International Journal of Obesity
https://doi.org/10.1038/s41366-019-0384-2

ARTICLE

Pediatrics

The effect of weight loss and weight gain on blood pressure in children and adolescents with obesity

Emilia Hagman[1] · Pernilla Danielsson[1] · Amira Elimam[1] · Claude Marcus[1]

Received: 17 September 2018 / Revised: 4 March 2019 / Accepted: 19 April 2019

Abstract

Objective Obesity in childhood is a profound risk factor for hypertension, and weight loss has positive effects on blood pressure (BP). However, the expected effect size on BP from weight reduction in children with obesity is insufficiently described. Therefore, the aim was to investigate the association between changes of degree of obesity and BP levels.

Subjects This prospective cohort study examined subjects receiving behavioral lifestyle modification treatment who were registered in the Swedish national registry for treatment of childhood obesity (BORIS). A total of 5279 obese subjects (51.3% boys) had repeated BP measurements. The average follow-up time was 32 months. Degree of obesity was expressed as BMI standard deviation score (SDS) and BP as BP SDS.

Results The mean age at treatment initiation was 10.3 years. The prevalence of hypertensive BP was 15.3% for systolic and 5.5% for diastolic pressure. Both systolic and diastolic BP SDS decreased when a lower BMI SDS was achieved; systolic BP SDS decreased 0.41 [0.33–0.49] and diastolic BP SDS decreased 0.26 [0.20–0.32] per BMI SDS unit reduction. The impact of BMI SDS reduction on BP SDS was greater in subjects with hypertensive levels at treatment initiation, but behavioral modification was an insufficient treatment for 27% of them. Obesity treatment failure increased the risk of developing hypertensive levels; HR = 1.81 [1.38–2.37] (systolic BP) HR = 3.82 [2.34–6.24] (diastolic BP), per unit increase in BMI SDS.

Conclusions Weight loss is a key factor for hypertension prevention and treatment in children with obesity. However, its limited effect suggests that additional pharmacological antihypertensive treatment more readily should be considered.

Introduction

Obesity in childhood and adolescence is associated with several metabolic complications [1–3], cardiovascular alterations such as left ventricular hypertrophy [1], and increased intima media thickness [4]. Obesity is currently the strongest risk factor for hypertension in childhood [5, 6], but the prevalence differs considerably between studies [6–11]. Both high blood pressure (BP) levels [5–8, 11–13] and disturbed diurnal variation [12, 13] have been reported in children and adolescents with obesity.

The risks associated with hypertension in adulthood are well documented [14], but even in children high BP is associated with negative consequences. End organ injury, such as left ventricular hypertrophy [15], and pathological vascular changes, such as increased intima media thickness (IMT) [16], has been observed in children with obesity and high BP. In addition, neurological consequences, manifested as reduced cognitive function, have been identified among children with high BP [17]. Even though the tracking of BP from childhood into adulthood appears limited [18], population-based studies reveal that both obesity and high BP in childhood increase the risk for adult cardiovascular changes, such as increased IMT [19, 20], which in turn is associated with hard endpoints such as stroke and heart failure [21].

Behavioral modification to decrease the degree of obesity is an established treatment approach to reduce high BP in individuals with obesity [22]. Although data on BP

Supplementary information The online version of this article (https://doi.org/10.1038/s41366-019-0384-2) contains supplementary material, which is available to authorized users.

✉ Emilia Hagman
emilia.hagman@ki.se

[1] Division of Pediatrics, Department of Clinical Science, Intervention and Technology, Karolinska Institutet, Blickagången 6A, 141 57 Stockholm, Sweden

Published online: 31 May 2019

reduction from randomized intervention trials for reducing weight are limited [15], a decrease in degree of obesity over 6 to 12 months has been shown to lower BP in children and adolescents with overweight and obesity [9, 23–25]. The effectiveness of weight loss on BP levels in adolescents has also been demonstrated after gastric bypass surgery [26]. However, thus far no detailed data are available on the expected effect size on BP of reduction in degree of obesity in children and adolescents in large-scale and long-term behavioral studies.

The aim of this study was to investigate the extent to which a change in degree of obesity affects BP levels in children and adolescents.

