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Obesity trends in children, adolescents, and young adults with congenital heart disease

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Abstract

Objectives: To determine the prevalence, age of onset, and risk factors for overweight and obesity in children with congenital heart disease (CHD).

Study Design: Children with CHD who were seen at our institution from 1996 to 2017 were studied. Patients were full-time residents of the United States and were receiving all cardiac care at our institution. Patients were categorized by age and CHD diagnosis. The date of last normal weight for age and the date of first recorded weight in the range of overweight and obese were documented.

Results: Nine hundred sixty-eight patients with CHD were included. The prevalence of overweight and obesity was 31.5% and 16.4%, respectively. For patients who became overweight or obese, the last recorded normal weight was between 6 and 10 years of age. Electrophysiologic disease and older age were risk factors for obesity.

Conclusions: Children with CHD have an increasing risk of becoming overweight and obese in early childhood. This study provides important information and identifies critical period to implement preventative measures and counsel families about the risk of obesity in CHD.

KEYWORDS

adult congenital heart disease, congenital heart disease, epidemiology, obesity, preventive cardiology

1 | INTRODUCTION

Over the past 20 years, pediatric obesity has increased to epidemic levels. As of 2016, the overall prevalence of obesity in children was 18.5%.[1] Data show statistically significant differences highlighting increased prevalence in adolescents, certain ethnicities, and those with lower socioeconomic status.[1-4]

Obesity has significant health consequences in congenital heart disease (CHD) including increased postoperative mortality and hospital utilization and would be disadvantageous for patients requiring multiple procedures.[5,6] There is controversy in the literature on the prevalence of obesity in children with CHD. Some studies suggest that children with CHD are at an increased risk of obesity compared to the general population.[7] This could be due to the more sedentary lifestyle, which was previously discovered by cardiologists, as well as psychosocial factors.[7] Other studies concluded that the prevalence of overweight and obesity in the CHD population mirrors that of the general population, with the exception of single ventricle disease.[5,8] Some other studies inferred that the obesity rates in children with CHD are likely much higher than the general population.[9] Data analyzed in previous studies are over 10 years old; while it is known in the general population that the risk of obesity has increased in

the last 10 years with the "obesity epidemic,"[6,10] previous studies on patients with CHD have not defined the timing of overweight and obesity onset which can be critical for preventive efforts.

It cannot be assumed that the risks for and prevalence of overweight and obesity are the same for patients with CHD as the general population. The goals of this study were to determine the current prevalence of overweight and obesity in children, adolescents, and young adults with CHD. More importantly, we sought to assess at what age range patients with CHD transition from underweight/normal weight to obesity and to identify the risk factors for overweight and obesity.

2 | METHODS

In this single center, cross-sectional, longitudinal-retrospective study, we reviewed the charts of all patients between the ages of 2 and 21 years who presented to the ambulatory Pediatric Cardiology Clinic at Cleveland Clinic Children's from 1 January 1996 until 31 December 2017. Only patients with hemodynamically significant CHD, defined as patients who required surgical or catheter intervention on their CHD before 21 years of age, were included. Patients were required to have had at least three visits to our clinic and be evaluated at least annually. They had to be full-time residents of the United States of America. To eliminate referral bias, our pediatric cardiology clinic had to be their only location of cardiac care. Exclusion criteria were any patients with a genetic disease or syndrome (eg, Marfan syndrome or Duchenne muscular dystrophy), chromosome anomaly (eg, trisomy 21) or comorbidities which are known to adversely affect body habitus (eg, severe mental health disease, endocrinopathy—even if well controlled), malignancy, tumor, or history of premature birth. Patients with any cardiomyopathy, regardless of function, were excluded. The approval for this study was received from our institutional review board.

Clinical and demographic data collected from the electronic health record included: age, gender, race, zip code (to assess socioeconomic status based on median income of that area obtained from census data), cardiac diagnosis and date of diagnosis, date of most recent catheterization or surgery, status of repair/palliation, history of gastrostomy (G) or jejunostomy (J) tube, current weight and body mass index (BMI), and the date of last normal weight. Patients were divided into normal weight, overweight, and obese based on their most recent clinic visit. All measurements were obtained from pediatric cardiology clinic visits only to minimize discrepancies in the technique. BMI was calculated at each visit (kg/m^2) and was plotted on the growth charts of the Center for Disease Control for patients' ages 2-19 years to determine the percentile.[1] A BMI below the 85th percentile was defined as normal weight, 85th-94th percentile was classified as overweight, and 95th percentile or higher is classified as obese.[11] Patients between the ages of 20-21 were defined as overweight and obese according to adult norms.

