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KIDNEY RESEARCH AND CLINICAL PRACTICE

Clinical guidelines for the diagnosis, evaluation, and management of hypertension for Korean children and adolescents: the Korean Working Group of Pediatric Hypertension

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Pediatric hypertension (HTN) is a significant, growing health concern worldwide and also in Korea. Diagnosis, evaluation, and treatment of HTN in Korean children and adolescents are uncertain due to limitations in using the current international guidelines, since the recommendations by the American Academy of Pediatrics (AAP) and European Society of Hypertension (ESH) guidelines differ. Furthermore, these are guidelines for Western youth, who are racially and ethnically different from Koreans. In addition, reference blood pressure values for all pediatric age groups, which are essential for the diagnosis of HTN according to these two guidelines, are absent in Korea. Therefore, HTN guidelines for Korean children and adolescents should be established. The Korean Working Group of Pediatric Hypertension established clinical guidelines for the diagnosis, evaluation, and management of HTN in Korean children and adolescents. These guidelines were based on reported clinical evidence, expert recommendations, and AAP and ESH guidelines. The characteristics of Korean youth and the Korean medical and insurance system were considered during the establishment of the guidelines. By providing recommendations suitable for Korean youth, these guidelines will help in the prevention and management of childhood HTN, thus relieving the burden of cardiovascular disease in adulthood in Korea.

Keywords: Adolescent, Blood pressure, Child, Guideline, Hypertension

Introduction

Pediatric hypertension (HTN) has become a significant health concern. While HTN was previously considered uncommon in the pediatric population, recent studies have shown an increase in the prevalence of HTN in children and adolescents globally and in Korea, largely due to an increase in childhood obesity [1,2]. Accordingly, primary HTN has become more prevalent than secondary HTN in youths, especially in developed countries [3–5].

Childhood HTN commonly continues into adulthood and may lead to cardiovascular disease (CVD) and the pro-

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gression of kidney disease in adulthood [6–8]. In addition, HTN-induced target organ damage (TOD), such as left ventricular hypertrophy (LVH) and arterial thickening and stiffening, has been observed, even in childhood [9–16]. Thus, the early diagnosis and management of HTN in childhood are essential.

The most widely used international HTN guidelines for children and adolescents are the American Academy of Pediatrics (AAP) and European Society of Hypertension (ESH) guidelines [17–20]. These two guidelines contain many differences in the diagnosis, evaluation, and management of HTN and were developed for Western youths, who are racially and ethnically different from Korean youths. In addition, Korea has a medical and insurance system that differs from those of the United States and the European Union. Therefore, HTN guidelines for Korean children and adolescents should be established. The Korean Working Group of Pediatric Hypertension has established clinical guidelines for the diagnosis, evaluation, and management of HTN in Korean children and adolescents. These guidelines are based on reported clinical evidence, expert recommendations, and AAP and ESH guidelines. The characteristics of Korean youth were considered during the establishment of the guidelines. By providing recommendations suitable for Korean youths, these guidelines will help in the prevention and management of HTN in childhood, thus relieving the burden of CVD in adulthood in Korea.

Evidence grading

A. Recommendations are based on randomized trials (or systematic reviews of trials) with high levels of internal validity and statistical precision, provided that the trial results can be directly applied to patients because they have similar clinical characteristics and clinically relevant outcomes.
B. Recommendations are based on randomized trials, systematic reviews, or prespecified subgroup analyses that have lower levels of precision or require extrapolation from studies in different populations or using validated intermediate or surrogate outcomes.
C. Recommendations are based on trials with lower levels of internal validity and/or precision, trials for which non-validated surrogate outcomes were used, or results from observational studies.
D. Recommendations are based on expert opinion alone.

Recommendation class

Class I: Evidence and/or general agreement exists that a given treatment or procedure is beneficial, useful, and effective. Therefore, this treatment or procedure is recommended or indicated.
Class IIa: Conflicting evidence and/or a divergence of opinion exists about the usefulness or efficacy of the given treatment or procedure. However, in general, the weight of evidence/opinion favors usefulness/efficacy. Therefore, this treatment or procedure should be performed.
Class IIb: Conflicting evidence and/or a divergence of opinion exists about the usefulness or efficacy of the given treatment or procedure. The usefulness or efficacy is not well established by evidence or opinion. Therefore, this treatment or procedure may be considered.
Class III: Evidence or general agreement exists that a given treatment or procedure is not beneficial and may be harmful in some cases Therefore, this method is not recommended.

Epidemiology

The AAP and ESH HTN guidelines for children and adolescents classify blood pressure (BP) values differently. HTN is defined as a BP ≥the 95th percentile for sex, age, and height in younger youths aged <13 and <16 years in the AAP and ESH guidelines, respectively, using different reference BP values. Furthermore, HTN for older youths is defined as a BP ≥130/80 mmHg for adolescents aged ≥13 years in the AAP guideline and a BP ≥140/90 mmHg for those aged ≥16 years in the ESH guideline [17–19]. Therefore, the estimated prevalence of HTN in children and adolescents can vary according to the guidelines used as references for BP classification.

The estimated nationwide prevalence of HTN among Korean children and adolescents aged 10 to 18 years, based on data from the Korea National Health and Nutrition Examination Survey (KNHANES) from 2011–2020, was 4.2% when using the BP classification of the ESH guideline [21]. The HTN prevalence was higher in boys (5.2%) than in girls (3.1%). The prevalence of HTN in Korean youths increased with an increase in obesity: 3.1% in normal weight, 7.1%

in overweight, and 12.7% in obese individuals. The prevalence tended to increase with age: 3.6% in youths aged 10 to 15 years and 6% in those aged 16 to 18 years.

Another study on secular trends in elevated BP and HTN among Korean children and adolescents aged 10 to 18 years, analyzing KNHANES data from 2007 to 2015 and using the BP classification of the AAP guidelines, reported that the prevalence of elevated BP and HTN increased during the study period: 5.4% (2007–2009), 7.3% (2010–2012), and 8.9% (2013–2015) for elevated BP and 6.9% (2007–2009), 8.2% (2010–2012), and 9% (2013–2015) for HTN. The associated factors were sex, age, and body mass index (BMI) [2].

A study investigating the parent-offspring association of HTN in youths aged 10 to 18 years using KNHANES data from 2008 to 2018 based on the BP classification of the ESH guidelines showed that when neither parent, only the father, only the mother, or both parents were hypertensive, 6.6%, 10.4%, 13.3%, and 25.3% of boys and 6%, 12%, 12.7%, and 22.1% of girls had HTN, respectively. The risk of having HTN among offspring was approximately twice as high when one parent was hypertensive and greater than four times higher when both parents were hypertensive compared to that of children whose parents were not hypertensive (odds ratio: 2.230, 1.655, and 5.021 in boys with HTN and 2.321, 2.169, and 4.554 in girls with HTN in mothers only, fathers only, and both parents, respectively) [22].

Causes of hypertension

Primary hypertension

Recent studies have reported that primary HTN is becoming the predominant type of HTN among clinically diagnosed hypertensive children and adolescents, especially in developed countries [3–5]. Children and adolescents with primary HTN are associated with being overweight or obese [4,23–25], having a family history of HTN [4,24,26], and having an onset at an older age (≥6 years) [25,26].

Kidney and renovascular hypertension

Kidney and renovascular diseases are the most common causes of secondary HTN in children and adolescents [26–28]. More than 60% of children with HTN aged <6 years who were enrolled in large-scale clinical trials had kidney and renovascular HTN [25,29–31]. Accordingly, kidney and renovascular diseases should be suspected as the most probable causes of HTN in children aged <6 years.

Cardiac disease (e.g., aortic coarctation)

Coarctation of the aorta is the narrowing of the aorta that most commonly occurs just beyond the left subclavian artery. This narrowing increases the upper body BP, causing upper extremity HTN. Aortic coarctation should be suspected in hypertensive youths when the upper extremity systolic BP (SBP) is higher than the lower extremity SBP [32]. The SBP in the leg is usually 10% to 20% higher than that in the arm.