Methods

Subjects

This cohort consisted of individuals included in the Swedish national registry for treatment of childhood obesity—BORIS (www.e-boris.se)—from December 1994 until June 2016. Local health care providers are required by Swedish health authorities to enter children undergoing obesity treatment into BORIS. Pediatric obesity treatment is intended to be long-term, comprising diet and physical activity, as well as lifestyle modification. The treatment of pediatric obesity in children in Sweden has been described previously [27, 28]. In Sweden, all health care is free of charge for children and adolescents under 18 years of age, which reduces the risk for selection bias.

Inclusion criteria were as follows: all children first registered in BORIS between 5 and 18 years of age who were diagnosed with obesity according to International Obesity Task Force [29] and who had a registered BP measurement at treatment initiation and a follow-up (at least one month after treatment initiation). We excluded participants with genetic syndromes (Laurence-Moon-Bardet-Biedls [LMBB], Prader-Willis [PWS], Mb. Down, Klinefelter, Fragile-X), medications (psychostimulants used for ADHD, Sibutramine, and antihypertensive drugs) that possibly could affect blood pressure and participant with height-for-age below −3.5 or above +3.5 standard deviation scores (SDS) to avoid including biologically implausible values.

The study was approved by the regional ethical committee in Stockholm, Sweden; number 2016/922-31/1.

Definitions

To compare BP levels in children, a sex, age, and height-adjusted reference from the National High Blood Pressure Education Program (NHBPEP) Working Group was applied [22]. The NHBPEP is based on pooled data of the first BP measurement values in several pediatric cohort studies [22] and estimates neither the highest nor the lowest prevalence when different references for pediatric BP have been evaluated in parallel [11]. High BP levels were defined at the 95th percentile or above, and BP SDS was used as a continuous variable. Stage II hypertensive levels were defined as BP levels that are higher than 5 mmHg above the 99th percentile [22]. National guidelines on how to measure blood pressure in children are provided by the Swedish Paediatric Society. The first and last registered measurement of BP were used to investigate the effect of reduction in degree of obesity.

To evaluate degree of obesity an international age-dependent and sex-dependent BMI SDS was used [29]. To investigate whether migration background was associated with the outcome, the subjects were divided into two groups based on both the subjects' and their parents' countries of birth: Scandinavian—subjects born in Scandinavian countries (Sweden, Finland, Denmark, Norway, and Iceland) with one or two parents born in Scandinavia; and Non-Scandinavian—subjects born outside Scandinavia or born in Scandinavia with two parents born outside Scandinavia. These data were retrieved from Statistics Sweden (a governmental agency compiling demographic data). Data regarding genetic syndromes and medical drug usage during childhood obesity treatment was retrieved from the national patient registry and the national medical drug registry, respectively.

Statistical analyses

Descriptive statistics are presented as mean ± SD, and differences between sexes were determined using the *t*-test or chi-square test. A random effects mixed model approach (proc mixed) was used to investigate the impact of the degree of treatment success (i.e., change in BMI SDS). Estimation method used was the restricted maximum likelihood (REML) approach. For covariance structure and estimation of degrees of freedom, variance components and containment method was used, respectively. This examined changes in BP SDS, adjusted for time to follow-up, BP SDS at treatment initiation, sex, age, BMI SDS, and migration background. Mixed model analysis corrects standard errors and *p*-values to account for repeat measures. The choice of using a linear model approach was supported by investigating the effect of change in BMI SDS on the main outcome (change in BP SDS) using quantile regression. The change in BMI SDS had a somewhat higher estimate at the higher quantiles of change in systolic BP. The linear regression approach was still considered suitable, since

most intervals of change in BP SDS for systolic and all intervals for diastolic generates fairly similar estimates by change in BMI SDS (data not shown).

The predictive value of change in BMI SDS for developing hypertensive BP levels during treatment was analyzed using Cox proportional hazard regression (proc phreg). Covariates in these analyses included sex, migration background, age, and BMI SDS at treatment initiation. When illustrating this in a figure, change in BMI SDS was arbitrarily divided into four groups. SAS Statistical software (version 9.4) was used for all analyses, and a p-value < 0.01 was considered to be statistically significant.

Results

In total, 5279 out of 13,273 children and adolescents were included in the analyses (Fig. 1). Out of the 11 subjects excluded due to hypertensive medication, only two did not receive potential BP elevating medications (sibutramine or psychostimulants). The mean ± SD age was 10.3 ± 3.1 years, 51.3% were boys, and 34.8% had a migration background. Boys had a slightly greater degree of obesity compared with girls, 2.97 vs. 2.87 BMI SDS ($p < 0.0001$), and were 3.6 months older ($p = 0.0004$), indicating that boys have a tendency to attend the obesity treatment facilities at an older age and with more severe obesity.

