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REVIEW

Clinical practice

The effect of obesity in children with congenital heart disease

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Abstract Childhood obesity has reached epidemic proportions in many parts of the world. This epidemic has also affected children and adults with congenital heart disease (CHD). Over one quarter of children with CHD are overweight or obese. Important comorbidities are associated with obesity including type 2 diabetes, systemic hypertension, hyperlipidemia, and obstructive sleep apnea. Obese children with CHD often have the traditional risk factors such as genetic predisposition, sedentary lifestyle, and poor dietary habits. However, they may also have unique risk factors such as higher caloric needs in early infancy and exercise restriction in childhood. Similar to children with normal hearts, those with CHD have higher left ventricular mass and abnormal vascular function and are more likely to have systemic hypertension. In the long term, these comorbidities may have a more profound effect on children who have underlying functional and/or anatomical abnormalities of the heart. As more children with CHD are now surviving into adulthood, investigating therapeutic interventions to treat and prevent obesity in this population is of utmost importance. Recommendations for safe physical activity, recreation sport, and exercise training for children with CHD have recently been published. These guidelines may help health care providers to change their practice of exercise restriction.

Keywords Congenital heart disease · Obesity · Children

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Introduction

In the twenty-first century, the majority of children diagnosed with congenital heart disease (CHD) will survive into adulthood [6, 15]. In fact, the population of adults with CHD has reached approximately one million [42]. At the same time that the CHD population has aged, childhood and adult obesity rates have skyrocketed in most developed regions of the world. The adult obesity rate in the USA and Canada is as high as 30 % (if overweight is included, 65 % of the population is affected). Though the obesity rate is lower in most European countries, some including the UK, Hungary, and the Czech Republic have rates over 20 %. The International Obesity Task Force estimates that 1.1 billion adults in the world are overweight [16]. The epidemic of obesity has also affected the pediatric population. Approximately 17 % of children aged 2–19 years in the USA are obese with an additional 15 % that are overweight [17]. Children with CHD may be at increased risk for obesity compared to their healthy peers; they are often restricted from physical activity because of a perceived risk of sudden death.

Childhood obesity

Childhood obesity has become a major public health problem and is the topic of much media coverage [17, 38]. Over the course of the last century, children have participated in less physical labor in and outside the home and have spent more time performing sedentary activities. At the same time, access to inexpensive, high-caloric, low-nutrition foods has increased at an alarming rate. Importantly, childhood obesity is not an isolated problem. Many important comorbidities are associated with obesity, including type 2 diabetes, systemic hypertension, obstructive sleep apnea, and hyperlipidemia; social isolation, clinical depression, and behavioral problems are common as well [4, 7]. Obesity and insulin resistance have been shown to be associated with at least a threefold increased risk for elevated levels of prothrombotic factors which may be precursors to atherosclerotic disease [14]. Moreover, 70 % of obese children become obese adults [12].

There is a strong correlation between cardiovascular risk factors such as obesity and the development of atherosclerotic disease even as early as childhood. An autopsy study on children and young adults who died from non-cardiovascular events reported that fibrous plaques or fatty streaks were common in the aorta and coronary arteries, particularly in those with more than one cardiovascular risk factor [5]. A large longitudinal cohort study of Danish adults (where childhood body mass index (BMI) was known) reported that childhood obesity was significantly associated with coronary artery events in adulthood [2].

Surrogate markers of cardiovascular disease such as left ventricular mass, carotid intima–medial thickness, and peripheral vascular function have been utilized as tools to evaluate high-risk populations such as patients with obesity [21]. The Framingham study identified that increased left ventricular mass was significantly associated with cardiovascular events and death in adults [23]. In the Strong Heart Study, anthropometric and echocardiographic data collected on 460 adolescents aged 14–20 years demonstrated that obese subjects were more likely to have inappropriate left ventricular mass and reduced left ventricular myocardial performance compared to normal-weight participants [7]. In adults, carotid intima–media thickness and peripheral endothelial dysfunction have also been associated with coronary artery disease and coronary events [1, 28]. In the pediatric population, abnormal brachial flow-mediated vasodilation (a measure of endothelial function) and increased carotid intima–media thickness (a precursor to atherosclerosis) have been reported in obese children [43]. Importantly, there is evidence that some of these findings are reversible with long-term exercise and diet regimens [44].