The records of at least the previous three visits to our clinic were reviewed and the BMI percentile were recorded. If a patient progressed from normal weight to overweight or obese, the date of those first recorded weights was obtained in order to determine the age range in which the patient transitioned from normal weight to overweight or obese, or from overweight to obese. Only the most recent measurements were used. If a patient was previously overweight or obese but returned to normal weight, they were classified as a normal weight patient.

Patients were categorized into four groups according to the date of birth to account for advances in the management of CHD and compared across time. Examples of such changes include the introduction of the Sano shunt in single ventricle disease. Patient cohorts by birth year were: 1996-2000, 2001-2005, 2006-2010, and 2011-2015. Patients were then divided into four main categories based on their diagnosis: acyanotic, cyanotic, complex (single ventricle physiology, systemic right ventricle, and patients requiring staged operations/palliation), and electrophysiologic. The definition of an electrophysiology patient is one with an otherwise structurally normal heart with an electrophysiology diagnosis in childhood. In the electrophysiology group, the definition of intervention included electrophysiologic studies, internal cardiac defibrillator (ICD) and pacemaker placement, and the need for medications due to the risk of hemodynamically significant arrhythmias.

3 | STATISTICAL ANALYSIS

Data were described using median and quartiles for continuous variables and counts and percentages for categorical variables. The prevalence of overweight and obese was calculated for the entire cohort. Group comparisons used analysis of variance (ANOVA) or Kruskal-Wallis test for continuous variables and Pearson's chi-square test or Fisher's exact test for categorical variables.

Two analyses were performed: the first on the entire cohort based on the most recent clinic visit data; and the second to assess the timing of progression to overweight and obese in patients who began at a normal weight. The second analysis excluded patients who were overweight/obese before the beginning of the study and patients who returned to normal weight during the study. The rates of overweight and obesity at different ages were estimated using the Kaplan-Meier method for time to event data, for the events "becoming overweight from normal weight" or "becoming obese from nonobese," with censoring at last follow-up, and the date of diagnosis (or surgery date if the diagnosis date was missing) as the start point. Cox proportional hazards regression left-truncated models were used to compare patients with different lesions and to adjust for birth year. In the time-to-overweight analysis, 147 patients were excluded for the following reasons: as they either have incomplete information available in the chart or were overweight before the beginning of the study. Similarly, in

TABLE 1 Demographics and clinical features of the entire cohort at the time of the last clinic visit

Factor	Total (*N* = 968) Statistics
Overweight	305 (31.5)
Obese	159 (16.4)
Gender	
Male	549 (56.7)
Female	419 (43.3)
Race	
Caucasian	784 (81.0)
Black/African American	97 (10.0)
Latino/Hispanic	30 (3.1)
Asian	25 (2.6)
Unknown	32 (3.3)
Birth cohort (5 year intervals)	
1996-2000	327 (33.8)
2001-2005	336 (34.7)
2006-2010	230 (23.8)
2011-2015	75 (7.7)
Median household income (per $1000)	*
<46	240 (24.9)
46-<57.8	241 (25.0)
57.8-<69	240 (24.9)
>69	243 (25.2)
Lesion type	
Cyanotic	256 (26.4)
Acyanotic	258 (26.7)
Complex	208 (21.5)
Electrophysiologic	246 (25.4)
Repaired or palliated	837 (86.5)
History of G or J tube	28 (2.9)

Statistics presented as median (min, max) or N (column %).
*Only 964 patients' income could be assessed.

the time-to-obese analysis, 76 patients were excluded due to similar circumstances.

All *P* values are two-sided, with 0.05 as the level of statistical significance. Statistical analysis was performed using SAS software version 9.4 (SAS Institute Inc., Cary, North Carolina).