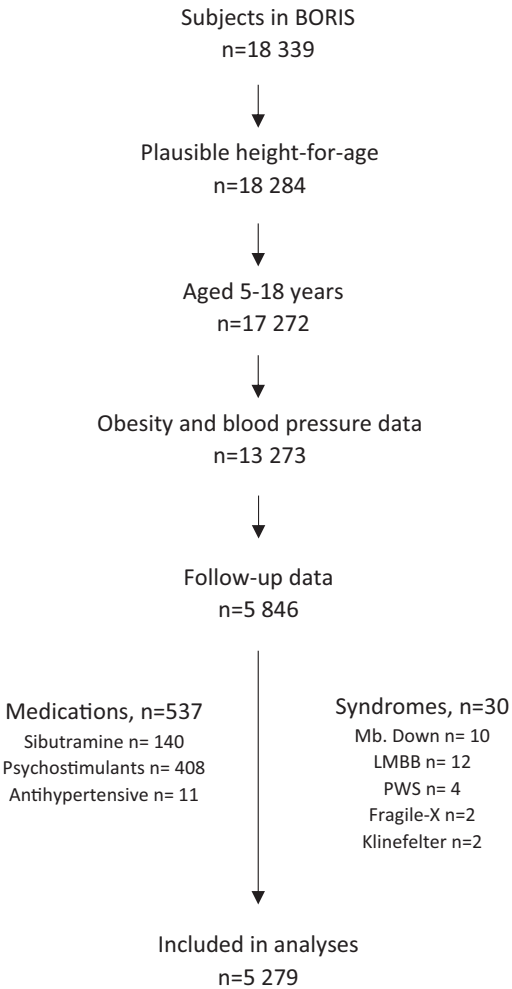

Fig. 1 Flowchart of included patients. Subjects with a height-for-age Z-score below −3.5 or above +3.5 were excluded. Obesity was defined according to International Obesity Task Force[28]

The group without reported follow-up BP measurements or excluded due to criteria ($n = 7\ 994$) had a 0.03 lower BMI SDS ($p < 0.0001$) and were 0.94 years older ($p < 0.0001$), but had a similar sex distribution ($p = 0.03$) and migration background ($p = 0.09$). Further, the group without reported follow-up BP measurements had a 0.08 higher systolic BP SDS ($p < 0.0001$), but similar diastolic BP SDS ($p = 0.80$), and similar proportions with systolic ($p = 0.44$) and diastolic ($p = 0.20$) hypertensive BP levels.

The number of follow-up visits with a recorded BP ranged from 1 to 11, with a mean of 3.0 ± 1.4, corresponding to 32.1 ± 22.5 months of follow-up, and a mean change in BMI SDS of −0.24 ± 0.48 from the first to the last measurement. The prevalence of hypertensive BP levels at treatment initiation was 18.1% and, more specifically, 15.3% for systolic pressure and 5.5% for diastolic pressure. Only 2.7% had both systolic and diastolic hypertensive levels. In subjects with systolic hypertensive levels, the mean systolic BP SDS was 2.35 ± 0.78, and in subjects with diastolic hypertensive BP levels the mean diastolic BP SDS was 2.05 ± 0.36. Stage II hypertensive levels were present in 167 (3.2%) of the subjects. Subjects' characteristics are shown in Table 1. Over the investigated years systolic BP remained at the same level (crude $p = 0.39$, adjusted for sex, BMI SDS, and age $p = 0.41$), while diastolic BP SDS had a minor, but yet statistically significant decrease, of 0.00005 BP SDS per year, crude and adjusted $p < 0.0001$. This is illustrated in Supplementary Fig. 1.

Risk factors for hypertension

Factors increasing the risk for hypertensive BP levels are shown in Table 2. In analyses adjusted for age, sex, and migration background, the odds ratio (OR) for hypertensive levels increased with higher degree of obesity: systolic hypertension OR = 1.74 [99% CL: 1.42–2.14] and diastolic hypertension OR = 2.68 [1.98–3.62] per unit BMI SDS. Girls had a greater risk of both systolic and diastolic hypertensive levels (OR = 1.24 [1.02–1.51] and 1.73 [1.25–2.38], respectively) compared with boys. Increasing age was associated with a minor, but statistically significant, increase in risk of hypertension. Migration background did not affect the risk of hypertensive BP levels.