Prevalence of obesity in children with CHD

It has only recently emerged that the epidemic of obesity has impacted the pediatric and adult CHD populations. Recently, a working group of the National Heart, Lung, and Blood Institute met to delineate the scope of the problem [32]. The first study to recognize that obesity is prevalent in children with CHD and acquired heart disease was a cross-sectional study performed in a population of children aged 6–19 years from Philadelphia and Boston [34]. In this study, patients were classified as obese (BMI ≥95 %) or overweight (BMI ≥85–95 %) according to the CDC guidelines [17]. Blood pressure measurements from the clinic visit were also recorded and converted to percentiles. Almost 3,000 subjects were included in the study; the mean age was 12.3±3.7 years. The results are shown in Fig. 1. The population was divided into study groups to distinguish severity of heart disease including those who had a cardiology outpatient visit but had no heart disease (controls, n=1,398), those with “mild” heart disease that did not require surgical intervention (n=401), those with an arrhythmia but no structural heart disease (n=447), those who underwent a biventricular cardiac repair (e.g., septal defect closure, tetralogy of Fallot repair, arterial switch operation; n=511), those who underwent univentricular palliation culminating in the Fontan procedure (n=108), and those who underwent heart transplantation (n=56). The entire cohort had a combined prevalence of obesity and overweight of 29 %, similar to the concurrent National Health and Nutrition Examination Survey (NHANES) data in the USA. Patients with any type of heart disease had a slightly lower prevalence of overweight and obesity (26.2 %) compared to NHANES (p<0.001) and study controls (p<0.001). However, the arrhythmia patients, those with mild heart disease (HD), and those who underwent heart transplantation had the same prevalence of overweight and obesity as the NHANES data (Fig. 1). The patients who underwent Fontan operation had the lowest prevalence of overweight and obesity at 16 %. This is not surprising as patients who have undergone the Fontan procedure often have growth failure [8]. Some secondary effects of obesity were also found in this study. Systolic blood pressure and blood pressure percentiles were higher in obese and overweight patients with heart disease compared to those of normal weight (Fig. 2). A more recent study has confirmed that obesity and overweight affects 30 % of the pediatric CHD population [36]. In this New York population, Hispanic ethnicity and male gender were the groups found to be at highest risk for the development of obesity. Importantly, the prevalence of obesity appears to increase in adulthood. Approximately 54 % of adults with CHD have a BMI >25 kg/m^2 [31].

Acquired and congenital heart disease at risk for early atherosclerotic disease

A recent joint statement of the American Heart Association and American Academy of Pediatrics on risk reduction in pediatric patients emphasized the need for additional study of the prevalence and etiology of obesity, as well as presence of other cardiovascular risk factors in children with congenital and acquired HD [20]. Several categories of patients were identified to be at increased risk for the