Patients may remain hypertensive or develop HTN even after early and successful repair of aortic coarctation [33]. Additionally, patients with repaired coarctation are at high risk for masked HTN (MH) and LVH [33,34]. Therefore, continuous follow-up assessment using four extremity BP measurements, ambulatory BP monitoring (ABPM), and echocardiography are required in patients with repaired aortic coarctation.

Endocrine hypertension

Endocrine HTN, i.e., HTN caused by hormonal excess, is uncommon in children and adolescents, with a prevalence ranging from 0.05% to 6% among hypertensive youth [24–27]. However, identifying endocrine HTN is important, as each requires specific and appropriate treatment. Endocrine diseases associated with HTN include disorders of catecholamines (pheochromocytoma), mineralocorticoids (congenital adrenal hyperplasia and familial hyperaldosteronism), glucocorticoids (Cushing syndrome), and thyroid and parathyroid hormones (hyperthyroidism and hyperparathyroidism).

Neurofibromatosis

Neurofibromatosis type I (NF-I) is an autosomal dominant disorder characterized by café-au-lait macules, neurofibromas, Lisch nodules of the iris, axillary and groin freckling, optic nerve gliomas, macrocephaly, scoliosis, limb deformities, short stature, and learning disabilities. NF-1

can cause secondary HTN through kidney artery stenosis, aortic coarctation, middle aortic syndrome, and pheochromocytoma [35–40]. Additionally, the incidence of idiopathic HTN is approximately 6% in patients with NF-1, which is higher than that in general papulation [41].

Monogenic hypertension

Monogenic HTN is very rare and includes familial hyperaldosteronism type I, glucocorticoid remediable aldosteronism, Liddle syndrome, pseudohypoaldosteronism type II, apparent mineralocorticoid excess, familial glucocorticoid resistance, mineralocorticoid receptor activating mutation, and congenital adrenal hyperplasia [42,43]. Monogenic HTN should be suspected in hypertensive patients with low plasma renin activity or a high aldosterone-to-renin ratio (>10), especially when they have a family history of early-onset HTN [42].

Environmental exposure and medication-related hypertension

Studies have reported that exposure to lead, cadmium, mercury, and phthalates may be associated with HTN [44–61]. Common medications associated with an elevated BP include oral contraceptives [62–64], central nerve stimulants [65], and corticosteroids [66]. The elevation in BP caused by medication is rarely significant and is mostly reversible upon cessation of the medication. When evaluating hypertensive patients, physicians should inquire about exposure to harmful materials and the intake of drugs, including herbal medications, which might affect BP.

Comorbidities

Obesity

Evidence from studies on children and adolescents indicates that the incidence of HTN increases with increasing severity of obesity, as estimated by the BMI, waist circumference, or other adiposity indexes [67–77]. In addition, up to 50% of children and adolescents with obesity have abnormal circadian BP patterns, with not having nocturnal BP dipping, a physiological decrease in BP during the nighttime to a 10% to 20% lower level than the daytime BP [78–82]. Accordingly, obese children and adolescents are at risk of developing MH. Even normotensive children with obesity may be at risk of developing HTN [71]. This risk appears to increase with the severity of obesity [83]. Therefore, children and adolescents with obesity require their BP values to be checked at every clinical visit and at least yearly.

In addition, obesity is likely associated with other cardiovascular risk factors including dyslipidemia and diabetes mellitus (DM) [84,85], which are independent risk factors for HTN [74,86]. Adolescents with multiple cardiovascular risk factors, including obesity, HTN, dyslipidemia, or DM, have a higher prevalence of cardiac TOD, including LVH and decreased left ventricular (LV) systolic and diastolic functions, than those with a single cardiovascular risk factor [87].

Chronic kidney disease

The ability of chronic kidney disease (CKD) to cause HTN in pediatric patients is well documented. Approximately 50% of children and adolescents with CKD may have HTN [13,14], and CKD may account for approximately 20% of pediatric HTN cases [88]. Studies of adults have shown that HTN may cause CKD; however, that correlation is unclear in children. Additionally, CKD may affect physiological circadian variations in BP, leading to non-dipping nocturnal BP. In adults, non-dipping is an independent cardiovascular risk factor [89] that may lead to faster progression of kidney failure [90,91], even though this association is uncertain in children.

Diabetes mellitus

HTN occurs in 20% to 50% and 5% to 40% of pediatric patients with type 2 and type 1 DM, respectively, which are much higher rates than those in the general population [92–94]. Patients with pediatric DM are likely to have decreased nocturnal BP dipping, which is a risk factor for MH. Elevated BP in pediatric patients with DM is associated with an increased risk of developing microalbuminuria (MA), nephropathy, hypertensive retinopathy, and carotid artery thickening and stiffening [95].

Blood pressure classification

Data identifying a specific BP value in childhood that leads to CVD in adulthood are lacking, and HTN in children and adolescents aged ≥1 year is defined statistically as having an SBP and/or diastolic BP (DBP) of ≥the 95th percentile based on age, sex, and height percentiles using BP reference values from the AAP and ESH guidelines. Because no BP reference values for Korean children aged <10 years exist [96], this definition cannot be used for the diagnosis of HTN in young children in Korea.

Additionally, the diagnosis of HTN in youths using this method is not as simple as that in adults, requiring a cumbersome process to find the 95th percentile BP value, even with an available online site (https://www.koreanhypertension.org/sense/calculator). In order to solve these problems, the Korean Working Group of Pediatric Hypertension compared and analyzed the United States and Korean BP reference values for youths (Table 1, Fig. 1) and suggested the following formula as an approximated definition of HTN in Korean children and adolescents aged 1 to 16 years: SBP ≥100 + (age × 2) mmHg and/or DBP ≥60 + (age × 1.5) mmHg [97].

In the HTN guidelines for Korean adults [98], BP was classified into normal BP, elevated BP, pre-HTN, stage 1 HTN, and stage 2 HTN. Since differences in SBP and DBP between the 90th and 95th percentile are only about 5 mmHg in children and adolescents, it is difficult to define elevated BP and pre-HTN between normal BP and HTN in all age groups in children and adolescents. Therefore, we included elevated BP in BP class only in adolescents aged ≥13 years. BP classification is summarized in Table 2.

Table 1. Comparison between the estimated approximate BP values and the percentile (P) BP values at the 50th P height

Variable	Correlation coefficient	p-value for correlation	Paired difference		
			Mean difference	95% CI of difference	p-value for difference
SBP 95th P vs. (age × 2) + 99	0.961	<0.001	−0.712	−1.480 to 0.057	0.07
SBP 90th P vs. (age × 2) + 95	0.962	<0.001	−0.558	−1.346 to 0.231	0.16
DBP 95th P vs. (age × 1.5) + 60	0.955	<0.001	−0.269	−0.878 to 0.339	0.38
DBP 90th P vs. (age × 1.5) + 56	0.953	<0.001	0.500	−0.124 to 1.124	0.11

BP, blood pressure; CI, confidence interval; DBP, diastolic blood pressure; SBP, systolic blood pressure.
Adapted from Kim et al. (*Clin Hypertens* 2022;28:19) [97] according to the Creative Commons License.

