

REVIEW

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Obesity and hypertension in children and adolescents

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Abstract

As childhood obesity rates increase worldwide, the prevalence of obesity-related hypertension is also on the rise. Obesity has been identified as a significant risk factor for hypertension in this age group. National Health Surveys and meta-analyses show increasing trends in obesity and pediatric hypertension in obese children. The diagnosis of hypertension in children involves percentiles relative to age, sex, and height, unlike in adults, where absolute values are considered. Elevated blood pressure (BP) in childhood is consistently associated with cardiovascular disease in adulthood, emphasizing the need for early detection and intervention. The pathogenesis of hypertension in obesity involves multiple factors, including increased sympathetic nervous system activity, activation of the renin-angiotensin-aldosterone system (RAAS), and renal compression due to fat accumulation. Obesity disrupts normal RAAS suppression and contributes to impaired pressure natriuresis and sodium retention, which are critical factors in the development of hypertension. Risk factors for hypertension in obesity include degree, duration, and distribution of obesity, patient age, hormonal changes during puberty, high-sodium diet, sedentary lifestyle, and socioeconomic status. Treatment involves lifestyle changes, with weight loss being crucial to lowering BP. Medications such as angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers may be considered first, and surgical approaches may be an option for severe obesity, requiring tailored antihypertensive medications that consider individual pathophysiology to avoid exacerbating insulin resistance and dyslipidemia.

Keywords Hypertension, Pediatric obesity, Children and adolescents, Treatment

Background

Hypertension is one of the most important chronic diseases associated with severe cardiovascular complications. The diagnosis and treatment of hypertension in children and adolescents is essential because it is associated with hypertension and metabolic syndrome in adults. While hypertension in children was previously characterized by secondary hypertension, primary

hypertension is becoming an important cause in many countries with increasing numbers of overweight and obese children. In particular, obesity is a significant risk factor for hypertension in this age group.

The authors aim to explore the prevalence of hypertension, its association with obesity, the mechanisms by which it develops in obese children, and the treatment of hypertension in obese children and adolescents.

Hypertension definition, hypertension trajectory, hypertension prevalence, and trends

Unlike in adults, the diagnosis of hypertension in children and adolescents is defined as being above the 95th percentile of the distribution by sex, age, and height, not the absolute value [1]. In addition, the European Society

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of Hypertension proposes to diagnose hypertension using the same criteria as adults aged 16 years and older [2], and the American Academy of Pediatrics (AAP) suggests diagnosing hypertension using the same criteria as adults in those 13 years and older [3]. In recent years, there has been a movement toward more uncomplicated diagnoses of hypertension, with the Canadian Society of Hypertension adding to the previous definition to define hypertension as >120/80 mmHg in 6- to 11-year-olds and 130/85 mmHg in 12- to 17-year-olds [4]. Japan also reintroduced absolute values for specific age groups (Table 1) [5]. Hypertension is diagnosed when blood pressure (BP) is elevated on three or more visits and can be measured using mercury sphygmomanometers, nonmercury aneroid sphygmomanometers, and oscillating sphygmomanometers, with confirmation recommended by aneroid auscultatory sphygmomanometers [1–4].

Studies have consistently shown that high BP in children contributes to cardiovascular disease in adults [6–9]. In the Atherosclerosis Risk in Healthy Young Adults study, systolic BP (SBP) at a mean age of 13 years was associated with intima-media thickening in adulthood [6]. In the Young Finns Study, which followed 2204 people for 27 years, BP, serum lipid levels, and body mass index (BMI) in childhood were also strongly associated with those in middle age [7]. The Coronary Artery Risk Development in Young Adults Study, which followed 4681 people for 25 years, found that the risk of a coronary artery calcium score of 100 HU or more at 25 years was significantly higher in the group with increased BP than

those with low, controlled BP [8]. A cohort study that followed 975 people aged 7 to 38 also found that those with elevated BP had worse cardiovascular outcomes, demonstrating the importance of early detection, prevention, and intervention [9].

