Review Article

Diagnosis and Management of Pediatric Hypertension

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Abstract

Hypertension (HT), a modifiable risk factor in adults, is a major risk factor for cardiovascular disease. Blood pressure (BP) originates in childhood and tracks to adulthood and hence very important to diagnose and appropriately manage childhood HT for healthy adulthood. In this article, we have attempted to answer the following questions: (1) What is HT in children and adolescents? (2) When does it begin? (3) What initiates it? (4) Who is susceptible? (5) What can be done during childhood to prevent consequences of HT during adult life? The updates of the recent American Academy of Pediatrics 2017 guidelines on HT for children have been included with importance to the prevention of HT through healthy lifestyle and vigilant screening including 24 h ambulatory BP monitoring.

Key words: Adult hypertension, childhood blood pressure, obesity, pediatric hypertension, preterm, prevention, risk factors

Childhood Hypertension (Ht): A Window to Adult Ht

The first report on pediatric HT by the National Heart, Lung, and Blood Institute, published in 1977, declared that "detection and management of HT in children and the precursors of HT in adults are the next major frontier.[1] HT in adults is a major modifiable risk factor for cardiovascular disease and is often associated with other cardiovascular risk factors, including impaired glucose tolerance, obesity, and dyslipidemia.[2] The standard approach of treating high BP in middle and old age can help mitigate these risks, but considerable burden remains. An approach that identifies those at greatest risk of developing high BP much earlier in life could permit more effective risk reduction through earlier, age-appropriate prevention, and intervention strategies. This made Ellin Lieberman post a series of questions for pediatricians way back in 1974.[3] (1) What is HT in children and adolescents? (2) When does it begin? (3) What initiates it? (4) Who is susceptible? (5) What can be done during childhood to prevent consequences of HT during adult life? In this review, an attempt has been made to answer by presenting information for diagnosis, management, and preventive aspects of pediatric HT.

What is HT in Children and Adolescents?

HT is defined as average of three clinics measured systolic BP (SBP) and/or diastolic BP (DBP) ≥95th percentile on the basis of age, sex, and height percentiles. The classification of HT as per the recent American Academy of Pediatrics (AAP) 2017 Guidelines is shown in Table 1.[4] and Fig 1 shows the procedure for BP measurement and classification in children

In the recent AAP Guidelines of 2017, the normative data are based on the auscultatory findings obtained from 50,000 normal children and adolescence and carries a number of modifications. (1) The term "Prehypertension" has been replaced by "elevated BP" to be consistent with adult American Heart Association/American College of Cardiology guideline. This change in terminology also conveys that BP is already abnormally elevated and the importance of lifestyle modifications to prevent HT. (2) Definitions that categorize BP values were modified into two age groups, for children and adolescents. (3) The staging criteria have been revised for Stage 1 and Stage 2 HT. (4) The classification of adolescent HT is aligned with adult guidelines for the detection of chronic elevated BP. (5) Unlike the previous guidelines, the BP tables are based on BPs from normal-weight children. This decision was taken as overweight and obesity

Table 1: Classification of HT in children and adolescents[4]

For children aged 1–13 years	For children aged ≥13 years
Normal BP: <90th Percentile	Normal BP: <120/<80 mmHg
Elevated BP: ≥90th percentile to <95th percentile or 120/80 mmHg to <95th percentile (whichever is lower)	Elevated BP: 120/<80–129/<80 mmHg
Stage 1 HT: 95th percentile–< 95th percentile+12 mmHg, or 130/80–139/89 mmHg (whichever is lower)	Stage 1 HT: 130/80–139/89 mmHg
Stage 2 HT: ≥95th percentile+12 mmHg, or≥140/90 mmHg (whichever is lower)	Stage 2 HT: ≥140/90 mmHg

HT: Hypertension

has a strong association for HT. (6) Emphasis on use of 24-h ambulatory BP monitoring to confirm the diagnosis of HT. (7) Added is a simplified screening table for ease of use in the consulting room [Table 2]. (8) The height centiles and the corresponding height in inches and centimeter have been included in the BP chart. Hence, BP staging can be directly plotted in the BP charts compared with the earlier guideline.