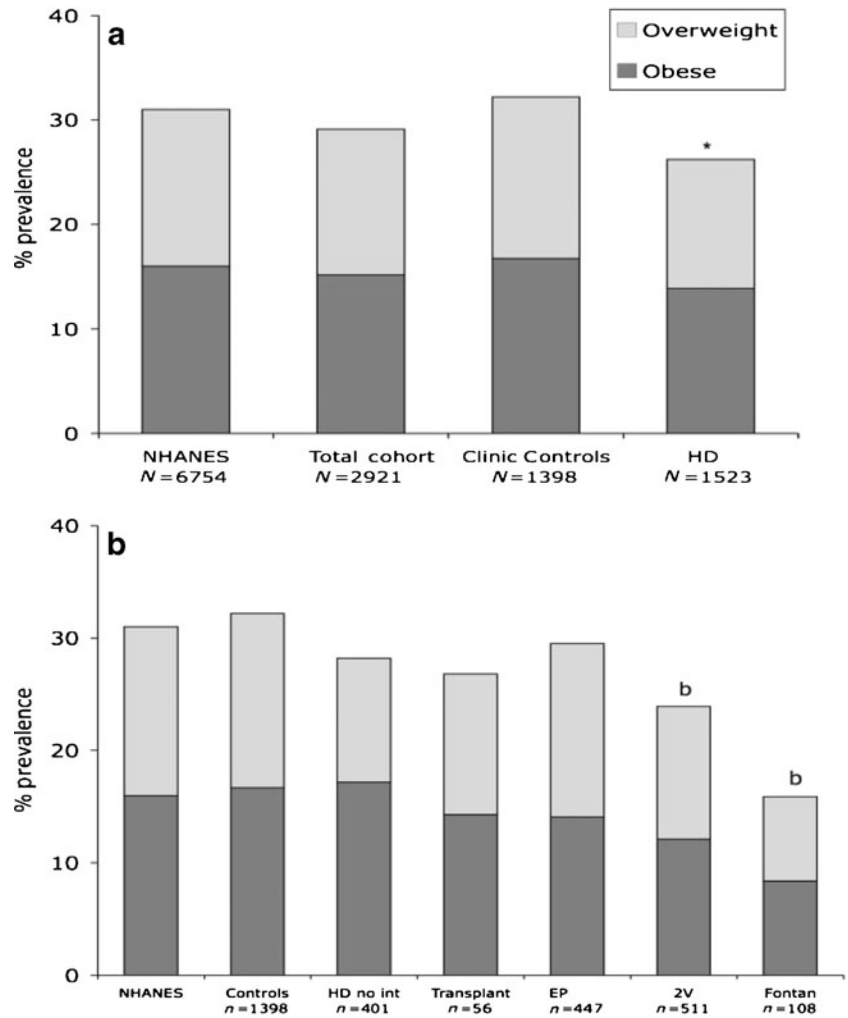

Fig. 1 Prevalence of obesity and overweight. **a** Prevalence of national data (from NHANES 1999–2002) compared with the clinic cohort and patients with HD. The heart-disease group includes all patients who had mild heart disease, EP, biventricular repair (2V), univentricular palliation, and transplant (a $P<0.03$ when compared with control patients). **b** Prevalence of national data and clinic controls against categories of heart disease (*b* $P<0.01$ for comparisons with national data and controls). All other *P* values were not significant. (Pinto et al. [34], used with permission)

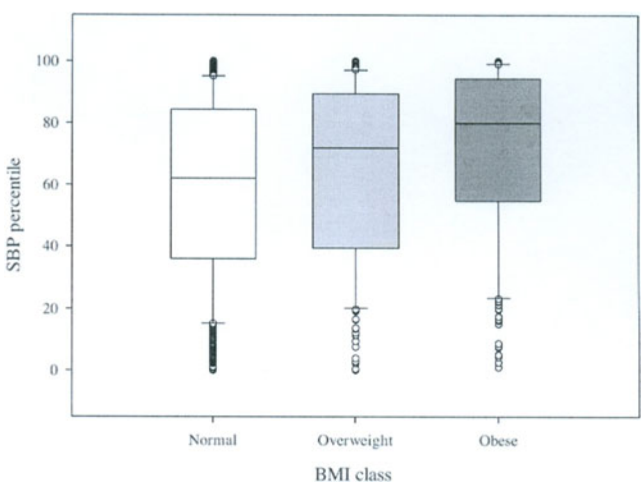

Fig. 2 Systolic blood pressure (*SBP*) percentiles according to BMI class for patients with heart disease. Systolic blood pressure percentile was increasingly higher in obese and overweight patients when compared to those patients with normal BMI ($p<0.001$ for each comparison of overweight and obese against normal BMI patients). These figures were unadjusted for medication use. (Pinto et al. [34], used with permission)

development of premature atherosclerotic cardiovascular disease based on their underlying cardiac disease. These include patients with congenital (e.g., anomalous aortic origin of the coronary artery) or acquired coronary artery abnormalities (e.g., Kawasaki disease), patients who have undergone surgical translocation of the coronary arteries (e.g., arterial switch operation [18] or Ross operation [35]), patients with left heart obstructive lesions (e.g., coarctation of the aorta), and patients who have undergone cardiac transplantation. Children with Kawasaki disease and other coronary artery anomalies are at continued risk for coronary events through childhood and adulthood [13]. Sudden coronary death is also known to occur after the arterial switch operation [22]. Approximately 11 % of heart transplant recipients develop coronary artery vasculopathy within 5 years after cardiac transplant; it is the leading cause of mortality in this population [9]. Thus, monitoring for the development of atherosclerotic disease in high-risk patients is critically important, and avoidance of obesity should be one of the goals of long-term care.