According to the Korea National Health and Nutrition Examination Survey (KNHANES), SBP showed an increasing trend between 2007 and 2015, and this trend was particularly pronounced in obese children. The prevalence of hypertension also increased from 6.9% in 2007–2009 to 9.0% in 2013–2015, with a significant increase from 14.9 to 27.7% in obese children [10]. According to the China Health and Nutrition Survey, BP and the prevalence of hypertension showed an increasing trend between 1991 and 2015, with hypertension increasing from 8.5% in 1991 to 19.2% in 2015 [11]. Factors specifically associated with hypertension included adolescents aged 13–17 years (OR=1.76), general obesity (OR=2.69), and central obesity (OR=1.49). The prevalence of hypertension in children and adolescents aged 12–19 years based on data from the National Health and Nutrition Examination Survey (NHANES) in the United States, on the other hand, decreased somewhat in 2013–2016 compared to 2001–2004, from 7.7 to 4.2% under the 2017 AAP guidelines [12]. However, the prevalence of obesity increased from 17.8 to 21.8% during this period, and the prevalence of hypertension among obese children was 9.43% during 2013–2016, which is higher than the prevalence of hypertension among all children [12]. Meanwhile, a study based on a school-based BP

Table 1 Various definitions of hypertension in children and adolescents

Guideline	SBP and/or DBP Percentile of Category					
TFR [1]	Normal < 90th	Prehypertension 90th to < 95th of if BP exceeds 120/80 even if < 90th percentile up to < 95th percentile	Hypertension ≥ 95th percentile			
ESH [2]	1–15 yr Normal < 90th percentile	High-normal ≥ 90th percentile to < 95th percentile	Hypertension ≥ 95th percentile	≥ 16 years Normal < 130/85 mmHg	High-normal 130–139/85–89 mmHg	Hypertension ≥ 140/90 mmHg
AAP [3]	1–13 yr Normal < 90th percentile	Elevated BP ≥ 90th percentile to < 95th percentile or 12/80 mmHg to < 95th percentile (whichever is lower)	Hypertension ≥ 95th percentile	≥ 13 yr Normal < 120/80 mmHg	Elevated BP 120/<80 to 129/<80 mmHg	Hypertension ≥ 130/80 mmHg
Canadian [4]	Criteria for hypertension					
	6–11 years: ≥ 95th or ≥ 120/80 mmHg (whichever is lower)		12–17 years ≥ 95th or ≥ 130/85 mmHg (whichever is lower)			
Japan [5]	Pre-School ≥ 120/70 mmHg	Elementary 1 ~ 3rd grade ≥ 130/80 mmHg	Elementary 4 ~ 6 grade ≥ 135/80 mmHg	Junior high boys ≥ 140/85 mmHg	Junior high girls ≥ 135/80 mmHg	High school ≥ 140/85 mmHg

AAP American Academy of Pediatrics, BP blood pressure, DBP diastolic blood pressure, ESH, European Society of Heart, SBP systolic blood pressure, TFR The Fourth Report

screening program reported 16.3% elevated BP and 2.7% hypertension among students aged 10–17 years. In particular, 10.6% had stage 1 hypertension at initial screening, but only 2.7% had confirmed hypertension after two additional visits, demonstrating the importance of repeat visits and BP measurements in pediatric hypertension [13]. In the meta-analysis of 47 articles, the global pooled prevalence of hypertension was 4.0% (95% confidence interval (CI), 3.29–4.78%), and that of prehypertension was 9.67% (95% CI, 7.26–12.38%) (Table 2) [14]. In addition, pediatric hypertension has shown an increasing trend over the past two decades in this analysis. However, another systematic review analyzed 18 studies of changes in childhood hypertension between 1963 and 2012 and found that BP gradually decreased in 13 studies, increased in four, and remained unchanged in one [15].