Primary and secondary HT in children
Primary HT in childhood was thought previously to be rare. Secondary HT is more common in adolescents than in infants, children, and preadolescents. At present, as with adults, children and adolescents with mild-to-moderate HT have primary HT in which a cause is not identifiable. The worldwide childhood obesity epidemic has had a profound impact on the frequency of HT and other obesity-related conditions with the result that primary HT should now be considered as a common health problem in the young.

Gupta-Malhotra *et al.* evaluated the etiology of HT among 423 children from a pediatric HT clinic. A total of 275 children were diagnosed with HT of whom 156 (57%) had an identifiable secondary cause; 119 (43%) had primary HT.[5] In a cross-sectional study a total of 1085 apparently healthy student, aged between 11 and 17 years from rural and urban schools in hills of northern India, were examined using standard methods. After two evaluations, HT was identified in 62 (5.9%) children and pre-HT in 130 (12.3%). Urban and rural children had comparable rates of high BP (HT and pre-HT). Rates of elevated BP were significantly higher (46.5% vs. 17%, *P* < 0.001) among those with high body mass index (BMI) (overweight and obese) compared to those with normal BMI. In conclusion, nearly 20% of the school children had high BPs.[6]

In a review publication from a developed country primary, HT was identified in 16% of cases and 70% had secondary HT.[7] In a study of 351 hypertensive children and adolescents it was observed that the younger children (<6 years of age) had higher secondary HT, were less obese, and had higher diastolic BP as compared to children in mid-childhood (age 6–<12 years) and adolescents (age 12–<17 years). Thus, secondary HT is more likely to be detected in non-obese younger children with higher BP, whereas, primary HT is more commonly found in late childhood and adolescence and is associated with overweight/obesity and modest BP elevations.[8]

General characteristics of children with primary HT include older age (≥6 years), positive family history of HT, and

Table 2: Screening BP values requiring further evaluation[4]

| Age (years)	Boys (BP, mmHg)		Girls (BP, mmHg)	
	SBP	DBP	SBP	DBP
1	98	52	98	54
2	100	55	101	58
3	101	58	102	60
4	102	60	103	62
5	103	63	104	64
6	105	66	105	67
7	106	68	106	68
8	107	69	107	69
9	107	70	108	71
10	108	72	109	72
11	110	74	111	74
12	113	75	114	75
≥13	120	80	120	80

SBP: Systolic blood pressure, DBP: Diastolic blood pressure

overweight, and/or obesity. DBP elevation appears to be more predictive of secondary HT, whereas systolic HT appears to be more predictive of primary HT.[8]

Further, the data published documents a progressive increase in the frequency of primary HT at the varying period of time from different centers as shown in Figure 2.[9] Common causes of HT in children include renal and renovascular disease, coarctation of the aorta, and endocrine disease.