Table 1 Patient characteristics at initial visit, $n = 5279$

	Proportion or mean ± SD for all	Girls ($n = 2572$) 48.7%	Boys ($n = 2707$) 51.3%	p between girls and boys
Age (years)	10.3 ± 3.1	10.2 ± 3.1	10.5 ± 3.0	0.0004
BMI SDS	2.92 ± 0.47	2.87 ± 0.46	2.97 ± 0.47	<0.0001
Scandinavian/Migration background (%)	65.2 / 34.8	67.1 / 32.9	63.3 / 36.7	0.0039
Systolic bp (mmHg)	111.9 ± 12.6	110.9 ± 12.5	112.8 ± 12.7	<0.0001
Diastolic bp (mmHg)	66.5 ± 9.0	66.5 ± 9.0	66.5 ± 9.0	0.9417
Systolic blood pressure SDS	0.61 ± 1.1	0.64 ± 1.1	0.58 ± 1.0	0.0433
Diastolic blood pressure SDS	0.40 ± 0.76	0.45 ± 0.78	0.36 ± 0.74	<0.0001
Hypertensive systolic BP (% ≥ 95prc)	15.3	16.2	14.4	0.0684
Hypertensive diastolic BP (% ≥ 95prc)	5.5	6.6	4.5	0.0005
Stage II hypertensive systolic BP (% ≥ 99prc + 5 mmHg)	3.1	3.2	2.9	0.5688
Stage II hypertensive diastolic BP (% ≥ 99prc + 5 mmHg)	0.2	0.2	0.3	0.2857
Height *Z*-score	1.06 ± 0.98	0.99 ± 0.99	1.13 ± 0.97	<0.0001

SDS standard deviation score, *BP* blood pressure, *prc* percentile

Table 2 Odds ratios (OR) and 99% confidence limits (CL) for systolic and diastolic hypertension, respectively

	Systolic hypertension			Diastolic hypertension		
	OR	99% CL	p	OR	99% CL	p
Girls vs. boys	1.24	1.02–1.51	0.0057	1.73	1.25–2.38	<0.0001
Age (years)	1.04	1.01–1.08	0.0016	1.07	1.02–1.13	0.0002
BMI SDS	1.74	1.42–2.14	<0.0001	2.68	1.98–3.62	<0.0001
Migration background	1.11	0.91–1.37	0.1844	0.80	0.57–1.12	0.0875

Analyses are adjusted for sex, age, BMI SDS, and migration background. $n = 5279$

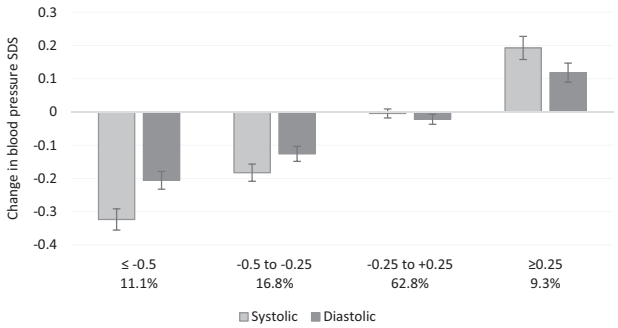

Fig. 2 Effect of behavioral obesity treatment outcome on change in blood pressure SDS, categorized by change in BMI SDS. Bars represent mean ± SE adjusted for age, sex, migration background, treatment duration, and baseline blood pressure SDS

Obesity treatment outcome

We investigated the effect of obesity treatment outcome, i.e., change in BMI SDS, on systolic and diastolic BP SDS values (based on 15,997 BP measurements), respectively, using a mixed model approach, adjusted for time to follow-up, sex, age, BP SDS at treatment initiation, and migration background.

Both systolic and diastolic BP SDS were lower when a lower BMI SDS was achieved. The greatest effect was seen upon systolic BP SDS, where a decrease in BP SDS of 0.41 [0.33–0.49; $p < 0.0001$] per BMI SDS unit decrease, was observed. The corresponding decrease for diastolic BP SDS was 0.26 [0.20–0.32; $p < 0.0001$] per BMI SDS unit decrease. This is illustrated in Fig. 2.