Obesity definition, obesity prevalence and trends

Obesity in children and adolescents is defined by BMI, which is calculated by dividing weight by the square of height. This calculated BMI is then compared to normal for age and gender, with overweight defined as between the 85th percentile and 95th percentile, obesity defined as above the 95th percentile, and severe obesity defined as above 120% of the 95th percentile or above 35 kg/m² [16]. The AAP further defines severe obesity as BMI ≥ 120% to < 140% of the 95th percentile or BMI ≥ 35 to < 40 kg/m² as class 2 obesity and BMI ≥ 140% of the 95th percentile or BMI ≥ 40 kg/m² as class 3 obesity [17].

The World Health Organization reports that globally, 33 million children under the age of 5 years [5.3% (UI: 5.1–5.5)] were classified as overweight in 2000, rising to

37 million [5.6% (UI: 5.1–6.1)] by 2022 [18]. In 2416 population-based studies, from 1975 to 2016, the global age-standardized prevalence of obesity among children and adolescents aged 5 to 19 years in 200 countries increased from 0.7% (95% credible index 0.4–1.2) in 1975 to 5.6% (4.8–6.5) in 2016 for girls and from 0.9% (0.5–1.3) to 7.8% (6.7–9.1) for boys [19]. Trends in BMI change varied by region, flattening somewhat in Northwest Europe and high-income countries but increasing for both sexes in East and South Asia. The number of obese girls grew from 5 million in 1975 to 50 million in 2016, while the number of obese boys grew from 6 million to 74 million during the same period. 73% of this increase is attributable to the rise in obese children [19]. According to the 2017–2018 NHANES in the United States, 19.3% of children aged 2–19 years were obese, 6.1% of whom were severely obese, and 16.1% were overweight. This is a significant increase compared to 1971–1974, when 5.2% were obese [20]. The prevalence of obesity among children and adolescents in Korea is also increasing based on data from the National School Health Examination (NSHE) and the KNHANES, from 8.7% in 2007 to 15.0% in 2017 according to the NSHE data and from 8.6% in 2001 to 9.8% in 2017 according to the KNHANES data. This increase was particularly pronounced among boys and high school students (Table 3) [21].

The relationship between BP and BMI has been shown in several studies [22, 23]. In a study comparing 167 obese adolescents with 31 nonobese youth, SBP was significantly higher, and elevated BP was substantially more common in those with a BMI Z score of 2.5 or greater compared to those with a BMI Z score of less than 2 [22].

Table 2 The prevalence of hypertension in the various studies

Study	Age group (year)	Year	Hypertension prevalence	Hypertension prevalence in obesity	Data source
Cho et al. [10]	10–18	2007–2009	6.9%	14.9%	KNHANES
Ye et al. [11]	7–17	2013–2015	9.0%	27.7%	The China Health and Nutrition Survey
		1991	8.5%		
Jackson et al. [12]*	12–19	2001	7.7%		NHANES
		2016	4.2%	9.43%	
Bell et al. [13] †	10–17	2000–2017	2.7%		The Houston Pediatric and Hypertension Program
Song et al. [14]	-19	1990s	4.0%	15.27%	Meta-analysis
		2000s	1.26%		
		2010–2014	3.30%		
		2010–2014	6.02%		

KNHANES Korea National Health and Nutrition Examination Survey, NHANES National Health and Nutrition Examination Survey

* The prevalence of hypertension defined by the 2017 AAP guideline

† The prevalence of hypertension defined by the Fourth Report

Table 3 The prevalence of obesity in the various studies

Study	Age group (year)	Region	Year	Obesity prevalence	Data source
NCD Risk Factor Collaboration [19]	5–19	Worldwide	1975	0.7% (girls) 0.9% (boys)	A pooled analysis of 2416 population-based measurement studies
			2016	5.6% (girls) 7.8% (boys)	
Fryar et al. [20]	2–19	USA	1971–1974	5.2%	NHANES
			2017–2018	19.3%	
Kim et al. [21]	6–18	Korea	2007	8.7%	NSHE
			2017	15.0%	
	2–18	Korea	2001	8.6%	KNHANES
			2017	9.8%	