HT in neonates
HT is detected in 1–2.5% of all neonates admitted to the neonatal intensive care unit (NICU). In neonates, HT is defined as persistent SBP and/or DBP that exceeds the 95th percentile for postconceptional age.[10] Most hypertensive newborns are asymptomatic, and diagnosis is made by routine BP measurement. BP is measured by an oscillometric device after an appropriate sized BP cuff is positioned on the right upper arm and preferably 1.5 h after feed or medical intervention when the infant is in quiet state and either in prone or supine position. It is challenging to establish normative values for neonatal BP, especially in pre-term infants, due to the effects of gestational age and maturation on BP values. The 1987 "Report of the Second Task Force on BP control in Children" published curves of normative BP values in older infants up to 1 year of age which is currently being used.[11]
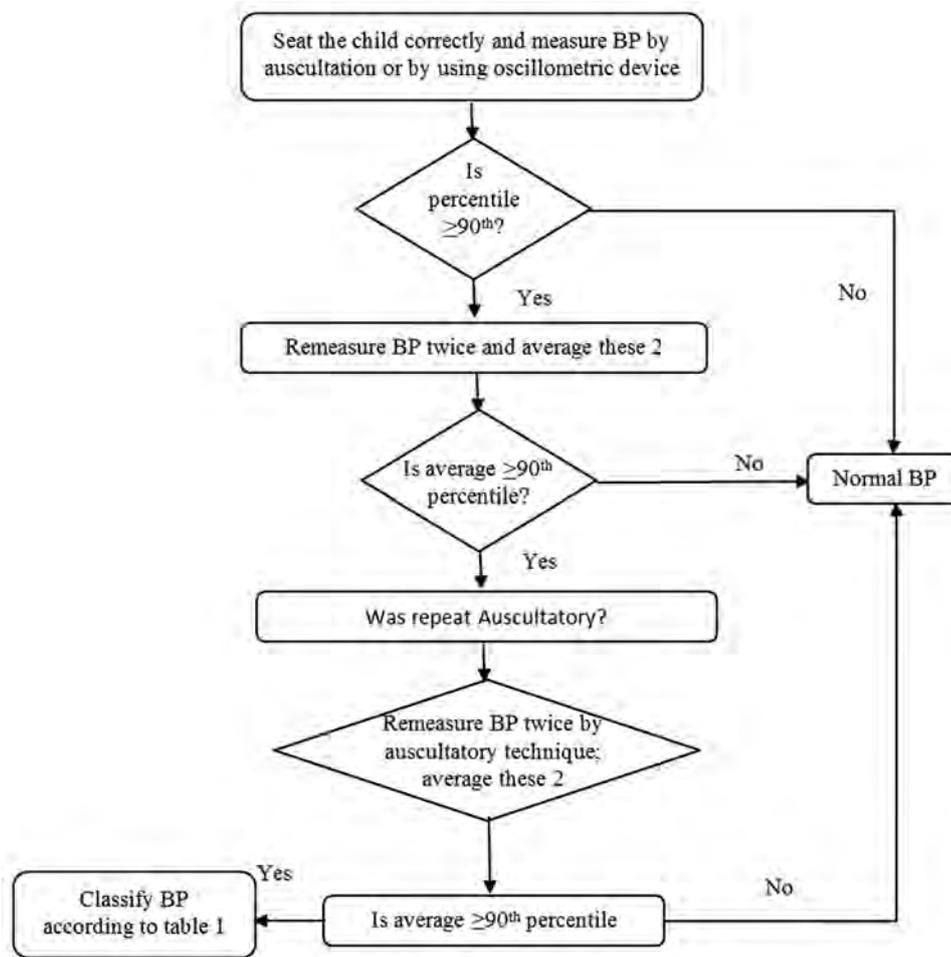
Figure 1: Flow chart reflecting the procedure for blood pressure measurement in children and classification[4]

Common causes of neonatal HT are umbilical artery catheter-associated thromboembolism, bronchopulmonary dysplasia, intraventricular hemorrhage, patent ductus arteriosus, and congenital renal structural malformation, renovascular diseases, acute kidney injury, and certain medications.[10]

Once the diagnosis of neonatal HT is confirmed, an evaluation is performed to identify the underlying cause of HT as in children, which may potentially be corrected. Angiotensin-converting enzyme inhibitors (ACE I) or angiotensin receptor blockers (ARB) are not recommended in neonates in view of potential side effects. Calcium channel blockers, vasodilators, and beta-blockers are used in the treatment of neonatal HT.

When Does it Begin?

Childhood BP originates in childhood and tracks to adulthood. In a review study, it has been reported that childhood HT ranges from 0.5% to 11.7% in Indian children.[12] Theodore *et al.* followed 975 subjects to identify childhood to early-midlife SBP trajectories. The BP data at ages 7, 11, 18, 26, 32, and 38 years from a longitudinal, representative birth cohort study was used
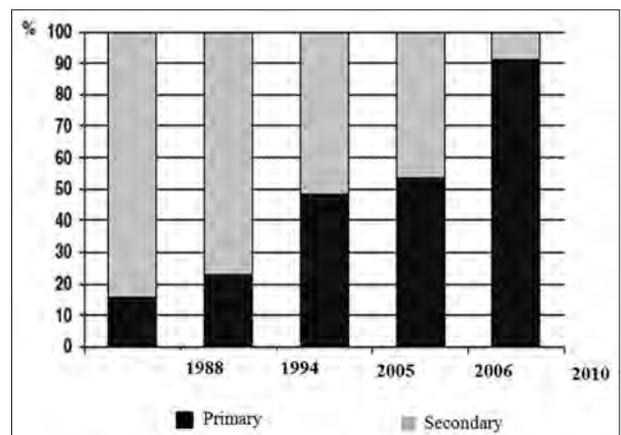
Figure 2: Bar diagram showing an increasing trend of primary hypertension in American youths[9]

