05$; red, $p<0.01$. Amber-shaded panels are those in which the association reached significance in all three models. The horizontal reference line marks $\beta = 0$. PHN, Pediatric Heart Network

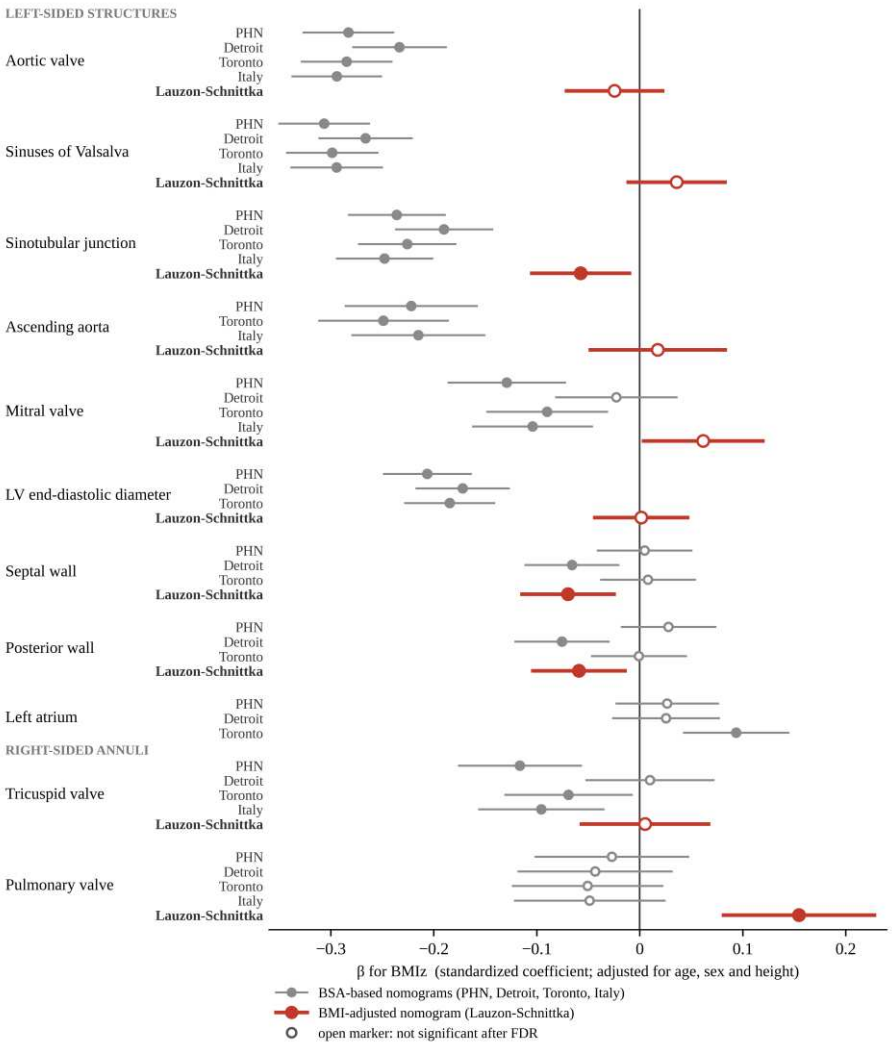


Fig. 3 Regression coefficient β for BMIz (fully adjusted model) for each cardiac structure across the five nomograms. Each point is the standardized β for the association between BMIz and the structure Z score, adjusted for age, sex and height; horizontal lines are 95% confidence intervals. Grey denotes the four body-surface-area-based nomograms and red the BMI-adjusted GAMLSS nomogram (Lauzon-Schnittka); within each structure the rows follow the order given by the axis labels. Filled markers indicate $p<0.05$ after Benjamini-Hochberg false discovery rate adjustment and open markers are not significant. A negative β means the Z score falls as BMI rises. The BSA-based nomograms give consistently negative coefficients for the aortic levels, mitral annulus and left ventricular end-diastolic diameter, whereas the BMI-adjusted nomogram gives coefficients close to zero for most of them, with associations persisting in the ventricular walls and sinotubular junction and reversing in sign in the pulmonary annulus

Supplementary Files

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