Risk factors for the development of obesity in children with CHD

Traditional risk factors may contribute to the development of obesity in children and adults with CHD. In fact, the very same risk factors that have led to the epidemic of obesity in the general pediatric population have had a significant impact on the CHD population as well. A recent study assessed lifestyle risk factors for future atherosclerotic disease in a cohort of over 300 Belgian and German children with CHD [26]. Nearly half the children surveyed drank sweetened beverages, and almost 90 % ate foods high in fat at least three times a week. In addition, 13 % were active smokers, and 54 % spent more than 2 h per day at sedentary activities. Over 9 % of European adults with CHD are active smokers, and it has had an impact on mortality [11]. In a US cohort of adolescents who underwent the Ross operation or the arterial switch operation (both include coronary translocation), the median amount of time per week spent in sedentary activity was 40 h with only 3 h per week spent participating in vigorous physical activity [30]. Others have confirmed that healthy patients who have undergone the arterial switch operation participate in significantly less vigorous physical activity than healthy controls [37].

There are also unique risk factors for obesity that affect the CHD population. Failure to thrive is a common symptom of heart failure in children with unrepaired heart defects. Thus, maximizing caloric intake and encouraging weight gain have been an important component of overall health care in these at-risk children. Though failure to gain weight is a valid concern prior to cardiac intervention, many children have normal caloric requirements and metabolism after cardiac repair. Yet parents and health care providers may continue to encourage and emphasize increased caloric intake and weight gain even as the children are becoming overweight [41]. It has recently been reported in a large global population study that infant weight gain is associated with the development of childhood obesity [10]. Each one-unit increase in weight *z* score between 0 and 12 months of age conferred a twofold risk of childhood obesity and a 23 % higher risk of obesity in adulthood [10]. Others have shown that the earliest sign of a trend toward childhood obesity was the rapid increase of weight and weight gain velocity after 6 months of age [45].

Restriction from physical activity is also a common practice that is relatively unique to the CHD population. Prior to the ability to perform catheter-directed and surgical interventions, patients with uncorrected CHD were often quite disabled, and exercise restriction was a valid recommendation to prevent sudden death. Though some at-risk populations such as those with valvar aortic stenosis, pulmonary hypertension, and hypertrophic cardiomyopathy remain vulnerable during periods of intense physical activity [24], the majority of children and adults with repaired or mild CHD can exercise safely and even participate in competitive athletics [27, 39, 40]. Exercise restriction may be recommended by health care providers even when not specifically indicated; some base their recommendations on outdated practice models, fear of medical liability, or personal bias. However, some restrictions are imposed by parents or guardians rather than practitioners. With the media reporting many instances of sudden death on sports fields, parents are often fearful of allowing their children with CHD to participate in athletic activities. These children are sometimes perceived as being more vulnerable to harm than their peers. As children with CHD become young adults, they may impose restrictions on themselves as well [3, 25]. Patients are often self-conscious about their appearance or their ability to exercise with the same capacity as their healthy peers. Adolescents with single ventricle palliation have been reported to have lower body image than age-matched controls [33]. Importantly, formal activity restriction in children with CHD has been associated with higher BMI [37].

Cardiovascular sequelae of obesity in children with CHD

Obese children without HD have been shown to have surrogate markers of early cardiovascular disease including higher carotid intima–media thickness and abnormal endothelial function [21, 43]. The presence of childhood obesity in association with CHD may have an even more profound impact on long-term outcome; patients with CHD often have underlying anatomic or functional abnormalities that cannot be altered by treatment. Thus, obesity may be the only modifiable risk factor in this population. Several studies suggest that obesity in the setting of CHD has an impact on overall health. An at-risk population of children who had the arterial switch operation (for transposition of the great arteries) or the Ross procedure (a pulmonary autograft aortic valve replacement) was recently investigated; this population was chosen because they are considered at risk because of the coronary translocation that is performed in both operations [20]. Nearly one third of the cohort was obese or overweight [30]. The most

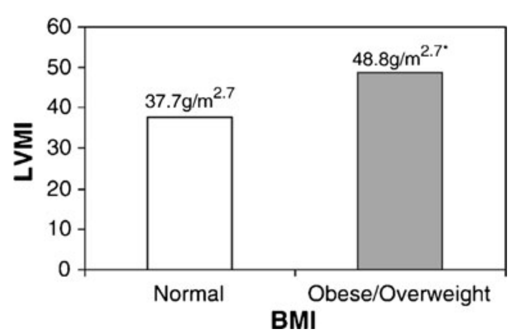

Fig. 3 Left ventricular mass index measured in children who have undergone the arterial switch operation. *LVMI* left ventricular mass index. P=0.01 obese and overweight patients compared to normal-weight patients. (Pasquali et al. [30], used with permission)