4 | RESULTS

Nine hundred and sixty-eight patients were studied. Demographic information of the entire cohort is presented in Table 1. The prevalence of overweight and obesity was 31.5% and 16.4%, respectively. When compared to patients with normal weight, patients who were overweight were older (more born 1996-2005), had lower median household income, and were less likely to have had G or J Tube. The overweight patients were on average 1.2 years older at the time of their last intervention.

When compared to patients with normal weight, obese patients were less likely to be Asian or unknown race, and more likely to be older (born between 1996 and 2005) with lower median household income. Patients in the 2011-2015 cohort had an obesity prevalence of 3% compared to 13.9% of children that age in the general population.[1] Patients born in the 1996-2000 cohort reached approximately 20% overweight at 16 years compared to the 2006-2010 cohort who reached the same level at approximately 10 years of age (Figure 1). A similar finding was noted in regard to the development of obesity (Figure 2).

4.1 | Time-to-overweight analysis

On univariate analysis, age was a risk factor for becoming overweight ($P < .001$), while gender ($P = .092$) and median household income ($P = .30$) were not (Table 2).

When stratified by birth year, we noticed a significant gradual increase in the hazard of becoming overweight in the more recent year ($P < .001$, Figure 1). Using 2011-2015 as a reference, the hazard ratio of becoming overweight in the 1996-2000 cohort was 0.12 (CI 0.04-0.33) which reflects an 88% decrease in the hazard of becoming overweight (Table 3). Similarly in the 2001-2005 cohort, the HR is 0.35 (CI: 0.13-0.96) and in the 2006-2010 the HR is 0.55 (CI: 0.20-1.49).

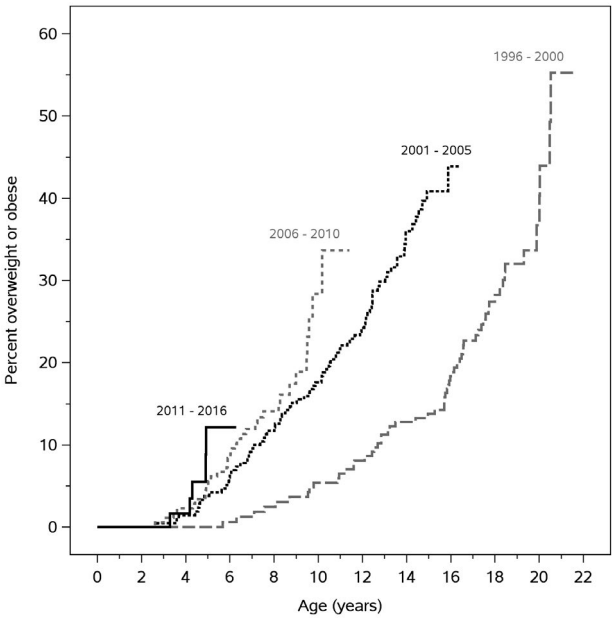

FIGURE 1 Survival function plot for age of progression from normal weight to overweight or obese, by birth year cohort

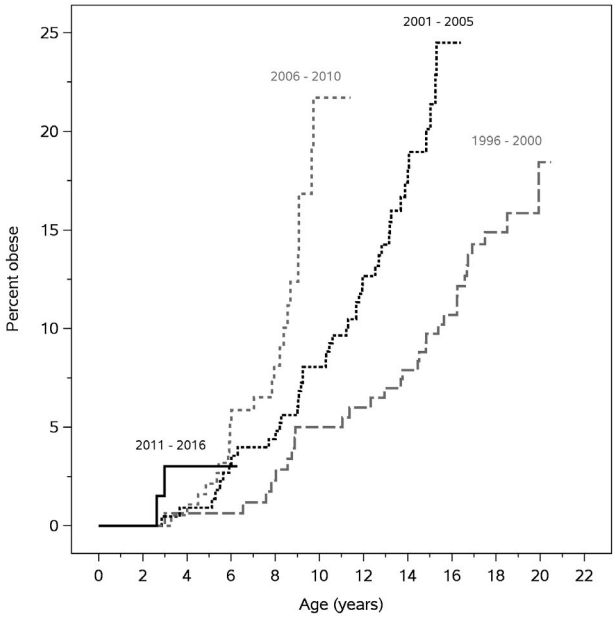

FIGURE 2 Survival function plot for age of progression from nonobese to obese, by birth year cohort

CHD subgroup diagnosis had no significant association with "becoming overweight" ($P = .058$).