to identify four distinct trajectory groups. Figure 3 shows the plotted predicted trajectory lines for each of the four groups which shows that each trajectory follows the same path from childhood to adulthood thus confirming that childhood BP
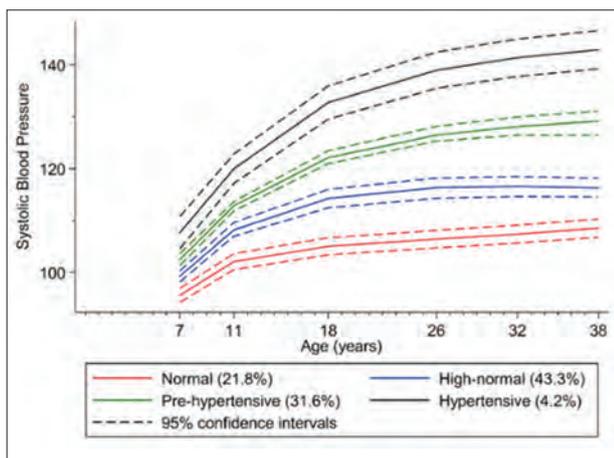
Figure 3: Blood pressure trajectory from childhood to early-mid adulthood[13]

tracks to adulthood. Prehypertensive and hypertensive trajectory groups had worse cardiovascular outcomes by early midlife. They concluded that harmful BP trajectories are identifiable in childhood, associated with both antecedent and modifiable risk factors over time, and predict adult cardiovascular disease risk. The means for all four groups significantly differed from each other at all ages, beginning at the age of 7 years. The normal group and high-normal group had mean BP in the normal SBP range (90–120 mmHg) as in adults. The prehypertensive group had a mean SBP within the prehypertensive range (120–139 mmHg) throughout adulthood. The hypertensive group had the highest mean BP at the age of 7 years and displayed the steepest rise in BP with mean BP in the hypertensive range at the age of 38 years (≥140 mmHg). Early detection and subsequent targeted prevention and intervention may reduce the life course burden associated with higher BP.[13]

What Initiates Pediatric HT?

Low birth weight (LBW)
Brenner *et al.* postulated that developmental programming in the intrauterine environment influences BP during adult life.[14] Barker *et al.* analyzed two large samples of 9921 children and 3259 adults in Britain and found that SBP was inversely related to birth weight. The association was independent of gestational age, and therefore, HT was attributed to reduced fetal growth.[15] Individuals with nephron numbers on the lower side of the spectrum are those at higher risk of HT and kidney disease.[16] Nephron numbers increase in proportion to birth weight and gestational age and vice versa in individual's born as LBW and prematurely.[17]

Prematurity
Prematurity increases the risk of HT through decreased glomerulogenesis independent of birth weight. A meta-analysis of 10 studies including 3083 individuals from eight countries reported the association of prematurity with adolescent or adult

BP (measured at an average age of 18 years). Those who were born premature had modestly but significantly higher SBP (by 2.5 mmHg), regardless of weight.[18]

Obesity
This is one of the major contributing factors in recent times to HT and the reason for the shift to an era of primary HT in children. Today, many tiniest neonates leave NICU without apparent morbidity, and adverse effects were marked among those who became overweight or obese.[19] In India, Patil *et al.* screened 1486 adolescents and found the prevalence of overweight and obesity to be 20% and 16%, respectively. The prevalence of pre-HT was noted in 7.5% and HT in 5.4% children.[20] Evidence shows that rapid "catch-up" growth with a body weight higher than expected leads to the development of high BP, insulin resistance, cardiovascular, and renal risk. Catch-up growth is necessary as it improves child survival, stunting, and malnutrition but not to an extent of obesity.