KNHANES Korea National Health and Nutrition Examination Survey, NCD non-communicable diseases, NHANES National Health and Nutrition Examination Survey, NSHE National School Health Examination

In another study of BP and BMI associations in adolescents, mean SBP and diastolic BP (DBP) were correlated with increasing BMI deciles, in which an increase in BMI from the 1st to the 10th decile was associated with a 10 mmHg increase in SBP and a 3–4 mmHg increase in DBP [23]. In children, excess weight can be a risk factor for later BP. When 17,816 students were followed for 8.2 years, the risk of hypertension was 1.53 times higher for boys and 1.28 times higher for girls if their BMI remained persistently high compared with those whose BMI remained persistently low [24]. Additionally, several studies have shown a higher prevalence of hypertension in overweight or obese children. In a school-based screening of 5102 children, the prevalence of hypertension was 2% for BMI below the 5th percentile, increasing to 11% for those above the 95th percentile, with overweight being a significant relative risk for hypertension [25]. Data from the NHANES of the United States showed that the prevalence of hypertension according to the 2017 AAP guidelines was 4.2% among all children aged 12–19 years from 2013 to 2016, compared to 9.43% among obese children and 14.7% among severely obese children [12]. In a study of 57,915 overweight and obese children aged 6–18 years from 188 centers in Germany, Austria, and Switzerland, the prevalence of hypertension and prehypertension ranged from 27 to 47%, depending on the hypertension criteria [26].

Pathogenesis of hypertension in obesity

Overweight and obesity affect BP in many ways, and adiposity and weight gain are essential contributors to primary hypertension [27]. The development of hypertension in obesity is influenced by several factors, including increased sympathetic nervous system (SNS) activity, activation of the renin-angiotensin-aldosterone system (RAAS), and compression of the kidneys due to

fat accumulation, resulting in increased renal sodium reabsorption and impaired pressure natriuresis.

Increased SNS is thought to play an essential role as a mechanism for hypertension in obesity. In a study showing that weight gain itself increases SNS, 12 healthy men were made to gain 5 kg of weight by overeating, and when comparing before and after, there was a significant increase in total fat and abdominal fat and a substantial increase in muscle SNS and SBP, suggesting a relationship between obesity and hypertension [28]. In a study of 18 lean and 25 overweight healthy college students, overweight was associated with subclinical alterations in renal and endothelial function in addition to left ventricular wall thickness regardless of hypertension [29]. SNS activity was highly associated with cardiovascular and renal changes in these individuals. In obesity, not all SNSs are increased, but the kidneys and muscles seem to play an important role [30]. The fat distribution also plays an important role, with increased muscle SNS activity observed in individuals with visceral adiposity alone [31]. SNS overactivity is also associated with ethnicity. Native American Pima Indians, who have very high rates of obesity, have a relatively low frequency of hypertension. They have increased adiposity and insulinemia but have lower basal muscle SNS activity than white individuals and a lower frequency of hypertension [32]. In addition, sleep apnea, which is common in obesity, causes chronic hypoxia, which activates chemoreceptors in the carotid body and upregulates SNS activity [33].

Mediators of increased SNS activity include hyperinsulinemia, angiotensin II, impairment of baroreceptor reflexes, activation of chemoreceptor-mediated reflexes associated with sleep apnea, and cytokines such as leptin, tumor necrosis factor- α , and interleukin-6 secreted by adipocytes. Of these, leptin is secreted by adipocytes and is particularly elevated in obesity and hypertension. In one study, normotension was observed in children with

severe obesity and a leptin gene mutation, and in these children, SNS activity was actually decreased [34]. In addition, leptin is selectively resistant in obesity, resulting in a reduced appetite-reducing effect but a preserved SNS activity response in the kidneys [35].