Stratification for sex revealed that the impact of reduction in degree of obesity on BP SDS was greater in boys. The decrease in systolic BP SDS in boys was 0.44 [0.33–0.55; $p < 0.0001$], for each BMI SDS unit decrease, compared with 0.37 [0.25–0.49; $p < 0.0001$] in girls. The corresponding decrease in diastolic BP SDS was 0.29 [0.21–0.37; $p < 0.0001$] in boys, and 0.22 [0.13–0.31; $p < 0.0001$] in girls, for each unit decrease in BMI SDS.

Sub-analyses examining subjects with hypertensive BP levels at obesity treatment initiation revealed that a reduction in degree of obesity had an even greater impact in this group. In subjects with initially hypertensive systolic BP levels with follow-up measurements ($n = 807$), each unit decrease in BMI SDS resulted in a reduction of systolic BP SDS of 0.66 [0.41–0.92; $p < 0.0001$], with a slightly higher estimate in boys of 0.69 [0.33–1.06; $p < 0.0001$], than in girls 0.62 [0.27–0.98; $p < 0.0001$]. The corresponding numbers in subjects with diastolic hypertensive levels ($n = 292$) were a 0.41 [0.09–0.73; $p = 0.0011$] decrease for each

Table 3 Hazard ratios (HR) and 99% confidence limits (CL) for developing systolic or diastolic hypertensive levels, respectively, in subjects with normal pressure at start of treatment

	Systolic hypertension ($n = 4472$)			Diastolic hypertension ($n = 4987$)		
	HR	99% CL	p	HR	99% CL	p
Girls vs. boys	1.08	0.84–1.40	0.43	1.59	1.06–2.39	0.0032
Migration background	1.19	0.91–1.54	0.09	1.36	0.92–2.06	0.04
Age at start of treatment (years)	1.08	1.03–1.14	0.0002	1.07	1.00–1.14	0.0083
Blood pressure SDS at start of treatment	1.53	1.28–1.83	<0.0001	2.12	1.52–2.94	<.0001
BMI SDS at start of treatment	1.35	1.00–1.82	0.011	2.74	1.74–4.32	<.0001
Δ BMI SDS (1 unit increase)	1.81	1.38–2.37	<0.0001	3.82	2.34–6.24	<.0001

Analyses are adjusted for sex, age, blood pressure SDS and BMI SDS at baseline, migration background, and change in degree of obesity (BMI SDS)

Analyses are based on 412 new cases of systolic hypertensive levels and 170 new cases of diastolic hypertensive levels

BMI SDS unit reduction; boys: 0.49 [−0.02–1.01; $p = 0.01$]; girls: 0.37 [−0.04–0.79; $p = 0.02$]. However, behavioral treatment failed to lower BP in 27% of those with hypertensive BP levels at treatment initiation, corresponding to 29.4% of those with systolic hypertensive levels and 14.0% of those with diastolic hypertensive levels.

In subjects with normal blood pressure at treatment initiation, 412 new cases (9.2%) with systolic and 170 new cases (3.4%) with diastolic hypertensive BP levels occurred. They arose among all weight trajectories, but was more frequent among individuals with increased BMI SDS. In adjusted analyses the hazard ratios (HR) [99% CL] for developing systolic and diastolic hypertensive BP levels, respectively, were 1.81 [1.38–2.37; $p < 0.0001$] and 3.82 [2.34–6.24; $p < 0.0001$] for each BMI SDS unit increase (Table 3). In boys the adjusted HRs per BMI SDS unit increase were 1.71 [1.19–2.44; $p = 0.0001$] and 4.91 [4.37–10.17; $p < 0.0001$] for systolic and diastolic hypertensive BP levels, respectively. In girls the adjusted HRs were 1.97 [1.30–2.97; $p < 0.0001$] and 3.04 [1.57–5.92; $p < 0.0001$], respectively. Other risk factors for developing systolic and diastolic hypertensive BP levels were increasing age and BMI SDS at start of obesity treatment, and girls were at greater risk than boys for developing diastolic hypertensive levels.

Discussion

This study, based on a large nationwide cohort of children and adolescents with obesity, shows the effect size of BP reduction according to the decrease in degree of obesity. The effect of obesity treatment on BP was greatest in subjects with hypertensive BP levels at treatment initiation. Even though most subjects with hypertensive levels at baseline normalized their BP during treatment, more than one quarter did not achieve a lowered BP, and several cases of new hypertensive BP levels occurred.

A higher degree of obesity was associated with an increased risk of high BP levels, which is in accordance with previously published data [5, 8, 11]. In the present study, BP within the range of hypertension was more common among girls. This is somewhat surprising since previous studies have shown the opposite [7, 11], but sex differences might be affected by the BP reference used [11].