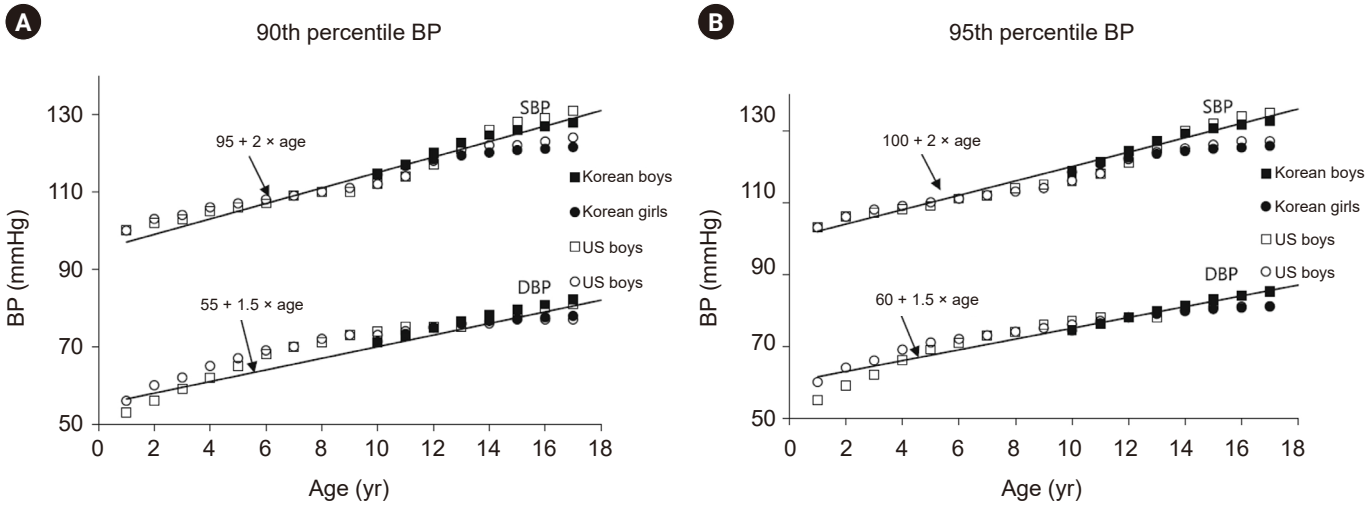

Figure 1. Percentile BP trends by age and sex at the 50th percentile height. The 90th (A) and 95th (B) percentile SBP and DBP with approximate trend lines for Korean and the United States (US) boys and girls.
BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure.

Table 2. Modified simple BP classification in children and adolescents

Category	1–16 years		≥17 years
	SBP and/or DBP (percentile)	SBP and/or DBP (mmHg)	SBP and/or DBP (mmHg)
Normal BP	<90th (<120 mmHg)[a] and <90th	<(95 + 2A) (<120)[a] and <(55 + 1.5A)	<120 and <80
Elevated BP[b]	≥120 mmHg to <90th and <90th	120 ~ (95 + 2A) and <(55 + 1.5A)	120–129 and <80
Pre-HTN	≥90th to <95th or ≥90th to <95th	(95 + 2A) to (99 + 2A) or (55 + 1.5A) to (59 + 1.5A)	130–139 or 80–89
Stage 1 HTN	≥95th to <99th + 5 mmHg or ≥95th to <99th + 5 mmHg	(100 + 2A) to (114 + 2A) or (60 + 1.5A) to (69 + 1.5A)	140–159 or 90–99
Stage 2 HTN	≥99th + 5 mmHg or ≥99th + 5 mmHg	≥(115 + 2A) or ≥(70 + 1.5A)	≥160 or ≥100

BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure; HTN, hypertension; A, age (year).
[a]In adolescents aged ≥13 years. [b]Elevated BP is included in BP class only in adolescents aged ≥13 years.

Neonates and infants aged 0 to 1 year

Defining HTN in neonates is difficult because BP values change significantly during the first few weeks of life [99]. These BP changes are likely influenced by gestational age, birth weight, and maternal conditions [100]. The available data on BP values for neonates and infants include the table containing BP values for the 95th and 99th percentiles for neonates aged 26 to 44 weeks reported by Dionne et al. [99] and the BP percentile curves for infants aged ≤1 year published in the 1987 "*Report of the Second Task Force on Blood Pressure Control in Children*" [101]. The BPs of neonates and infants may be classified using these BP data with a method similar to that used for older children [99].

Blood pressure measurement

Clinic blood pressure measurement

The method of BP measurement is presented in Table 3 [18].

In stage 2 or resistant stage 1 HTN, the upper and lower extremity BP should be checked (right arm, left arm, and one leg) (Class IIb, level D) [18]. Oscillometric devices validated for use in pediatric patients can be used for BP screening (Class IIa, level B) [102–106]. However, the pediatric BP reference values used for the determination of BP status can be obtained using the auscultatory method. The BP values obtained using the oscillometric method may differ from those obtained using the auscultatory method. Therefore, to confirm the diagnosis of HTN, auscultatory devices should be used (Class IIb level B) [107–111]. The oscillometric and auscultatory devices validated for use in children and adolescents are available at www.hyperchild-net.eu and www.dableducational.org.

Table 3. Clinic BP measurement method

- Be seated in a quiet room for 5 minutes before measurement with back supported and feet uncrossed and flat on the floor.
- Measure BP in the right arm.
- Right arm should be at heart level, supported, and uncovered above the cuff.
- Cuff size:
  - Bladder length and width: 80%–100% and >40% of the arm circumference, respectively.
- Auscultatory BP
  - The bell of the stethoscope: over the brachial artery in the antecubital fossa.
  - The lower end of the cuff: 2–3 cm above the antecubital fossa.
  - Cuff inflation: 20–30 mmHg above the point at which the radial pulse disappears.
  - Cuff deflation: at a rate of 2–3 mmHg per second.
  - The first audible sound (phase I Korotkoff): SBP.
  - The last audible (phase V Korotkoff) or muffled (phase IV Korotkoff) sound: DBP.
- BP in the leg
  - Be in the prone position.
  - Place an appropriately sized cuff on the mid-thigh and the stethoscope over the popliteal artery.
  - The SBP is usually 10%–20% higher in the leg than in the arm.
- Measure BP 3 times with 3-minute intervals and average the last two BP values.

BP, blood pressure; DBP, diastolic blood pressure; SBP, systolic blood pressure.
Data from Flynn et al. (*Pediatrics* 2017;140:e20171904) [18].

Nwankwo et al. [112] provided the standardized protocol for BP measurement in neonates, which is presented in Table 4.

When the initial clinically measured BP is ≥the 90th per-

Table 4. BP measurement techniques in neonate

· Measure BP by oscillometric device
· More than 1.5 hours after a feed or medical intervention
· In a prone or supine position
· Use appropriately sized BP cuff (bladder length and width: 80%–100% and 45%–55% of arm circumference, respectively)
· Measure BP in the right upper arm
· After cuff placement, infant should be left undisturbed for 15 minutes
· Asleep or in quiet awake state
· Three successive BP readings at 2-minute intervals, and average the last two values.

BP, blood pressure.
Data from Nwankwo et al. (*Pediatrics* 1997;99:E10) [112].

Table 5. Method for ABPM

· Use a device validated in youths (www.hyperchildnet.eu and www.dableducational.org).
· Use a cuff with correct size (bladder length/width: 80%–100%/>40% of arm circumference).
· Fit the cuff on a bare nondominant arm.
· Set the measurement frequency with 15–20 minutes for daytime and 20–30 minutes for nighttime.
· To determine BP levels, reading numbers should be ≥20 during daytime and ≥7 during night.
· Keep regular activity (avoid vigorous exercise or whole day inactivity) during ABPM.
· Be still with arm extended and relaxed at each measurement.
· Patients should record sleep time, medications, symptoms, and problems during ABPM.

ABPM, ambulatory blood pressure monitoring; BP, blood pressure.
Data from Flynn et al. (*Hypertension* 2014;63:1116-1135) [128].

centile [approximately, SBP ≥95 + (age × 2) mmHg and/or DBP ≥55 + (age × 1.5) mmHg], BP should be remeasured at two additional clinic visits using the auscultatory method, and the average of these two BP should be used to determine the BP status (i.e., normal BP, pre-HTN, or HTN) (Class IIa, level C) [113–115].