Despite the volume expansion and sodium retention associated with obesity, they do not have a normal RAAS suppressive response. Related factors, such as plasma renin activity, angiotensinogen, angiotensin-converting enzyme (ACE), and aldosterone, are elevated compared to normal subjects [36, 37]. Renin secretion is also upregulated in obese patients under the pressure of increased visceral and retroperitoneal fat. Adipocytes from subcutaneous fat are also an essential source of angiotensin II and can downregulate the RAAS in subcutaneous adipocytes by regulating insulin [38]. In obese patients, RAAS and SNS activity interact to stimulate renin secretion.

In obesity, visceral, retroperitoneal, and renal sinus fat cause renal compression and structural changes in kidney tissue. Physical compression by fat in and around the kidneys impairs pressure natriuresis and increases renal tubular sodium reabsorption, resulting in sodium retention and increased BP [39–41]. Obesity increases the glomerular filtration rate and effective renal plasma flow but ultimately leads to glomerular injury, which increases BP and causes renal damage, creating a deleterious cycle [39, 40].

Risk factors for hypertension in obesity

Obesity is the most critical risk factor for hypertension in children and adolescents [42, 43]. Risk factors other than obesity that are known to contribute to the development of hypertension include male sex, a family history of hypertension, early life factors such as birth weight or gestational age, a high-sodium diet, the absence of a Dietary Approaches to Stop Hypertension (DASH)-type diet, larger amounts of sedentary time, and possibly other dietary factors [3, 44–46]. However, it is not yet clear what risk factors for hypertension are specific to overweight children and adolescents.

Factors related to the characteristics of obesity concerning the development of hypertension include the degree, duration, and distribution of obesity and the patient's age. In a study conducted by Babinska et al. on 109 obese children aged 7–18 years with a BMI Z score above 1.65, only 24% had normal BP on ambulatory BP monitoring, while 3% had hypertension, and 48% had severe ambulatory hypertension [47]. The study also found a proportional association between BMI and the severity of ambulatory hypertension and daytime BP. In adults, abdominal obesity is a significant predictive factor for hypertension. Chen et al. conducted a cross-sectional study on the correlation between the types of obesity and hypertension in adult males in the United States using

NNHANES data from 2007 to 2018 and found that the presence of abdominal obesity was significant in predicting hypertension compared to BMI alone [48]. The association of the waist-to-height ratio (WHtR), one of the measurements of abdominal obesity in children and adolescents, with hypertension varies across studies but is generally significant [49–52]. In a meta-analysis of nine studies involving 25,424 children aged 6 to 18, waist circumference (WC) and WHtR did not show superiority in detecting elevated BP [50]. In another meta-analysis including 21 cross-sectional studies involving 177,943 children aged 3–19, BMI, WC, and WHtR showed no significant difference in predicting hypertension and elevated BP [51]. However, in a cross-sectional study involving Malaysian adolescents between 12 and 16 years, WHtR presented good sensitivity and specificity in males and females [52]. Kułaga et al. investigated abdominal obesity cutoffs associated with adult cardiovascular risk thresholds based on anthropometric data from Polish children aged 3–18 years and found that the determined abdominal obesity cutoff significantly predicted hypertension and elevated BP [53]. The duration of obesity is also crucial in the development of hypertension in obese children. In a cross-sectional study by Li et al., children who had incident high weight or persistently high weight from birth to childhood had higher odds of childhood high BP than those who had persistently normal weight [54].

Generally, male sex has been considered a major risk factor in pediatric hypertension [55]. However, in the studies of overweight children and adolescents, the effect of sex has not been apparent [56, 57]. In a large population-based study, BMI level and age were more significant than sex differences [58]. Other studies also reported that the effect of obesity on BP was greater with age [59, 60]. This may be related to hormonal changes during puberty.

Excessive salt intake has been associated with the development of obesity and high BP [61, 62], and the impact of excessive salt intake on BP is even more significant in obese individuals. A recent meta-analysis revealed that sodium intake was positively associated with BP in children and adolescents, especially more strongly in children with overweight and low potassium intake [63]. Another cross-sectional study in Portugal found that high sodium intake was associated with higher SBP in boys, and this was more pronounced in those who were overweight [64].