A decrease in degree of obesity was associated with decreasing systolic and diastolic BP levels which has been demonstrated previously [9, 23–25], and the mean reduction in BP levels was greater for the systolic pressure than the diastolic. As an example, an 11-year-old girl could on average expect a 4 mmHg decrease in systolic BP and a 2 mmHg decrease in diastolic BP if her degree of obesity was reduced by one BMI SDS unit. The effect of weight loss on BP levels was more pronounced among children and adolescents with high BP levels at treatment initiation, corresponding to a 7 mmHg decrease in systolic and 4.5 mmHg decrease in diastolic BP for an 8-year-old boy with hypertensive BP levels.

A recent meta-analyses including 3807 children that received various forms of obesity treatment (diet and physical activity, drugs or surgery) demonstrated that a decrease in BMI resulted in a decrease in systolic but not in diastolic BP [30]. This effect was much lower than previously described by Reinehr et al. In 1388 overweight and obese children with hypertension, a reduction of 0.25–0.5 BMI SDS over 1 year generated a decrease of 16 mmHg in systolic and 10 mmHg in diastolic BP [23]. The effect of weight loss was 2–3 times higher than in the present study and the prevalence of hypertensive BP levels at start was also much higher than in our study (28 vs. 18%). The reasons for the large differences between the studies are unclear but differences in case mix, BP measurement procedure and white coat hypertension may contribute.

In the group with normal blood pressure at start of obesity treatment, several cases of new hypertensive levels occurred during treatment. The change in degree of obesity (BMI SDS) was a strong predictor of new onset

hypertensive BP levels. However, regardless of treatment effect, degree of obesity at treatment initiation affected the risk of developing hypertensive levels, emphasizing the importance of continuing to evaluate blood pressure throughout childhood obesity treatment.

The guidelines for managing hypertension in children and adolescents with obesity places a strong emphasis upon lifestyle modifications as the first line treatment, with pharmacological approaches clearly considered as secondary measures [5, 15, 22]. Our results confirm that lifestyle modification treatment can result in a satisfactory lowering of BP in hypertensive subjects to a large extent. However, in the clinical setting, we could identify a substantial proportion (27%) of children where lifestyle modification was insufficient to lower hypertensive BP levels, indicating that antihypertensive pharmacological treatment should have been initiated in addition to the lifestyle intervention in accordance to present guidelines [15, 22]. This equals to ~5% of all children in obesity treatment, but none of the subjects included in the present study had received BP-lowering medication despite that BP levels remained high or were within the range of severe hypertension. In Germany it has also been reported that children with obesity and hypertension are rarely treated pharmacologically [9], whereas normal-weight children with secondary hypertension regularly receive pharmacological treatment in accordance with recommendations [15, 22]. The reason is probably that many clinicians are reluctant to use pharmacological treatment when it seems obvious that weight loss should be sufficient. However, hypertension is probably more harmful in combination with other metabolic comorbidity markers, more often seen in childhood obesity, than in isolation, which is normally the case in normal weight children with idiopathic hypertension [19, 20].

Strengths and limitations

The present study is based on a large national cohort of children and adolescents with obesity followed over time at clinics specialized in pediatrics. This enabled us to study the outcome of conventional clinical behavioral obesity treatment on BP.

There are also limitations. First, the BP measurements were not standardized as in clinical studies. Thus, we cannot exclude the potential for overestimation of some BP levels, due to incorrect cuffs, insufficient rest, or stressful conditions. Second, there is a risk that BP levels may have been more frequently documented when high, potentially resulting in an overestimation of the prevalence of hypertensive levels. However, 85% of the total cohort had BP reported, which reduces the potential size of such an overestimation. Third, treatment approach was not standardized as regard to dietary advice, physical activity intervention, as well as individualized and group treatment sessions. Not only weight loss but also other lifestyle changes such as increased physical activity and intake of healthy food may have positive effects on BP even in the absence of any substantial effect on weight. Such factors, difficult to objectively verify, may contribute to the variation in the effect of treatment on BP.

It is also important to emphasize that the BP levels we identified as high in the present study, although within the range of hypertension, are not sufficient for the diagnosis of hypertension. Thorough investigation, including 24-h ambulatory measurements, assessment of other comorbidities, and family history of cardiovascular disease are recommended before the decision to start pharmacological treatment [15].