BP should be measured annually in youths aged ≥3 years (Class IIa, level B) [116]. BP should be measured at every clinical visit and at least yearly in children and adolescents with HTN and high-risk conditions for HTN, including kidney disease, DM, solid organ transplant, aortic coarctation (pre- and postoperative), obesity, HTN-related endocrine or genetic diseases, premature birth, pre-HTN, and treated HTN (Class IIa, level B) [33,76,88,117,118].

Ambulatory blood pressure monitoring

White coat hypertension

White coat hypertension (WCH) is defined as an elevated BP in the clinic but a normal BP outside the clinic. This phenomenon is likely due to anxiety experienced during clinic visits. WCH can be diagnosed when patients have elevated clinical BP but normal BP on ABPM. Recent studies have reported that up to half of the children referred for evaluation of elevated clinical BP have WCH [119,120]. Due to the lack of long-term follow-up data on WCH in children and adolescents, little is known about its actual impact. Hence, whether WCH is a harmless phenomenon or a prelude to future sustained HTN remains unclear.

Masked hypertension

MH is a contrasting phenomenon to WCH, whereby BP values are normal in the clinic but elevated outside the clinic. MH occurs in approximately 5% to 10% of unselected children and adolescents [34,121–124]. MH can be diagnosed when patients have normal clinical BP but elevated BP on ABPM. Patients with MH have a significant risk of TOD [124,125]. Patients with CKD, DM, solid organ transplantation, aortic coarctation (pre- and postoperative), obesity, HTN-related endocrine or genetic diseases, and treated HTN are at a high risk for MH. In particular, CKD is strongly associated with MH [126] and TOD [125].

Clinical use of ambulatory blood pressure monitoring

ABPM-based BP values provide useful information for the diagnosis and management of HTN by revealing BP patterns throughout the day in real-life conditions and allowing comparison of clinical BP values obtained in an unusual environment at a specific time. BP values obtained using ABPM are more closely associated with the presence of TOD and have a higher reproducibility than those obtained using clinical measurements [127]. The ABPM method is presented in Table 5 [128].

HTN can be diagnosed in children and adolescents using ABPM based on an average SBP and/or DBP for 24 hours, during the daytime, and/or during the nighttime that is in the ≥95th percentile for age, sex, and height, as

long as the threshold values are inferior to the adult criteria (130/80 mmHg for 24 hours, 135/85 mmHg in the daytime, or 120/70 mmHg in the nighttime) (Class IIa, level C) [129,130]. The reference ABPM BP values are presented in Tables 6 and 7 [131].

ABPM should be performed before starting antihypertensive drug treatment to rule out WCH (Table 8) (Class IIa, level B) [119,132]. ABPM should also be performed in children and adolescents with CKD, DM, solid organ transplant, aortic coarctation (pre- and postoperative), obesity, HTN-related endocrine or genetic diseases, and treated HTN in order to detect MH and assess HTN sever-

Table 6. Reference blood pressure values for height in ABPM in boys

Height (cm)	Boys (percentile)											
	24 hours				Day				Night			
	50th	75th	90th	95th	50th	75th	90th	95th	50th	75th	90th	95th
120	105/66	109/70	114/74	117/77	111/72	116/77	122/80	125/82	94/54	99/58	103/61	106/63
125	105/66	110/70	115/74	118/77	111/72	117/76	122/80	125/82	95/55	100/58	105/61	108/63
130	106/66	111/70	116/74	119/77	112/72	117/76	122/80	126/82	96/55	101/59	106/62	110/64
135	107/66	112/70	117/74	120/77	112/72	117/76	123/80	126/82	97/56	102/59	108/63	111/65
140	108/67	113/71	118/75	121/77	113/72	118/76	123/80	126/82	98/56	104/60	109/63	113/65
145	110/67	115/71	120/75	123/77	114/72	119/76	124/79	127/81	99/56	105/60	111/64	114/66
150	111/67	116/71	121/75	124/77	115/72	120/76	125/79	128/81	100/56	106/60	112/64	116/66
155	113/67	118/71	123/75	126/77	117/72	122/76	127/79	130/81	101/56	107/60	113/64	117/66
160	114/67	120/71	124/75	127/77	119/72	124/76	129/79	133/81	103/56	108/60	114/64	118/66
165	116/68	121/71	126/75	129/78	121/72	126/76	132/80	135/82	104/57	110/60	116/64	119/66
170	118/68	123/72	128/75	131/78	123/73	128/77	134/80	138/82	106/57	112/61	117/64	121/66
175	120/68	125/72	130/75	133/78	124/73	130/77	136/81	140/83	107/57	113/61	119/64	122/66
180	122/68	127/72	131/76	134/78	126/73	132/77	138/81	142/83	109/57	115/61	120/64	124/66
185	123/68	128/72	133/76	136/78	128/73	134/78	140/81	144/84	110/57	116/61	122/64	125/66

Data are expressed as systolic blood pressure/diastolic blood pressure (mmHg).
ABPM, ambulatory blood pressure monitoring.
Data from Wühl et al. (*J Hypertens* 2002;20:1995-2007) [131].

Table 7. Reference blood pressure values for height in ABPM in girls

Height (cm)	Girls (percentile)											
	24 hours				Day				Night			
	50th	75th	90th	95th	50th	75th	90th	95th	50th	75th	90th	95th
120	104/66	108/69	112/71	114/72	110/73	114/77	118/80	120/82	95/55	99/60	103/63	106/65
125	105/66	109/69	113/71	116/73	111/73	115/77	119/80	121/82	96/55	100/60	104/63	107/66
130	106/66	110/69	114/72	117/73	111/72	116/76	120/80	122/82	96/55	101/59	106/63	108/66
135	107/66	111/70	115/72	118/74	112/72	116/76	120/80	123/82	97/55	102/59	107/63	109/66
140	108/66	112/70	116/73	119/75	112/72	117/76	121/80	124/82	98/55	103/59	108/63	110/66
145	109/66	113/70	117/73	120/75	113/72	118/76	123/80	125/82	98/54	103/59	109/63	112/66
150	110/67	115/70	119/74	121/76	114/72	119/76	124/80	127/82	99/54	104/59	110/63	113/66
155	111/67	116/71	120/74	123/76	116/72	121/76	125/80	128/82	100/54	106/59	111/63	114/66
160	112/67	117/71	121/74	123/76	117/72	122/76	126/80	129/82	101/55	106/59	111/63	114/66
165	114/67	118/71	122/74	124/76	118/73	123/77	127/80	130/82	102/55	107/59	112/63	114/66
170	115/68	119/71	123/74	125/76	120/74	124/77	128/80	131/82	103/55	108/61	112/67	115/71
175	116/69	120/72	124/75	126/76	121/75	125/78	129/81	131/82	105/55	109/59	113/63	115/66

Data are expressed as systolic blood pressure/diastolic blood pressure (mmHg).
ABPM, ambulatory blood pressure monitoring.
Data from Wühl et al. (*J Hypertens* 2002;20:1995-2007) [131].

Table 8. Indications for ABPM

- To exclude WCH
  - Before starting antihypertensive drug treatment
- To detect MH
  - CKD (repeat periodically)
  - DM
  - Solid organ transplant
  - Aortic coarctation (pre- and postoperative)
  - Obesity
  - Endocrine or genetic diseases known to be related to HTN
  - Treated HTN
- During antihypertensive drug treatment
  - Insufficient BP response to treatment
  - Symptoms of hypotension

ABPM, ambulatory blood pressure monitoring; BP, blood pressure; CKD, chronic kidney disease; DM, diabetes mellitus; HTN, hypertension; MH, masked hypertension; WCH, white coat hypertension.