The lack of physical activity and sedentary behaviors, such as playing video games and watching television, are related to an increased risk of developing hypertension in overweight adolescents [43, 65, 66].

Recent studies have indicated a potential correlation between socioeconomic status (SES), hypertension, and BMI, particularly in the context of evolving social

economies [67–70]. It is noteworthy that research in adults has revealed a paradox: in developing or developed nations, there is an inverse correlation between SES and hypertension, in contrast to under-developed countries. In Ghana, there was a positive association between SES and hypertension, which was partly due to differences in BMI [67]. Conversely, lower SES has been linked to higher hypertension rates in China and Iran [68, 69]. A study conducted in Hong Kong examined the relationship between SES and childhood obesity and hypertension; the findings revealed that children residing in the lowest SES neighborhoods were more likely to be underweight, overweight, or obese [70]. Additionally, the study indicated that having a less educated mother was associated with a higher risk of obesity and hypertension in children. This indicates the necessity for more sophisticated economic policy solutions to address these health issues.

Treatment of hypertension in obesity

There are two main approaches to treating obesity-related hypertension. The first is to treat obesity and lower the BMI to below the 85th percentile, and the second is to treat BP itself. According to the published guidelines, lifestyle counseling for the DASH diet and moderate to vigorous exercise are recommended for all pediatric patients regardless of hypertension stage (Table 4) [2, 3].

Weight reduction is the key element in managing hypertension among overweight adolescents, considering the robust connection between adiposity and BP. For reducing BMI, diet and physical activity enhancement are essential [71]. In a meta-analysis study regarding non-pharmacological interventions and childhood obesity, combining diet and physical activity interventions was effective in reducing the risk of obesity in young children aged 0 to 5 years, while interventions focusing on physical activity alone were not effective in this age group [72]. However, interventions concentrating only on physical activity can reduce the risk of obesity in children aged 6 to 12 years and adolescents aged 13 to 18 years. There is no evidence that diet-only interventions are effective in these age groups, but interventions combining diet and physical activity may be effective.

In adults, weight loss has been associated with a reduction in BP. A meta-analysis of 18 studies revealed that losing 3–9% of body weight reduced SBP by 3 mmHg and DBP by 3 mmHg [73]. Another meta-analysis, encompassing 25 randomized controlled trials with 34 strata involving 4,874 individuals aged 37 to 66, explored weight reduction's impact on BP. A reduction of 5.1 kg in body weight was associated with a decrease in SBP of 4.44 mmHg and DBP of 3.57 mmHg. Among those who lost more than 5 kg, the reduction in SBP was 6.63 mmHg; in DBP, it was 5.12 mmHg [74]. In obese adults, the greater

Table 4 Lifestyle modifications for pediatric hypertension summarized from references

	2017 AAP guidelines [3]	2016 European Guidelines [2]
General Recommendation	Motivational interviewing may be a useful tool. Reducing stress	Implement of the behavioral change (Physical activity and diet) tailored to individual and family characteristics. Encourage parents/family participation. Encourage a smoke-free environment. Provide educational support and materials. Establish realistic goals. Develop a health-promoting reward system.
Weight reduction	Intensive weight-loss therapy for obese children with hypertension	Weight maintenance or gradual weight loss to achieve value < 85th percentile
Physical activity	Moderate to vigorous physical activity at least 3 to 5 days per week (30–60 min per session)	At least 60 min of activity per day, at least moderate-to-vigorous-intensity physical activity daily More activity provides additional health benefits. Aerobic mostly but with resistance components (3 times/week). Avoid more than 2 h of sedentary behavior per day. If uncontrolled stage 2 hypertension, avoid competitive sports.
Diet	DASH diet Fruits and vegetables 4–5 servings per day Low-fat milk products ≥ 2 servings per day Whole grains 6 servings per day Fish, poultry, and lean red meats ≤ 2 servings per day Legumes and nuts 1 serving per day Oils and fats 2–3 servings per day Added sugar and sweets (including sweetened beverages) ≤ 1 serving per day Dietary sodium < 2300 mg per d	Avoid intake of excess sugar, excess soft-sweetened drinks, saturated fat, and salt. Recommend fruits, vegetables, and grain products Limit sodium intake (< 2300 mg/daily).

the weight loss, the more significant the improvements in cardiovascular health and BP parameters [75, 76].