On multivariate analysis, complex CHD ($P = .008$) and median household income < 46 (per $1000) were risk factors for becoming overweight ($P = .031$); however, the remaining CHD subgroups and income categories did not reach statistical significance. Birth cohort remained significant with $P < .001$. Hazard ratio (HR) and 95% confidence interval (CI) for each level of categorical variables are listed in Table 3.

4.2 | Time-to-obese analysis

On univariate analysis, gender ($P = .67$) and median household income ($P = .33$) were not risk factors for "becoming obese." Stratified by birth year, we noticed the probability of becoming obese increased with age ($P < .001$, Figure 2). Again, using 2011-2015 as a reference, the hazard of becoming obese in the 1996-2000 cohort was 0.18 (CI 0.04-0.86). Similarly, in the 2001-2005 cohort, the HR is 0.43 (CI: 0.09-1.99) and in the 2006-2010 cohort, the HR is 0.91 (CI: 0.20-4.13) (Table 4).

CHD diagnosis also had significant association with "becoming obese" ($P = .009$, Figure 3). Patients with electrophysiologic disease were at the highest risk of becoming obese and were used as a reference followed by:

acyanotic: HR 0.68 (CI: 0.39-1.18),

complex HR 0.38 (CI: 0.21-0.70)

and cyanotic: HR 0.48 (CI: 0.27-0.83).

On multivariate analysis, median household income of the 46 - <57.8 (per $1000), birth year cohort, and CHD diagnosis were significant predictors of obesity. Hazard ratio (HR) and 95% confidence interval (CI) for each level of categorical variables are listed in Table 4.

For patients who became overweight, the median age of last normal weight was nine years, and the first recorded overweight was 10.3 years. Children who developed obesity had a last normal weight recorded at a median age of 6.5 years and were obese at a median age of 9.6 years (Tables 2 and 5).

5 | DISCUSSION

Our results indicate that the current overall prevalence of overweight (31.5%) and obesity (16.4%) in CHD is comparable to published data on the general population.[1-3] The prevalence of overweight and obesity in our study is similar to that obtained from a recent study of patients with congenital heart disease.[12] Our study identified a high risk period of 6-10 years of age as the time when patients in this population who were of normal weight were most likely to become overweight and obese.

Our study found that after adjusting for age, there is an increased risk for overweight/obesity in patients born in the more recent eras. The 1996-2000 patient birth year cohort was at a lower risk of developing overweight/obesity compared to patients born between 2001 and 2015. In our study population, gender, race, and socioeconomic status were not identified as significant risk factors for becoming overweight or obese.

Type of CHD was not a risk factor for the development of overweight, but comparable to previous studies, complex CHD was found to be protective from developing obesity and electrophysiologic disease was found to be a risk factor for becoming obese.[5,8]

Isolated interpretation of our data could be significantly misleading. Analysis of the incidence of becoming overweight and obese showed that the younger patients, those born between 2006 and 2015, are becoming overweight and obese at a significantly more rapid pace (Figure 1). A similar finding was noted in regard to the development of obesity (Figure 2). Thus, if this study was repeated in 5 to 10 years, allowing the oldest cohort to phase out, the prevalence of overweight and obesity in patients with CHD would be expected to be significantly higher. Though our youngest cohort had only a 3% prevalence of obesity, the number of patients was small (75). Most of our patients required interventions prior to age five, thus the hemodynamic significance of their CHD is highest during that time period which hinders their growth during that time period. As these patients become older, our results suggest the prevalence of obesity will increase.