Conclusion

In this prospective nationwide cohort study hypertensive blood pressure levels were prevalent across all age groups in pediatric obesity. Successful behavioral obesity treatment was associated with reduction of blood pressure, and treatment failure with increased risk for hypertensive BP levels. This confirms that weight loss is both a key factor and a first line of treatment of hypertension in children with obesity. However, the reduction of the degree of obesity obtained in Sweden is frequently insufficient to normalize BP levels and, to the best of our knowledge, behavioral obesity treatment results are not better in other countries [31, 32]. Intensified obesity treatment and/or additional pharmacological antihypertensive treatment may therefore be considered more frequently for children and adolescents with obesity and hypertension.

Acknowledgements The authors would like to thank all Swedish pediatric departments and staff working with childhood obesity health care and the BORIS steering committee for establishing and maintaining the registry. We would also like to thank Ulf Hammar (Karolinska Institutet, Sweden) for statistical advice.

Funding This research has been financially supported by the Swedish Society for Medical Research, the Swedish Heart and Lung Foundation (grant no 20150790), the Foundation of Fredrik and Ingrid Thuring (grant no 2017–00309), the Swedish Childhood Diabetes Foundation, and Stockholm County Council (ALF project, grant no 20160548).

Author contributions AE, PD, and CM made suggestions on appropriate analytic approach and helped interpret the results, improved the initial drafts of the manuscript, and contributed to the manuscript's development. EH performed the register linkage, conceived the study, did the analyses and drafted the initial manuscript. All authors reviewed and approved the final version of the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Publisher's note: Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