Several studies have investigated the BP-lowering effect of weight loss in obese or overweight children and adolescents [77–80]. The interventions included components such as diet, physical activity, education, and counseling and demonstrated a significant decrease in BP with weight loss. In a study that followed 5279 obese children for 32 months, both SBP and DBP decreased with lower BMI, and failure to treat obesity increased the risk of developing high BP levels [77]. In another study, a 12-week weight loss intervention in 115 obese children resulted in significant reductions in both SBP and DBP when weight was reduced [78].

Medical treatment for obesity

Drug therapy for the treatment of obesity in children is currently not approved. Furthermore, when considering drug therapy for children, a strong emphasis must be placed on assessing the risk-benefit ratio. Some drugs that have been considered for their potential effectiveness in treating obesity include amphetamines, fenfluramine, and dexfenfluramine.

Orlistat is an intestinal lipase inhibitor that reduces dietary fat and cholesterol absorption by 25%. The American Food and Drug Administration has approved it for obese adolescents over 12 years of age [81].

The obesity treatment drug liraglutide, approved for use in adults, has also been considered for use in pediatric diabetes. Recent results from a systematic review regarding its use for obesity treatment in pediatric populations indicate that it can be relatively safe and effective

in children. In this study, there was a significant reduction in BMI and BMI standard deviation score, but the drop in SBP and DBP was not statistically significant [82]. However, this systematic review included studies of a small number of pediatric obese patients, an unclear method of BP measurement for the study subjects, and an unknown method of determining hypertension. More research is needed to further investigate the relationship between obesity treatment and hypertension.

A surgical approach may be considered in cases of severe obesity, but it is challenging to implement in children. In one prospective study, 242 adolescents aged 19 and under underwent weight-loss surgery, and the researchers discovered significant improvements in weight, cardiometabolic health, and weight-related quality of life three years after the procedure. The remission rate for elevated BP was 74% (95% CI, 64–84%) after three years [83].

Antihypertensive medication

According to the 2017 AAP guidelines, children with persistent or symptomatic hypertension despite attempts at lifestyle modification, stage 2 hypertension without clearly modifiable factors such as obesity, or any stage of hypertension associated with chronic kidney disease or diabetes should be started on a single drug at the low end of the dose range [3]. As is the case with non-obesity-related hypertension, BP should be managed to levels below the 90th percentile or 130/80 for children aged 13 years and older [3]. Drug choice should be targeted to the child’s underlying pathophysiology and the presence of concurrent disorders (Fig. 1).