There are no clear answers as to why children with CHD are trending toward increased prevalence of overweight and obesity. We believe the answer is multifactorial. Children with CHD may be perceived as more vulnerable by their families who then proceed to over restrict their activities.[7] This may explain why even completely recovered patients with CHD trend toward higher BMIs. In children

TABLE 2 Comparison of overweight vs nonoverweight in the time-to-event analysis

Factor	Overall (*N* = 821)		Non-Overweight (*N* = 641)		Overweight (*N* = 180)		*P* value
	N	Statistics	*N*	Statistics	*N*	Statistics	
Gender							.51[c]
Male		460 (56.0)		363 (56.6)		97 (53.9)	
Female		361 (44.0)		278 (43.4)		83 (46.1)	
Race							.24[c]
Caucasian		667 (81.2)		522 (81.4)		145 (80.6)	
Black/African American		83 (10.1)		62 (9.7)		21 (11.7)	
Latino/Hispanic		22 (2.7)		16 (2.5)		6 (3.3)	
Asian		23 (2.8)		22 (3.4)		1 (0.56)	
Unknown		26 (3.2)		19 (3.0)		7 (3.9)	
Birth cohort (5 year intervals)							**<.001[c]**
1996-2000		256 (31.2)		196 (30.6)		60 (33.3)	
2001-2005		295 (35.9)		213 (33.2)		82 (45.6)	
2006-2010		201 (24.5)		168 (26.2)		33 (18.3)	
2011-2015		69 (8.4)		64 (10.0)		5 (2.8)	
Median household income (per $1000)	818		639		179		.41[c]
<46		199 (24.3)		147 (23.0)		52 (29.1)	
46-<57.8		197 (24.1)		155 (24.3)		42 (23.5)	
57.8-<69		207 (25.3)		165 (25.8)		42 (23.5)	
>69		215 (26.3)		172 (26.9)		43 (24.0)	
Lesion type							.35[c]
Cyanotic		232 (28.3)		174 (27.1)		58 (32.2)	
Acyanotic		217 (26.4)		167 (26.1)		50 (27.8)	
Complex		188 (22.9)		149 (23.2)		39 (21.7)	
Electrophysiologic		184 (22.4)		151 (23.6)		33 (18.3)	
Repaired or palliated		719 (87.6)		557 (86.9)		162 (90.0)	.26[c]
History of G or J tube		25 (3.0)		21 (3.3)		4 (2.2)	.47[c]
Age of diagnosis	740	0.00[0.00,1.5]	579	0.00[0.00,1.5]	161	0.00[0.00,0.90]	.45[b]
Age of last treatment	716	4.1[0.57,10.9]	555	4.1[0.51,11.3]	161	4.5[0.80,10.6]	.60[b]
Age of last clinic visit	821	13.3[8.8,16.4]		12.9[8.4,16.3]		14.1[10.5,17.2]	**<.001[b]**
Age of last normal weight	819	12.1[7.7,15.7]		12.9[8.4,16.2]	178	9.0[5.7,12.7]	**<.001[b]**
Age of overweight	180	10.3[6.8,13.9]	0	—		10.3[6.8,13.9]	

Statistics presented as median [P25, P75] or N (column %).
P values: a = ANOVA, b = Kruskal-Wallis test, c = Pearson's chi-square test, d = Fisher's exact test.
Bold values represent statistically significant differences between the groups.
In "nonoverweight" column, N = 641 unless otherwise stated.
In "overall" column, N = 821 unless otherwise stated.
In "overweight" column, N = 180 unless otherwise stated.

with activity restrictions, parents may also not fully comprehend the true activity restrictions placed on their child, and therefore over restrict their activity.[13,14] Activity restriction has previously been shown to be a risk factor for becoming obese in this patient population.[15] Patients with CHD may also subconsciously restrict themselves even when no restrictions are placed on them by their cardiologists. Massin et al showed that patients with transposition of the great arteries after arterial switch operation were less likely than their peers to participate in moderate to vigorous activity.[16] There is also a psychological impact on parents with regard to feeding patterns in these children since many of them had poor growth or failure to thrive in the neonatal and infant period.[9]

Previously, electrophysiologic disease had been found to be a risk factor for becoming obese and this is likely due to activity restriction.[7]

TABLE 3 Hazard ratio, 95% confidence interval, and *P* values for the multiple regression model of overweight patients

Variable	Hazard ratio	95% CI		*P* value
Lesion type acyanotic	0.768	0.490	1.202	.25
Lesion type complex	0.527	0.327	0.849	.008
Lesion type cyanotic	0.722	0.466	1.119	.14
Birth cohort (5 year intervals)				
1996-2000	0.113	0.039	0.325	<.001
2001-2005	0.358	0.131	0.975	.044
2006-2010	0.538	0.198	1.459	.22
Median household income (per $1000)				
46-<57.8	1.187	0.775	1.818	.43
57.8-<69	0.998	0.650	1.533	.99
<46	1.569	1.042	2.364	.031