significant risk factor for obesity was parental obesity (defined as a parent with a BMI ≥30). Obese and overweight subjects had higher indexed left ventricular mass compared to normal-weight peers who had the same underlying CHD (Fig. 3) [30]. In a subgroup of this cohort, obese and normal-weight adolescents who had the arterial switch operation underwent echocardiography, exercise stress testing, ambulatory blood pressure monitoring, and vascular testing [29]. Though exercise performance was not different between the groups, elevated nocturnal blood pressure, an indicator of true systemic hypertension, was seen only in the obese group. In addition, obese patients had lower brachial artery reactivity (endothelial dysfunction) compared to their normal-weight peers [29]. Other patient populations with CHD have also recently been studied. In a large cohort of patients with single ventricle heart disease who underwent the Fontan operation (a connection of the inferior and superior vena cava directly to the pulmonary arteries), higher weight was associated with higher ventricular mass-to-volume ratio, a known risk factor for poor outcome in these patients [8]. Obesity in the setting of CHD may have other important short-term implications as well. In a group of children with CHD and cardiomyopathy who were listed for heart transplantation, pre-transplant obesity was found to be a significant risk factor for mortality [19].

Conclusions

The epidemic of obesity has not excluded children and adolescents with CHD; it affects more than one quarter of the population, at least in the USA. Children with CHD are not immune to the traditional risk factors for obesity such as genetic predisposition, sedentary behavior, and poor diet habits. Unique risk factors such as exercise restriction and increased caloric intake in infancy may also play a role in the development of obesity in this population. Obesity is emerging as a potential long-term risk factor for poor outcome in patients with CHD. As more children with CHD are now surviving into adulthood, investigating therapeutic interventions to treat and prevent obesity in this population is of utmost importance. Recommendations for safe physical activity, recreation sport, and exercise training for children with CHD have recently been published [27, 39]. These guidelines may help health care providers to change their practice of exercise restriction. Children who develop strong patterns of good diet and physical activity are likely to continue those habits into adulthood.

References

1. Anderson TJ, Uehata A, Gerhard MD, Meredith IT, Knab S, Delagrange D et al (1995) Close relation of endothelial function in the human coronary and peripheral circulations. J Am Coll Cardiol 26:1235–1241
2. Baker JL, Olsen LW, Sorensen TIA (2007) Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med 357:2329–2337
3. Bar-Mor G, Bar-Tal Y, Krulik T, Zeevi B (2000) Self-efficacy and physical activity in adolescents with trivial, mild, or moderate congenital cardiac malformations. Cardiol Young 10:561–566
4. Berenson GS, Pickoff AS (1995) Preventive cardiology and its potential influence on the early natural history of adult heart diseases: the Bogalusa Heart Study and the Heart Smart Program. Am J Med Sci 310(Suppl 1):S133–S138
5. Berenson GS, Srinivasan SR, Bao W, Newman WP 3rd, Tracy RE, Wattigney WA (1998) Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. N Engl J Med 338:1650–1656
6. Boneva RS, Botto LD, Moore CA, Yang Q, Correa A, Erickson JD (2001) Mortality associated with congenital heart defects in the United States: trends and racial disparities, 1979–1997. Circulation 103:2376–2381
7. Chinali M, de Simone G, Roman MJ, Lee ET, Best LG, Howard BV, Devereux RB (2006) Impact of obesity on cardiac geometry and function in a population of adolescents: the Strong Heart Study. J Am Coll Cardiol 47:2267–2273
8. Cohen MS, Zak V, Atz AM, Printz BF, Pinto N, Lambert L, Pemberton V, Li JS, Margossian R, Dunbar-Masterson C, McCrindle BW, Pediatric Heart Network Investigators (2010) The association of anthropometry with functional health status after the Fontan procedure. Am Heart J 160:1092–1098e1
9. Dent CL, Canter CE, Hirsch R, Balzer DT (2000) Transplant coronary artery disease in pediatric heart transplant recipients. J Heart Lung Transplant 19:240–248
10. Druet C, Stettler N, Sharp S, Simmons RK, Cooper C, Smith GD, Ekelund U, Lévy-Marchal C, Jarvelin MR, Kuh D, Ong KK (2012) Prediction of childhood obesity by infancy weight gain: an individual-level meta-analysis. Paediatr Perinat Epidemiol 26:19–26
11. Engelfriet PM, Drenthen W, Pieper PG, Tijssen JGP, Yap SC, Boersma E, Mulder BJM (2008) Smoking and its effects on mortality in adults with congenital heart disease. Internat J Cardiol 127:93–97
12. Freedman DS, Khan LK, Serdula MK, Dietz WH, Srinivasan SR, Berenson GS (2005) The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. Pediatrics 115(1):22–27
13. Fukasawa R (2010) Long-term prognosis of Kawasaki disease: increased cardiovascular risk? Curr Opin Pediatr 22:587–592
14. Galli-Tsinopoulou A, Kyrgios I, Maggana I, Giannopoulou EZ, Kotanidou EP, Stylianou C, Papadakis E, Korantzis I, Varlamis G (2011) Insulin resistance is associated with at least threefold increased risk for prothrombotic state in severely obese youngsters. Eur J Pediatr 170:879–886
15. Gatzoulis MA (2004) Adult congenital heart disease: a cardiovascular area of growth in urgent need of additional resource allocation. Int J Cardiol 97(suppl 1):1–2
16. Haslam DW, James WP (2005) Obesity. Lancet 366:1197–1209
17. Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LR, Flegal KM (2004) Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. JAMA 291:2847–2850
18. Jatene AD, Fontes VF, Souza LC, Paulista PP, Neto CA, Sousa JE (1982) Anatomic correction of transposition of the great arteries. J Thorac Cardiovasc Surg 83:20–26
19. Kaufman BD, Nagle ML, Levine SR, Vijayanathan N, Hanna BD, Paridon S, Ravishankar C, Chrisant MK (2008) Too fat or too thin? Body habitus assessment in children listed for heart transplant and impact on outcome. J Heart Lung Transplant 27:508–513