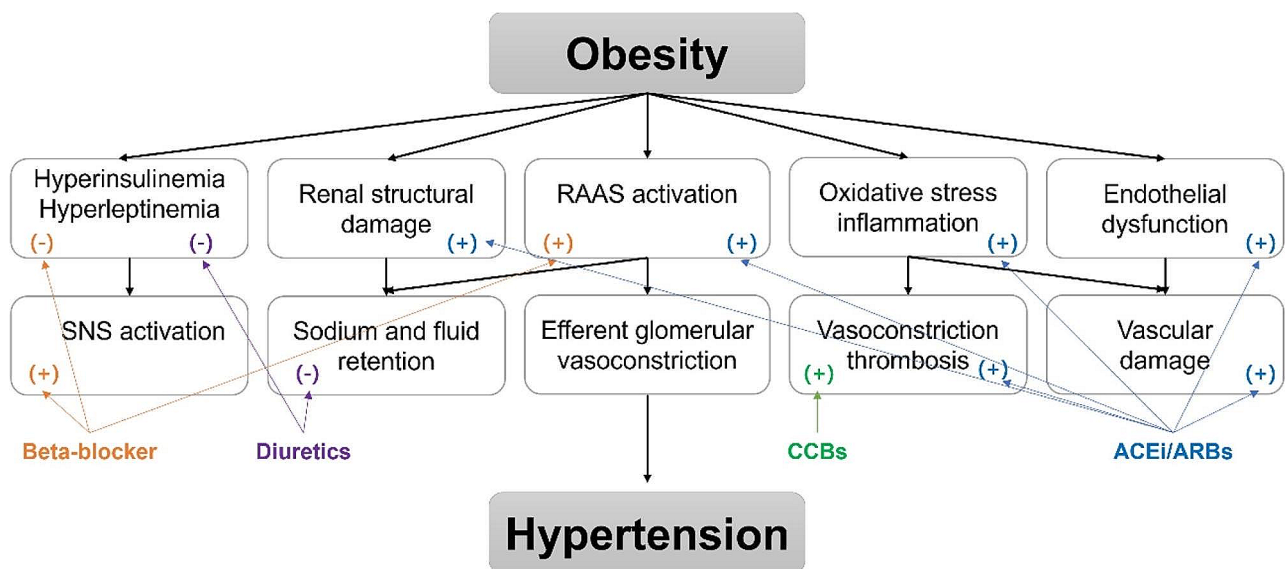


Fig. 1 Mechanisms of hypertension in obesity and targets of antihypertensive drugs. SNS sympathetic nervous system, RAAS renin-angiotensin-aldosterone system, ACEi angiotensin-converting enzyme inhibitor, ARB angiotensin receptor blocker, CCB calcium channel blocker. (+) positive or protective effect, (-), negative effect. Figure adapted from *Hypertension in childhood obesity*, E. Wühl, 2019; adapted with permission; copyright John Wiley and Sons

Since one of the primary pathophysiologies of obesity-related hypertension is through activation of the RAAS system, ACE inhibitors or angiotensin receptor blockers (ARBs) may be appropriate as an initial agent for pharmacologic therapy for hypertension in children. There is some evidence to suggest the use of ACE inhibitors and ARBs as first-line agents in the obesity-linked primary hypertension population; in adults, these agents appear to reduce the incidence of new-onset diabetes and may increase insulin sensitivity [84]. Where these are not tolerated, calcium channel blockers are a reasonable alternative.

Given their known effects on glucose metabolism and insulin resistance, it is sensible to avoid using beta-blockers without vasodilatory capacity and thiazide diuretics [85]. In particular, diuretics can reduce intravascular volume and cardiac output and may stimulate the SNS and RAAS. They also have the potential to exacerbate insulin resistance and dyslipidemia and may increase glucose and uric acid levels, especially in obese individuals [86].

Conclusion

In children, obesity is strongly associated with hypertension, and with the increase in obesity in recent years, hypertension has become an essential health condition in children. Several factors, including SNS and RAAS activity, cause the development of hypertension in obese children. Risk factors for hypertension in obesity include degree, duration, and distribution of obesity, patient age, hormonal changes during puberty, high-sodium diet, sedentary lifestyle, and SES. Treatment in obese children is a combination of treatment for obesity and hypertension. Treatment involves lifestyle changes, with weight loss being crucial to lowering BP.

Abbreviations

AAP	American Academy of Pediatrics
ACE	Angiotensin-converting enzyme
ARB	Angiotensin receptor blocker
BMI	Body mass index
BP	Blood pressure
CI	Confidence interval
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
KNHANES	Korean National Health and Nutrition Examination Survey
NHANES	National Health and Nutrition Examination Survey
NASH	National School Health Examination
RAAS	The renin-angiotensin-aldosterone system
SBP	Systolic blood pressure
SES	Socioeconomic status
SNS	Sympathetic nervous system
WC	Waist circumference
WHtR	Waist-to-height ratio

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