TABLE 4 Hazard ratio, 95% confidence interval, and *P* values for the multiple regression model of obese patients

Variable	Hazard ratio	95% CI		*P* value
Lesion type acyanotic	0.701	0.406	1.213	.20
Lesion type complex	0.401	0.217	0.744	.004
Lesion type cyanotic	0.498	0.283	0.876	.016
Birth cohort (5 year intervals)				
1996-2000	0.182	0.037	0.895	.036
2001-2005	0.439	0.094	2.049	.29
2006-2010	0.896	0.196	4.092	.89
Median household income (per $1000)				
46-<57.8	1.823	1.047	3.174	.034
57.8-<69	1.077	0.595	1.950	.81
<46	1.656	0.927	2.957	.088

Presently, there is a movement toward loosening activity restrictions on patients with rhythm disturbances. We also speculate that these patients were at a higher risk of becoming obese because many of these patients were treated with beta-blocker medications, which can negatively impact a child's desire to be active and their cardio-metabolic drive.[17,18] Secondly, because many inherited arrhythmias are detected after a serious event (eg, family member having a cardiac arrest), families may have a perceived higher fear of a child developing a fatal arrhythmia with increased activity.

We cannot discount risk factors that negatively affect all children, such as the increased use of screen time. Screen time greater than two hours daily has been shown to be a risk factor for the development of obesity.[19] Children with CHD who have prolonged hospital stays may become more accustomed to prolonged screen time during their recovery; however, this was not assessed during our study.

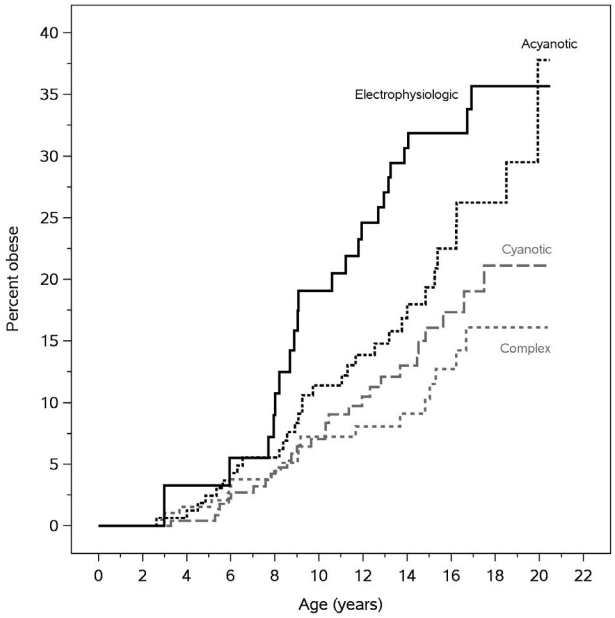

FIGURE 3 Survival function plot for age of progression from non-obese to obese, by lesion type

6 | LIMITATIONS

There were several limitations to our study. First, our study was a single-center study which may limit the generalizability of our data, although our findings are consistent with previous studies.[12] Second, our 2011-2015 cohort was smaller in size compared to the other three cohorts and obviously does not have comparable longitudinal follow-up. Third, as a retrospective study, it was not possible to obtain new information which was not previously in the patient chart such as imposed restrictions and parental body habitus which play an important role in the development of obesity. This also prevented us from obtaining details of the patients' overall physical activity level as it pertains to weight changes. Fourth, BMI is a somewhat crude measurement of growth and though it has been shown to be reliable in adults, it is certainly less reliable in children; however, it continues to be a marker of recommended use. Additionally, useful measurements such as adiposity index and abdominal girth were not available for this study. Though our study did not specifically look into the adult congenital heart disease population, a recent study showed that mortality in that population increased with decreasing BMI, suspected to be due to cardiac cachexia.[20] Despite the mentioned limitations, our study provides insights for the rising incidence of obesity and timing of incidence in CHD.