References

1. Daniels SR. Complications of obesity in children and adolescents. Int J Obes. 2009;33:S60–5.
2. Hagman E, Reinehr T, Kowalski J, Ekbom A, Marcus C, Holl RW. Impaired fasting glucose prevalence in two nationwide cohorts of obese children and adolescents. Int J Obes. 2014;38:40–5.
3. Zabarsky G, Beek C, Hagman E, Pierpont B, Caprio S, Weiss R. Impact of severe obesity on cardiovascular risk factors in youth. J Pediatrics. 2018;192:105–14.
4. Reinehr T, Kiess W, de Sousa G, Stoffel-Wagner B, Wunsch R. Intima media thickness in childhood obesity: relations to inflammatory marker, glucose metabolism, and blood pressure. Metabolism Clin Experiment. 2006;55:113–8.
5. Sorof J, Daniels S. Obesity hypertension in children: a problem of epidemic proportions. Hypertension. 2002;40:441–7.
6. Flynn J. The changing face of pediatric hypertension in the era of the childhood obesity epidemic. Pediatr Nephrol. 2013;28:1059–66.
7. Cao ZQ, Zhu L, Zhang T, Wu L, Wang Y. Blood pressure and obesity among adolescents: a school-based population study in China. Am J Hypertens. 2012;25:576–82.
8. I'Allemand D, Wiegand S, Reinehr T, Muller J, Wabitsch M, Widhalm K, et al. Cardiovascular risk in 26,008 European overweight children as established by a multicenter database. Obesity. 2008;16:1672–9.
9. Reinehr T, Wiegand S, Siegfried W, Keller KM, Widhalm K, l'Allemand D, et al. Comorbidities in overweight children and adolescents: do we treat them effectively? Int J Obes. 2013;37:493–9.
10. Wirix AJ, Nauta J, Groothoff JW, Rabelink TJ, HiraSing RA, Chinapaw MJ. Is the prevalence of hypertension in overweight children overestimated?. Arch Dis Child. 2016;101:998–1003.
11. Flechtner-Mors M, Neuhauser H, Reinehr T, Roost HP, Wiegand S, Siegfried W, et al. Blood pressure in 57,915 pediatric patients who are overweight or obese based on five reference systems. Am J Cardiol. 2015;115:1587–94.
12. Westerstahl M, Hedvall Kallerman P, Hagman E, Ek AE, Rossner SM, Marcus C. Nocturnal blood pressure non-dipping is prevalent in severely obese, prepubertal and early pubertal children. Acta Paediatr. 2014;103:225–30.
13. Westerstahl M, Marcus C. Association between nocturnal blood pressure dipping and insulin metabolism in obese adolescents. Int J Obes. 2010;34:472–7.
14. Drukteinis JS, Roman MJ, Fabsitz RR, Lee ET, Best LG, Russell M, et al. Cardiac and systemic hemodynamic characteristics of hypertension and prehypertension in adolescents and young adults: the Strong Heart Study. Circulation. 2007;115:221–7.
15. Lurbe E, Cifkova R, Cruickshank JK, Dillon MJ, Ferreira I, Invitti C, et al. Management of high blood pressure in children and adolescents: recommendations of the European Society of Hypertension. J Hypertens. 2009;27:1719–42.
16. Zhu W, Huang X, He J, Li M, Neubauer H. Arterial intima-media thickening and endothelial dysfunction in obese Chinese children. Eur J Pediatr. 2005;164:337–44.
17. Lande MB, Adams H, Falkner B, Waldstein SR, Schwartz GJ, Szilagyi PG, et al. Parental assessments of internalizing and externalizing behavior and executive function in children with primary hypertension. J Pediatr. 2009;154:207–12.
18. Juhola J, Magnussen CG, Viikari JS, Kahonen M, Hutri-Kahonen N, Jula A, et al. Tracking of serum lipid levels, blood pressure, and body mass index from childhood to adulthood: the Cardiovascular Risk in Young Finns Study. J Pediatr. 2011;159:584–90.
19. Li S, Chen W, Srinivasan SR, Bond MG, Tang R, Urbina EM, et al. Childhood cardiovascular risk factors and carotid vascular changes in adulthood: the Bogalusa Heart Study. JAMA. 2003;290:2271–6.
20. Raitakari OT, Juonala M, Kahonen M, Taittonen L, Laitinen T, Maki-Torkko N, et al. Cardiovascular risk factors in childhood and carotid artery intima-media thickness in adulthood: the Cardiovascular Risk in Young Finns Study. JAMA. 2003;290:2277–83.
21. Effoe VS, Rodriguez CJ, Wagenknecht LE, Evans GW, Chang PP, Mirabelli MC, et al. Carotid intima-media thickness is associated with incident heart failure among middle-aged whites and blacks: the Atherosclerosis Risk in Communities study. J Am Heart Assoc. 2014;3:e000797.
22. National High Blood Pressure Education Program Working Group on High Blood Pressure in C, Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. Pediatrics. 2004;114:555–76. 2 Suppl 4th Report.
23. Reinehr T, Lass N, Toschke C, Rothermel J, Lanzinger S, Holl RW. Which Amount of BMI-SDS Reduction Is Necessary to Improve Cardiovascular Risk Factors in Overweight Children? J Clin Endocrinol Metabolism. 2016;101:3171–9.
24. Reinehr T, Schaefer A, Winkel K, Finne E, Toschke AM, Kolip P. An effective lifestyle intervention in overweight children: findings from a randomized controlled trial on "Obeldicks light". Clin Nutr. 2010;29:331–6.
25. Reinehr T, de Sousa G, Toschke AM, Andler W. Long-term follow-up of cardiovascular disease risk factors in children after an obesity intervention. Am J Clin Nutr. 2006;84:490–6.
26. Olbers T, Gronowitz E, Werling M, Marlid S, Flodmark CE, Peltonen M, et al. Two-year outcome of laparoscopic Roux-en-Y gastric bypass in adolescents with severe obesity: results from a Swedish Nationwide Study (AMOS). Int J Obes. 2012;36:1388–95.
27. Danielsson P, Kowalski J, Ekblom O, Marcus C. Response of severely obese children and adolescents to behavioral treatment. Arch Pediatr Adoles Med. 2012;166:1103–8.
28. Danielsson P, Bohlin A, Bendito A, Svensson A, Klaesson S. Five-year outpatient programme that provided children with continuous behavioural obesity treatment enjoyed high success rate. Acta Paediatr. 2016;105:1181–90.
29. Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. Pediatr Obes. 2012;7:284–94.
30. Rajjo T, Almasri J, Al Nofal A, Farah W, Alsawas M, Ahmed AT, et al. The association of weight loss and cardiometabolic outcomes in obese children: systematic review and meta-regression. J Clin Endocrinol Metabolism. 2017;102:758–62.
31. Golan M, Kaufman V, Shahar DR. Childhood obesity treatment: targeting parents exclusively v. parents and children. Br J Nutr. 2006;95:1008–15.
32. Savoye M, Shaw M, Dziura J, Tamborlane WV, Rose P, Guandalini C, et al. Effects of a weight management program on body composition and metabolic parameters in overweight children: a randomized controlled trial. JAMA. 2007;297:2697–704.