Table 9. Method for home BP measurement

- Patients and parents require training for BP measurement.
- Use an oscillometric upper-arm cuff device validated in youths (www.hyperchildnet.eu and www.dableducational.org).
- Use a cuff with correct size (bladder length/width: 80%–100%/>40% of arm circumference).
- Be seated in a quiet room for 5 minutes before measurement with back supported and feet uncrossed and flat on the floor.
- Place the cuff on the bare right arm.
- Right arm should be at heart level, supported, and uncovered above the cuff.
- Measure BP
  - For ≥3 days (ideally for 7 consecutive days)
  - In the morning and evening (before medication)
  - Twice per every occasion with 1–2 minutes interval
- Discard the first day readings and average all the other readings.

BP, blood pressure.
Data from Parati et al. (*J Hypertens* 2008;26:1505-1526) [149].

ity and circadian BP patterns (Table 8) (Class IIa, level B) [33,34,121,124,125,129,133].

In children and adolescents with an insufficient BP response to treatment or symptoms of hypotension, ABPM should be performed during antihypertensive drug treatment (Table 8) (Class IIa, level B) [134–136]. Patients with CKD should be periodically evaluated for MH using ABPM as part of routine CKD management (Table 8) (Class IIa, level B) [137–140].

Home blood pressure monitoring

Home BP monitoring is a convenient and feasible method to obtain BP values at any time [141–145]. Considering ABPM as a reference method, the reported accuracy of home BP monitoring is higher than that of clinical BP in children and adolescents, even though it varies widely among studies (sensitivity, 55%–88%; specificity, 70%–92%) [122,144,146,147]. Children and adolescents have lower home BP than daytime ambulatory BP [137,144,146,148]. In addition, home BP monitoring has demonstrated high accuracy in diagnosing WCH but not MH in children and adolescents [122,146]. The home BP measurement methods are presented in Table 9 [149].

The suggested definition of HTN using home BP monitoring in youths is an SBP and/or DBP in the ≥95th percentile for sex and height. The available reference values for home BP are presented in Table 10 [143]. Home BP monitoring can be a useful supportive test for children and adolescents receiving antihypertensive treatment after the diagnosis of HTN or those suspected of having WCH or MH, in addition to clinical BP measurements and ABPM (Class IIa, level C) [137,150–152].

Table 10. Home blood pressure values

Height (cm)	Boys (percentile)		Girls (percentile)	
	50th	95th	50th	95th
120–129	105/64	119/76	101/64	119/74
130–139	108/64	121/77	103/64	120/76
140–149	110/65	125/77	105/65	122/77
150–159	112/65	126/78	108/66	123/77
160–169	115/65	128/78	110/66	124/78
170–179	117/66	132/78	112/66	125/79
180–189	121/67	134/79	114/67	128/80

Data are expressed as systolic blood pressure/diastolic blood pressure (mmHg).
Data from Stergiou et al. (*J Hypertens* 2007;25:1375-1379) [143].

Diagnostic evaluation

The first step in the evaluation of children and adolescents with HTN should be history taking (family history, symptoms of secondary HTN and TOD, and risk factors) and physical examination in order to identify findings suggestive of secondary causes of HTN and TOD (Class I, level B) [137,153–155]. History-taking and physical examination

methods are summarized in Tables 11 and 12. Laboratory and imaging tests should be performed in children and adolescents with HTN to identify the underlying secondary causes of HTN and TOD (Class IIa, level C) [156–158]. Laboratory and imaging tests are summarized in Table 13.

MA is a marker of HTN-related kidney injury and a predictor of CVD in adults [159–162]. However, MA can occur in the absence of HTN in children; it can occur in children with obesity, insulin resistance, DM, dyslipidemia, or vigorous physical activity [163]. In addition, the evidence suggesting that MA is a marker of HTN-induced TOD in children with primary HTN is insufficient. Accordingly, routine testing for MA is not recommended for children and adolescents with primary HTN (Class IIa, level C) [162,164–166].

Table 11. History taking

Family history
· Metabolic syndrome (HTN, DM, dyslipidemia, obesity)
· Cardiovascular disease
· Hereditary kidney disease: polycystic kidney disease, Alport syndrome, etc.
· Hereditary endocrine disease: pheochromocytoma, familial hyperaldosteronism, etc.
· Neurofibromatosis
History of symptoms of secondary HTN
· Perinatal history:
- Prematurity, oligohydramnios, low birth weight, anoxia, umbilical artery catheterization
· Cardiac, endocrine, or neurological disease:
- Cold extremities, intermittent claudication, palpitations, sweating, fever, pallor, flushing, muscle weakness, virilization, primary amenorrhea
· Kidney or urologic disease:
- Recurrent UTI, edema, poor weight gain, hematuria, polyuria, nocturia, thirst
· Systemic disease:
- Neurofibromatosis, lupus erythematosus, etc.
· Malignancy, solid organ transplantation, bone marrow transplantation
· Drugs known to raise BP:
- Oral contraceptives, central nerve stimulants, corticosteroids, etc.
History of symptoms of TOD
· Headache, epistaxis, vertigo, visual impairment, facial palsy, seizures, strokes, low school performance, dyspnea, chest pain, palpitations, syncope
Risk factors
· DM, dyslipidemia, obesity, physical activity, dietary habits, smoking, alcohol, snoring, sleep apnea history

BP, blood pressure; DM, diabetes mellitus; HTN, hypertension; TOD, target organ damage; UTI, urinary tract infection.

Imaging evaluation

The heart

HTN is strongly associated with LVH [9–12]. LVH has been independently and strongly associated with CVD in adults [167–169]. Antihypertensive treatments can reduce LVH. The definitions of LVH on echocardiography are presented in Tables 14 and 15 [20,170].

LV ejection fraction may be significantly decreased in patients with severe or acute-onset HTN with associated congestive heart failure. LV ejection fraction may rarely be mildly depressed in patients with chronic HTN. LV systolic dysfunction is defined as an ejection fraction of <53% (Table 14) [20,170].

Echocardiography should be performed in children and adolescents diagnosed with HTN to assess cardiac TOD and to determine the treatment modality for HTN (Class IIa, level C) [171–174]. Repeat echocardiography should be performed at 6- to 12-month intervals in children and adolescents with stage 2 HTN, secondary HTN, persistent HTN despite treatment, or cardiac TOD, including LVH and LV dysfunction (Class IIa, level C) [171–174].

Blood vessels

Improved imaging methods (high-definition ultrasound and echo-tracking) have enhanced 'reference' values for carotid intima-media thickness and arterial stiffness in healthy children aged 3 to 18 years [175,176]. Recent studies have reported that these parameters are elevated in children with HTN [177,178], familial hypercholesterolemia [179,180], overweight [181,182], and type 1 DM [183].

Arterial stiffening, measured by peak wave velocity, predicts cardiovascular events and mortality in adults [184]. Reference values for peak wave velocity are available from three studies performed in childhood [185–187]. Overall, current data suggest that childhood BP only variably predicts increased peak wave velocity [188–190]. Emerging data suggest that functional changes in large vessels are the earliest detectable findings in children, such as those with familial hypercholesterolemia and CKD [191,192]. Routine

Table 12. Physical examination

Organ system	Findings	Possible causes of HTN	TOD
Height, weight, WC, BMI	Obesity	Polycystic ovary syndrome, DM, Cushing syndrome, etc.	
	Growth retardation	Chronic kidney disease	
Vital sign	Tachycardia	Hyperthyroidism, pheochromocytoma, etc.	Hypertensive cardiomyopathy
	Weak femoral pulses	Coarctation of the aorta	
Skin	Hirsutism	Polycystic ovary syndrome, adrenal hyperplasia, etc.	
	Malar rash	SLE	
	Neurofibromas	Neurofibromatosis	
	Adenoma sebaceum	Tuberous sclerosis	
	Acanthosis nigricans	Polycystic ovary syndrome, DM, Cushing syndrome, etc.	
Eyes	Proptosis	Hyperthyroidism	Hypertensive retinopathy
	Cataract	DM, Corticosteroids, etc.	
	Retinal changes		
Throat	Tonsilar hypertrophy	Sleep disorder	
Head, neck	Webbed neck	Turner syndrome	
	Elfin face	Williams syndrome	
	Moon face	Cushing syndrome	
	Goiter	Hyperthyroidism	
Chest	Cardiac murmur	Coarctation of the aorta	Hypertensive cardiomyopathy
Abdomen	Bruit	Midaortic syndrome, renovascular hypertension	
	Mass	Neuroblastoma, Wilms tumor	
		Pheochromocytoma	
		Polycystic kidney disease, etc.	
Genitourinary	Ambiguous genitalia, virilization	Congenital adrenal hyperplasia	
Neurologic	Cranial nerve palsy		Hypertensive neuropathy
	Hemiparesis		

BMI, body mass index; DM, diabetes mellitus; HTN, hypertension; SLE, systemic lupus erythematosus; TOD, target organ damage; WC, waist circumference.

assessment of arterial stiffness and central BP parameters is not currently recommended and should remain a research area until further information becomes available (Class IIa, level C).