TABLE 5 Comparison of obese vs nonobese in the time-to-event analysis

	Overall (*N* = 892)		Nonobese (*N* = 787)		Obese (*N* = 105)		
Factor	***N***	**Statistics**	***N***	**Statistics**	***N***	**Statistics**	***P* value**
Gender							.98[c]
Male		500 (56.1)		441 (56.0)		59 (56.2)	
Female		392 (43.9)		346 (44.0)		46 (43.8)	
Race							.10[c]
Caucasian		719 (80.6)		635 (80.7)		84 (80.0)	
Black/African American		92 (10.3)		79 (10.0)		13 (12.4)	
Latino/Hispanic		26 (2.9)		20 (2.5)		6 (5.7)	
Asian		24 (2.7)		24 (3.0)		0 (0.0)	
Unknown		31 (3.5)		29 (3.7)		2 (1.9)	
Birth cohort (5 year intervals)							.063[c]
1996-2000		287 (32.2)		252 (32.0)		35 (33.3)	
2001-2005		316 (35.4)		272 (34.6)		44 (41.9)	
2006-2010		216 (24.2)		192 (24.4)		24 (22.9)	
2011-2015		73 (8.2)		71 (9.0)		2 (1.9)	
Median household income (per $1000)	889		784				.23[c]
<46		214 (24.1)		187 (23.9)		27 (25.7)	
46-<57.8		219 (24.6)		186 (23.7)		33 (31.4)	
57.8-<69		225 (25.3)		201 (25.6)		24 (22.9)	
>69		231 (26.0)		210 (26.8)		21 (20.0)	
Lesion type							.67[c]
Cyanotic		243 (27.2)		215 (27.3)		28 (26.7)	
Acyanotic		237 (26.6)		204 (25.9)		33 (31.4)	
Complex		200 (22.4)		179 (22.7)		21 (20.0)	
Electrophysiologic		212 (23.8)		189 (24.0)		23 (21.9)	
Repaired or palliated		775 (86.9)		686 (87.2)		89 (84.8)	.49[c]
History of G or J tube		28 (3.1)		27 (3.4)		1 (0.95)	.17[c]
Age of diagnosis	797	0.00[0.00,3.1]	708	0.00[0.00,3.3]	89	0.00[0.00,2.3]	.94[b]
Age of last treatment	770	4.2[0.67,11.5]	682	4.3[0.62,11.9]	88	4.1[1.1,9.4]	.56[b]
Age of last clinic visit		13.5[8.9,16.6]		13.5[8.6,16.5]		14.0[10.6,16.8]	.066[b]
Age of last normal weight	850	12.1[7.7,15.7]	765	12.8[8.3,16.1]	85	6.5[4.3,10.1]	**<.001[b]**
Age of obese	105	9.6[7.7,13.8]	0	—		9.6[7.7,13.8]	

Statistics presented as median [P25, P75] or *N* (column %).
P values: a = ANOVA, b = Kruskal-Wallis test, c = Pearson's chi-square test, d = Fisher's exact test.
Bold values represent statistically significant differences between the two groups.
In "overall" column N = 892, unless otherwise stated and includes overweight and obese patients.
In "nonobese" column N = 787, unless otherwise stated.
In "obese" column, N = 105, unless otherwise stated.

7 | CONCLUSIONS

Children with CHD are trending toward higher rates of overweight and obesity. The highest risk period for becoming overweight and obese is between 6 and 10 years of age. Determining this high risk period is important since multiple physicians are often involved in the care of children with CHD. Knowing their high risk period will hopefully allow physicians to work together to improve preventative measures and keep these patients within a normal weight range. Pediatric cardiologists can improve their documentation of weight concerns in clinic visit notes and be more diligent to ensure parents are aware of their child's restrictions, if any.[7] Patients with some forms of CHD have been shown to have an increased risk of premature atherosclerotic disease; thus, it is important to prevent compounding risk factors.[21]

CONFLICTS OF INTEREST

The authors have no conflicts of interest relevant to this article to disclose.

AUTHOR CONTRIBUTIONS

Jeremy Steele and Kenneth Zahka designed the study, collected the data, and are the primary authors of the manuscript.

Tamar Preminger, Francine Erenberg, Katherine Dell, and Tarek Alsaied were involved in assisting with the study design and providing important and critical editing to the final manuscript.

Lu Wang performed the complete statistical analysis and assisted with the study design.

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