20. Kavey RW, Allada V, Daniels SR, Hayman LL, McCrindle BW, Newburger JW, Parekh RS, Steinberger J (2006) Cardiovascular risk reduction in high-risk pediatric patients: a scientific statement from the American Heart Association Expert Panel on Population and Prevention Science; the Councils on Cardiovascular Disease in the Young, Epidemiology and Prevention, Nutrition, Physical Activity and Metabolism, High Blood Pressure Research, Cardiovascular Nursing, and the Kidney in Heart Disease; and the Interdisciplinary Working Group on Quality of Care and Outcomes Research: endorsed by the American Academy of Pediatrics. Circulation 114:2710–2738
21. Lamotte C, Iliescu C, Libersa C, Gottrand F (2011) Increased intima-media thickness of the carotid artery in childhood: a systematic review of observational studies. Eur J Pediatr 170:719–729
22. Legendre A, Losay J, Touchot-Koné A, Serraf A, Belli E, Piot JD, Lambert V, Capderou A, Planche C (2003) Coronary events after arterial switch operation for transposition of the great arteries. Circulation 108:1186–1190
23. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP (1990) Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. N Engl J Med 322:1561–1566
24. Liberthson RR (1996) Sudden death from cardiac causes in children and young adults. N Engl J Med 334:1039–1044
25. Massin MM, Hovels-Gurich HH, Gerard P, Seghaye MC (2006) Physical activity patterns of children after neonatal arterial switch operation. Ann Thorac Surg 81:665–670
26. Massin MM, Hovels-Gurich H, Seghaye MC (2007) Atherosclerosis lifestyle risk factors in children with congenital heart disease. Eur J Cardiovasc Prev Rehabil 14:349–351
27. Moola F, McCrindle BW, Longmuir PE (2009) Physical activity participation in youth with surgically corrected congenital heart disease: devising guidelines so Johnny can participate. Paediatr Child Health 14:167–170
28. O'Leary DH, Polak JF, Kronmai RA, Manolio TA, Burke GL, Wolfson SK (1999) Carotid artery intima and media thickness as a risk factor for myocardial infarction and stroke in older adults. N Engl J Med 340:14–22
29. Pasquali SK, Marino BS, Powell DJ, McBride MG, Paridon SM, Wernovsky G, Meyers KE, Mohler ER, Walker SA, Kren S, Cohen MS (2010) Following the arterial switch operation, obese children have markers of future cardiovascular disease. Congenit Heart Dis 5:16–24
30. Pasquali SK, Marino BS, Pudusseri A, Wernovsky G, Paridon SM, Walker SA, Cohen MS (2009) Risk factors associated with obesity after congenital heart surgery. Amer Heart J 158:473–479
31. Pearson D, Rodriguez E, Fernandes S (2008) Prevalence of obesity in adults with congenital heart disease. In: Program and abstracts of the fifth national conference of the adult congenital heart association; Philadelphia, PA. (abstr)
32. Pemberton VL, McCrindle BW, Barkin S, Daniels SR, Barlow SE, Binns HJ, Cohen MS, Economos C, Faith MS, Gidding SS, Golberg CS, Kavey RE, Longmuir P, Rocchini AP, Van Horn L, Kaltman JR (2012) Report of the National Heart, Lung, and Blood Institute's working group on obesity and other cardiovascular risk factors in congenital heart disease. Circulation 121:1153–1159