Kidneys

Recent studies have shown that kidney Doppler ultrasonography, computed tomography (CT) angiography, and magnetic resonance (MR) angiography have high sensitivity and specificity for the detection of kidney artery stenosis [193–195], comparable to that of the gold standard kidney angiography. Therefore, in children and adolescents suspected of having renovascular HTN [196], kidney Doppler ultrasonography, CT angiography, or MR angiography can be performed as noninvasive imaging studies (Class IIa, level C) [193–198]. Additionally, kidney ultrasonography may help identify possible causes of HTN, including Wilms tumor, neuroblastoma, and kidney cystic dysplasia.

Eyes and brain

Studies on the incidence, findings, and prognosis of hypertensive retinopathy in children are rare [199]. The reported incidence of hypertensive retinopathy is very low, even among children with essential HTN [200,201]. Fundoscopic examination is likely to be limited to children with symptoms (headache, visual impairment, dizziness, impaired consciousness, seizures, or neurological deficits, such as facial nerve paresis), encephalopathy, or malignant HTN. Sustained and severe HTN may cause brain damage with symptoms and signs including cerebral seizures, stroke,

Table 13. Laboratory and imaging tests

Subjects	Laboratory and imaging tests
All patients	Blood count and morphology
	Electrolytes, calcium, phosphorus
	Blood urea nitrogen, creatinine
	Protein, albumin, uric acid
	Glucose (fasting)
	ALT, AST
	Lipid profile (fasting)
	Urinalysis
	Echocardiography (TOD, for determining treatment modality)
Patients with	Renin, aldosterone (renovascular HTN, hyperaldosteronism, etc.)
Aged <6 yr	Plasma and urine catecholamines (pheochromocytoma, etc.)
Resistant HTN	Steroid hormone tests (congenital adrenal hyperplasia, etc.)
Suspected secondary HTN	Thyroid function tests (hyperthyroidism, etc.)
Or stage 2 HTN	Kidney ultrasonography (renovascular HTN, etc.)

ALT, alanine aminotransferase; AST, aspartate transaminase; HTN, hypertension; TOD, target organ damage.

Table 14. Target organ damage

Organ	Evaluation	Findings
Heart	Echocardiography	LV hypertrophy:
		Aged <9 yr: LVM/height$^{2.7}$ ≥95th percentile
		Aged ≥9 yr: LVM/height$^{2.7}$ >45 g/m$^{2.7}$ (boys), >40 g/m$^{2.7}$ (girls)
		(or aged ≥16 yr: LVM/BSA >115 g/m^2 [boys], >95 g/m^2 [girls])
		LV RWT (2PWT/LVID) >0.42 (concentric hypertrophy)
		LV systolic dysfunction: ejection fraction < 53%
Kidney	Urinalysis	Albuminuria: albumin/creatinine >30 mg/g
		Proteinuria: protein/creatinine >300 mg/g
Eye	Fundoscopy	Retinal vessel changes or papilledema (optional)
Vessel	cIMT, PWV	Arterial wall thickening or stiffening (optional)

BSA, body surface area; RWT, relative wall thickness; PWT, posterior wall thickness; LV, left ventricular; LVID, left ventricular internal dimension; LVM, left ventricular mass; cIMT, carotid intima-media thickness; PWV, pulse wave velocity.
Data from Wühl et al. (*Front Pediatr* 2023;11:1140617) [20] and Khoury et al. (*J Am Soc Echocardiogr* 2009;22:709-714) [170].

Table 15. Left ventricular mass index[a]

Subjects	Age				
	≤6 mo	6 mo to ≤2 yr	2 to ≤4 yr	4 to ≤6 yr	6 to ≤8 yr
Boys	85.6	68.6	55.3	48.1	44.6
Girls	80.1	57.1	52.4	44.3	43.5

Data are expressed as 95th percentile values (g/m$^{2.7}$)
[a]Left ventricular mass/height$^{2.7}$.
Data from Khoury et al. (*J Am Soc Echocardiogr* 2009;22:709-714) [170].

visual impairment, and retinal vascular changes.

Treatment

Overall goals

The purpose of treating HTN in children and adolescents is to reduce the risk of TOD in childhood and future CVD in adulthood by decreasing BP. Target BP values for hypertensive children and adolescents vary depending on the presence of risk factors, including DM, CKD, and proteinuria, as presented in Table 16 (Class IIa, level B) [202–213].

Lifestyle modification

When diagnosed with pre-HTN or HTN, children and adolescents should initiate lifestyle modifications related to diet, physical activity, and weight control (Class I, level C) [214,215]. A diet that is high in fruits, vegetables, low-fat milk products, whole grains, fish, poultry, nuts, and lean red meats and low in sugar, saturated fat, and salt is recommended (Class IIb, level C) [216–228]. Moderate-to-vigorous physical activity lasting 30 to 60 minutes and occurring

Table 16. Blood pressure goal in hypertensive children and adolescents

Subjects	Goals (SBP and DBP)
General patients	<95th percentile [approximately, <100 + (age × 2)/<60 + (age × 1.5) mmHg]
Patients with DM, but not CKD	<90th percentile [approximately, <95 + (age × 2)/<55 + (age × 1.5) mmHg]
Patients with non-proteinuric CKD (regardless of DM)	<75th percentile [approximately, <90 + (age × 2)/<50 + (age × 1.5) mmHg]
Patients with proteinuric CKD (regardless of DM)	<50th percentile [approximately, <85 + (age × 2)/<45 + (age × 1.5) mmHg]

CKD, chronic kidney disease; DBP, diastolic blood pressure; DM, diabetes mellitus; SBP, systolic blood pressure.

Table 17. Antihypertensive drugs for children and adolescents

Class of drug	Drug	Recommended starting daily dose	Maximal daily dose	Dosing interval
ACE inhibitors	Benazepril	0.2 mg/kg–10 mg	0.6 mg/kg–40 mg	q.d.
	Captopril	0.3–0.5 mg/kg/dose	6 mg/kg	b.i.d. to t.i.d.
	Enalapril	0.08–0.6 mg/kg		q.d.
	Fosinopril	0.1–0.6 mg/kg	40 mg	q.d.
	Lisinopril	0.08–0.6 mg/kg	0.6 mg/kg–40 mg	q.d.
	Ramipril	1.5–6 mg/kg		q.d.
ARBs	Candesartan	0.16–0.5 mg/kg		q.d.
	Irbesartan	75–150 mg	300 mg	q.d.
	Losartan	0.7 mg/kg–50 mg	1.4 mg/kg–100 mg	q.d. to b.i.d.
	Valsartan	0.4 mg/kg	40–80 mg	q.d.
Alpha and beta blocker	Labetalol	1–3 mg/kg	10–12 mg/kg–1,200 mg	b.i.d.
Beta blockers	Atenolol	0.5–1 mg/kg	2 mg/kg–100 mg	q.d. to b.i.d.
	Metoprolol	0.5–1 mg/kg	2 mg/kg	q.d. to b.i.d.
	Propranolol	1 mg/kg	4 mg/kg–640 mg	b.i.d. to t.i.d.
Calcium channel blockers	Amlodipine	0.06–0.3 mg/kg	5–10 mg	q.d.
	Felodipine	2.5 mg	10 mg	q.d.
	Nifedipine (SR)	0.25–0.5 mg/kg	3 mg/kg–120 mg	q.d. to b.i.d.
Central alpha-agonist	Clonidine	0.2 mg/kg	2.4 mg	b.i.d.
Diuretics	Amiloride	0.4–0.6 mg/kg	20 mg	q.d.
	Chlortalidone	0.3 mg/kg	2 mg/kg–50 mg	q.d.
	Furosemide	0.5–2 mg/kg	6 mg/kg	q.d. to b.i.d.
	Hydrochlorothiazide	0.5–1 mg/kg	3 mg/kg/day	q.d.
	Spironolactone	1 mg/kg	3.3 mg/kg–100 mg	q.d. to b.i.d.
	Eplerenone	25 mg	100 mg	q.d. to b.i.d.
	Triamterene	1–2 mg/kg	3–4 mg/kg–300 mg	b.i.d.
Peripheral alpha-blockers	Doxazosin	1 mg	4 mg	q.d.
	Prazosin	0.05–1 mg/kg	0.5 mg/kg	t.i.d.
Vasodilators	Hydralazine	0.75 mg/kg	7.5 mg/kg–200 mg	q.i.d.
	Minoxidil	0.2 mg/kg	50–100 mg/day	q.d. to t.i.d.

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; b.i.d., bis in did; q.d., quaque die; q.i.d., quater in die; SR, sustained release; t.i.d., ter in die.

Table 18. Indications and contraindications of antihypertensive drugs

Drug class	Indication	Contraindication
Diuretics potassium-sparing	Hyperaldosteronism	Chronic kidney failure
		Competitive athletes
Thiazide and thiazide-like diuretics	Chronic kidney failure	Competitive athletes
	Corticosteroid-induced HTN	Diabetes mellitus
Diuretics loop-acting	Congestive heart failure	
Beta blockers	Coarctation of aorta	Bronchial asthma
	Congestive heart failure	Diabetes mellitus
	Migraine	Competitive athletes
		Psoriasis
Calcium channel blockers	Post-transplantation	Congestive heart failure
	Migraine	
	Coarctation of aorta	
ACE inhibitors and ARBs	Chronic kidney disease	Bilateral kidney artery stenosis
	Diabetes mellitus	Kidney artery stenosis in solitary kidney
	Microalbuminuria	Hyperkalemia
	Congestive heart failure	Pregnancy
	Obesity-linked primary HTN	
Intravenous vasodilators	Life-threatening conditions	

ACE, angiotensin-converting enzyme; ARBs, angiotensin receptor blockers; HTN, hypertension.

Table 19. Antihypertensive drugs for hypertensive emergencies and urgencies

Drugs	Class	Route	Dose (onset of action)	Consideration
Sodium nitroprusside	Direct vasodilator	IV	0.5–8 mg/kg/min (within seconds)	Thiocyanate toxicity, inactivated by light
Labetalol	Alpha and beta blocker	IV	0.25–3 mg/kg/hr (5–10 min)	Contraindications in asthma, heart failure, bradycardia
Nicardipine	Calcium channel blocker	IV	1–3 mg/kg/min (within minutes)	Reflex tachycardia
Clonidine	Central alpha-agonist	IV	2–6 mg/kg/dose (10 min)	Dry mouth, sedation, rebound HTN
Esmolol	Beta blocker	IV	100–500 mg/kg/min (within seconds)	Contraindication in asthma, bradycardia
Furosemide	Loop diuretic	IV bolus	0.5–5 mg/kg/dose (within minutes)	Hypokalemia. Useful in volume HTN
Nifedipine	Calcium channel blocker	PO	0.25 mg/kg/dose (20–30 min)	Unpredictable hypotension, reflex tachycardia
Captopril	ACE inhibitor	PO	0.1–0.2 mg/kg/dose (10–20 min)	Contraindication in bilateral kidney artery stenosis
Minoxidil	Direct vasodilator	PO	0.1–0.2 mg/kg/dose (5–10 min)	Fluid retention

ACE, angiotensin-converting enzyme; HTN, hypertension; IV, intravenous; PO, per oral.

more than three times per week is recommended (Class IIb, level C) [229–234]. Sedentary screen-based entertainment should be limited to 2 hours per day (Class IIb, level C) [17,235]. A reduction in BMI to below the 85th percentile with gradual weight loss (1–2 kg/mo) is recommended for overweight adolescents (BMI in the 85th–95th percentile) and obese children and adolescents (BMI >the 95th percentile) (Class IIb, level C) [236–238].

Pharmacologic treatment

Clinicians can initiate pharmacological treatment in hyper-

tensive children and adolescents with TOD, symptomatic HTN, secondary HTN, CKD, DM, stage 2 HTN, or persistent HTN despite lifestyle modifications for approximately 1 year (Class IIa, level C) [17,18]. The antihypertensive drugs available for children and adolescents are summarized in Table 17.

Pharmacological treatment of primary HTN in children and adolescents should be initiated using angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARB), or calcium channel blockers (Class IIa, level B) [239–257]. Pharmacological treatment of HTN in children and adolescents with CKD, proteinuria, or DM should be initiated using an ACE inhibitor or ARB (Class I, level B) [207,254,258–262]. The indications and contraindications for antihypertensive medications are presented in Table 18.

When administering the maximal dose of any single agent does not achieve the target BP, clinicians should add an agent from a different drug class, avoiding the simultaneous use of an ACE inhibitor and ARB because of the risks of syncope, kidney dysfunction, and hypotension (Class IIa, level D) [263–270].

In children and adolescents with acute severe HTN [BP

Summary of recommendations

Recommendations	Class	Level
BP measurement		
Clinic BP measurement		
· The BP classification is summarized in Table 2.		
· BP should be measured annually in youths aged ≥3 years.	IIa	B
· In youths with kidney disease, DM, solid organ transplant, aortic coarctation (pre- and postoperative), obesity, HTN-related endocrine or genetic diseases, premature births, pre-HTN, and treated HTN, BP should be measured at every clinical visit and at least yearly.	IIa	B
· The BP measurement method is presented in Tables 3 and 4.		
· In stage 2 or resistant stage 1 HTN, BP should be checked in the right arm, left arm, and one leg.	IIb	D
· Oscillometric devices validated for use in youths (https://hyperchildnet.eu/) can be used for BP screening.	IIa	B
· For a confirmative diagnosis of HTN, an auscultatory device should be used.	IIb	B
· When the initial clinic BP is ≥the 90th percentile [approximately, ≥95 + (age × 2) mmHg and/or ≥55 + (age × 1.5) mmHg], remeasure the BP at two additional clinic visits using the auscultatory method, and use the average of these two BP to determine BP status (i.e., normal BP, pre-HTN, or HTN).	IIa	C
ABPM		
· In ABPM, pediatric HTN is defined as an average SBP and/or DBP in 24 hours, daytime, or nighttime that is ≥the 95th percentile but less than the adult criteria (≥130/80 mmHg, ≥135/85 mmHg, and/or ≥120/70 mmHg, respectively).	IIa	C
· Reference BP values for ABPM are presented in Tables 6 and 7.		
· The ABPM method is presented in Table 5.		
· ABPM should be performed before starting antihypertensive drug treatment to exclude WCH.	IIa	B
· In youths with kidney disease, DM, solid organ transplant, aortic coarctation (pre- and postoperative), obesity, HTN-related endocrine or genetic diseases, premature birth, pre-HTN, and treated HTN, ABPM should be performed to detect MH and assess HTN severity and circadian BP patterns.	IIa	B
· In patients with an insufficient BP response to treatment or symptoms of hypotension, ABPM should be performed during antihypertensive drug treatment.	IIa	B
· In patients with CKD, ABPM should be performed periodically.	IIa	B
Home BP monitoring		
· The home BP measurement method is presented in Table 9.		
· Home BP monitoring is recommended as a supportive test for youths receiving antihypertensive treatment after HTN diagnosis or those suspected of having WCH or MH, in addition to clinical BP and ABPM.	IIa	C
· Home BP reference values are presented in Table 10.		
Evaluation		
· In youths diagnosed with HTN, history taking (family history, symptoms of secondary HTN and TOD, and risk factors) and physical examination should be performed to identify secondary causes of HTN and TOD (Tables 11, 12).	I	B