33. Pike NA, Evangelista LS, Doering LV, Eastwood JA, Lewis AB, Child JS (2012) Sex and age differences in body-image, self-esteem, and body mass index in adolescents and adults after single-ventricle palliation. Pediatr Cardiol (in press)
34. Pinto NM, Marino BS, Wernovsky G, DeFerrenti SD, Walsh AZ, Laronde M, Hyland K, Dunn SO Jr, Cohen MS (2007) Obesity is a common comorbidity in children with congenital and acquired heart disease. Pediatrics 120:e1157–e1164
35. Ross DN (1967) Replacement of aortic and mitral valves with a pulmonary autograft. Lancet 2:956–958
36. Shustak RJ, McGuire SB, October TW, Phoon CKL, Chun AJL (2012) Prevalence of obesity among patients with congenital and acquired heart disease. Pediatr Cardiol 33:8–14
37. Stefan MA, Hopman WM, Smythe JF (2005) Effect of activity restriction owing to heart disease on obesity. Arch Pediatr Adolesc Med 159:477–481
38. Strauss RS, Pollack HA (2001) Epidemic increase in childhood overweight, 1986–1998. JAMA 286(22):2845–2848
39. Takken T, Giardini A, Reybrouck T, Gewillig M, Hövels-Gürich HH, Longmuir PE, McCrindle BW, Paridon SM, Hager A (2012) Recommendations for physical activity, recreation sport, and exercise training in paediatric patients with congenital heart disease: a report from the Exercise, Basic & Translational Research Section of the European Association of Cardiovascular Prevention and Rehabilitation, the European Congenital Heart and Lung Exercise Group, and the Association for European Paediatric Cardiology. Eur J Cardiovasc Prev Rehabil (in press)
40. Thompson PD, Franklin BA, Balady GJ, Blair SN, Corrado D, Estes NA 3rd, Fulton JE, Gordon NF, Haskell WL, Link MS, Maron BJ, Mittleman MA, Pelliccia A, Wenger NK, Willich SN, Costa F (2007) Exercise and acute cardiovascular events placing the risks into perspective: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism and the Council on Clinical Cardiology. Circulation 115:2358–2368
41. Vieira TCL, Trigo M, Alonso RR, Ribeiro RH, Cardoso MR, Cardoso AC, Cardoso MA (2007) Assessment of food intake in infants between 0 and 24 months with congenital heart disease. Arq Bras Cardiol 88:624–628
42. Williams RG, Pearson GD, Barst RJ, Child JS, del Nido P, Gersony WM, Kuehl KS, Landzberg MJ, Myerson M, Neish SR, Sahn DJ, Verstappen A, Warnes CA, Webb CL (2006) Report of the National Heart, Lung, and Blood Institute Working Group on research in adult congenital heart disease. J Am Coll Cardiol 47:701–707
43. Woo KS, Chook P, Yu CW, Sung RY, Qiao M, Leung SS, Lam CW, Metreweli C, Celermajer DS (2004) Overweight in children is associated with arterial endothelial dysfunction and intima-media thickening. Int J Obes Relat Metab Disord 28:852–857
44. Woo KS, Chook P, Yu CW, Sung RY, Qiao M, Leung SS, Lam CW, Metreweli C, Celermajer DS (2004) Effects of diet and exercise on obesity-related vascular dysfunction in children. Circulation 109:1981–1986
45. Yücel O, Kinik ST, Aka S (2011) Diagnosis of a trend towards obesity in preschool children: a longitudinal study. Eur J Pediatr 170:751–756