(Continued to the next page)

Recommendations	Class	Level
· In youths diagnosed with HTN, laboratory and imaging tests should be performed to identify secondary causes of HTN and TOD (Table 13).	IIa	C
· Routine testing for MA is not recommended in patients with primary HTN.	IIa	C
· Echocardiography should be performed in youths with HTN to assess cardiac TOD and determine the treatment modality for HTN.	IIa	C
· Repeat echocardiography should be performed at 6–12-month intervals in patients with stage 2 HTN, secondary HTN, persistent HTN despite treatment, or cardiac TOD including LVH and LV dysfunction.	IIa	C
· Definitions of LVH and LV dysfunction on echocardiography are presented in Tables 14 and 15.		
· Routine assessment of arterial stiffness or central BP parameters is not required.	IIa	C
· In patients suspected of having renovascular HTN, kidney Doppler ultrasonography, CT angiography, or MR angiography is appropriate as noninvasive imaging methods.	IIa	C
Treatment		
BP goals		
· SBP and DBP < the 95th percentile [approximately, <100 + (age × 2)/60 + (age × 1.5) mmHg] in general patients.	IIa	C
· SBP and DBP < the 90th percentile [approximately, <95 + (age × 2)/55 + (age × 1.5) mmHg] in patients with DM but not CKD.	IIa	D
· SBP and DBP < the 75th percentile [approximately, <90 + (age × 2)/50 + (age × 1.5) mmHg] in patients with non-proteinuric CKD, regardless of DM.	IIa	A
· SBP and DBP < the 50th percentile [approximately, <85 + (age × 2)/45 + (age × 1.5) mmHg) in patients with proteinuric CKD, regardless of DM.	IIa	A
Lifestyle modifications		
· Youths diagnosed with pre-HTN or HTN should initiate lifestyle modifications including diet, physical activity, and weight control.	I	C
· A diet that is high in fruits, vegetables, low-fat milk products, whole grains, fish, poultry, nuts, and lean red meats and low in sugar, saturated fat, and salt is recommended.	IIb	C
· Moderate-to-vigorous physical activity lasting 30–60 minutes and occurring >3 times per week is recommended.	IIb	C
· Sedentary screen-based entertainment should be limited to 2 hours per day.	IIb	C
· Reducing the BMI to <the 85th percentile with gradual weight loss (1–2 kg/mo) for overweight adolescents (BMI in the 85th–95th percentile) and obese children and adolescents (BMI > the 95th percentile) is recommended.	IIb	C
Pharmacologic treatment		
· Clinicians should initiate pharmacologic treatment in hypertensive patients who have TOD, symptomatic HTN, secondary HTN, CKD, DM, stage 2 HTN, or persistent HTN despite lifestyle modifications for approximately 1 year.	IIa	C
· Antihypertensive drugs available for youths are summarized in Table 17.		
· Indications and contraindications of antihypertensive drugs are presented in Table 18.		
· Pharmacologic treatment in youths with obesity-linked primary HTN should be initiated with an ACE inhibitor, ARB, or calcium channel blocker.	IIa	B
· Pharmacologic treatment of HTN in youths with CKD, proteinuria, or DM should be initiated with an ACE inhibitor or ARB.	I	B
· When the use of the maximal dose of any single agent does not achieve the target BP, another agent from a different drug class should be added, avoiding the simultaneous use of an ACE inhibitor and ARB, because of risks of syncope, kidney dysfunction, and hypotension.	IIa	D
· In acute severe HTN [e.g., BP > the 95th percentile + 30 mmHg, approximately, >130 + (age × 2)/90 + (age × 1.5) mmHg] or life-threatening conditions including neurological, kidney, or cardiac dysfunction, immediate treatment with short-acting antihypertensive medications should be initiated to reduce the BP by <25% of the planned reduction over the first 8 hours, followed by a further gradual reduction over the next 24–48 hours.	IIb	D
· Drugs for acute severe or life-threatening HTN are summarized in Table 19.		
Follow-up		
· During treatment with lifestyle modifications only, patients may be followed up every 3–6 months.	IIb	D
· When clinicians initiate antihypertensive medication or add an additional agent, patients may be followed up every 4–6 weeks until the target BP is achieved, and the interval may then be extended to 3–6 months.	IIb	D

ABPM, ambulatory blood pressure monitoring; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; CKD, chronic kidney disease; DBP, diastolic blood pressure; DM, diabetes mellitus; HTN, hypertension; LVH, left ventricular hypertrophy; MH, masked hypertension; SBP, systolic blood pressure; TOD, target organ damage; WCH, white coat hypertension.

>the 95th percentile + 30 mmHg, approximately, >130 + (age × 2)/90 + (age × 1.5) mmHg] and life-threatening conditions, including neurological, kidney, or cardiac dysfunction, immediate treatment with short-acting antihypertensive medication should be initiated, and the BP should be reduced by <25% of the planned reduction over the first 8 hours, followed by a further gradual reduction over the next 24 to 48 hours (Class IIb, level D) [271–276]. The antihypertensive drugs used for hypertensive emergencies and urgent care are summarized in Table 19.

Follow-up

For children and adolescents with pre-HTN or HTN treated with lifestyle modifications only, the follow-up interval may be 3 to 6 months (Class IIb, level D) [18]. When clinicians initiate antihypertensive medication or add an additional agent in children and adolescents, the follow-up interval may be 4 to 6 weeks until the target BP is achieved and then extended to 3 to 6 months (Class IIb, level D) [18].

Conclusions

HTN is a treatable, independent risk factor for cardiovascular morbidity and mortality. Therefore, early identification of HTN through regular BP measurements during childhood is important. The accurate measurement of clinical BP is an essential first step in the diagnosis of HTN. ABPM and home BP measurements are useful tools for identifying WCH and MH. After HTN is diagnosed, evaluation studies for TOD or secondary causes of HTN should be performed in high-risk patients. Antihypertensive management, including lifestyle modifications and pharmacological treatment, should be initiated immediately after HTN diagnosis. BP goals in the treatment of HTN depend on the associated risk factors, including CKD, DM, and proteinuria. Recommended and contraindicated conditions should be considered when using antihypertensive drugs.

This guideline had the following limitations:

- The definition of HTN in clinical, ambulatory, or home BP measurements was not based on clinical evidence.
- Reference values for clinical, ambulatory, or home BP for the entire pediatric age range of Korean youths do not exist.
- Evaluation studies to assess TOD or exclude secondary causes of HTN remain incomplete.
- BP goals in various hypertensive conditions, such as no risk factors, CKD, DM, or proteinuria, were not entirely based on clinical evidence.
- Evidence on the effectiveness of lifestyle modifications or antihypertensive medications in preventing CVD later in life is lacking.
- Recommendations for treatment, which include lifestyle modifications and antihypertensive medications, were not completely supported by the clinical outcomes related to cardiovascular morbidity and mortality.

Future research should be conducted to address these limitations, and a nationwide childhood cardiovascular cohort study is required.

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Conflicts of interest

All authors have no conflicts of interest to declare.

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Data sharing statement

The data presented in this study are available from the corresponding author upon reasonable request.

Authors' contributions

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