Sleep disordered breathing
Researchers in numerous studies have identified an association between sleep-disordered breathing and HT in the pediatric population.[21]

Other risk factors
Family history of high BP, male sex, high salt intake, first born added to LBW were associated with hypertensive group. Higher body mass index and cigarette smoking resulted in increasing BP across trajectories, particularly for the higher BP groups. Maternal malnutrition, gestational diabetes, gestational HT, maternal overweight and obesity, preeclampsia indirectly, through LBW, and prematurity, contribute to the development of HT. Congenital anomalies of the kidneys and urinary tract, and neonatal AKI, perinatal exposure to nephrotoxic drugs and primary renal disease, acquired and hereditary, contribute to renal injury, and HT.

Genetics of HT is complex and many genes react to different environmental stimuli and contribute to BP. About 30–50% of the variance of BP readings are attributable to genetic heritability and about 50% to environmental factors. Genetic studies have identified (a) specific enzymes, channels, and receptors implicating sodium handling in the regulation of BP, (b) genes involved with the renin-angiotensin-aldosterone system controlling BP and salt-water homeostasis, (c) proteins in hormonal regulation of BP, and (d) regulation of vascular tone through endothelins and their receptors.[22]

When to Measure BP and Who Are Susceptible to HT?

BP is measured annually in healthy children more than 3 years of age. In children <3 years of age BP is measured and at every health-care encounter in those with the history of (a) prematurity (<32 weeks) (b) very LBW, (c) neonates requiring NICU care, (d) overweight, obesity or diabetes, (e) associated renal, cardiac or neurological ailment, (f) systemic illness leading

to HT, (g) solid organ transplant, and (h) treatment with drugs known to cause HT.[4]

What can be Done During Childhood To PreventHT During Adult Life?

In 2013, the US Preventive Services Task Force presented a controversial statement that "the current evidence is insufficient to assess the balance of benefits and harms of screening for primary HT in asymptomatic children and adolescents to prevent subsequent cardiovascular disease in childhood or adulthood."[23]

The International Childhood Cardiovascular Cohort Consortium has, however, documented evidence that pediatric HT is predictive of adult BP and has a significant impact on the heart and blood vessels.[24] Left ventricular hypertrophy (LVH) has been identified as an important side effect with HT in children. It is estimated that 8–41% of hypertensive children have left ventricular mass of >95th percentile, adjusted for age, sex, and height, and roughly 10–15.5% of children have values >51 g/m^2, a level known to be associated with significant cardiovascular morbidity and mortality in adults.[25] HT during childhood has been shown to be associated with early markers of cardiovascular disease, including carotid intima-media thickness, arterial compliance, atherosclerosis, and diastolic dysfunction.[26,27] Likewise, retinal arteriolar narrowing and microalbuminuria have been documented.

Analysis of the National Childhood BP database found that 7% of adolescents with elevated BP per year progressed to true hypertensive BP levels.[28] Therefore, efforts should be made to prevent progression to sustained HT through: (a) Good antenatal care, (b) prevention of obesity, (c) vigilant screening for HT in high-risk children, and (e) prevent or control HT and target organ damage through healthy lifestyle and pharmacotherapy.

Good antenatal care
It is evident that kidney diseases including HT in adulthood often springs from childhood legacy. The care should start from the womb. Decreasing teenage pregnancy, empowering and educating girls and women, reducing maternal infections and malnutrition, appropriate antenatal care can reduce the risk of LBW, small for gestational age, preterm birth, pre-eclampsia, gestational diabetes mellitus, maternal and childhood obesity and hold promise of a positive impact on the renal health of future generations.[29]

Prevention and management of obesity
Weight loss is particularly important for children with obesity-related HT because it addresses the underlying etiology, improves comorbidities and reduces sympathetic overactivation, and leading to lowering of BP. Guidelines recommend a staged approach to obesity treatment, with weight loss recommended for children 6years of age and above when BMI is in the obese category and weight maintenance for growing children when BMI is in the overweight category.[30]

Avoidance of sugar-sweetened beverages leads to weight loss among children. Sodium intake to <1.5 g/day has a significant impact on BP among children and adolescents who are overweight/obese.

A review of 9 studies of the physical activity interventions in children and adolescents with obesity suggested that 40 min of moderate to vigorous, aerobic physical activity at least 3–5 days per week improved SBP by an average of 6.6 mm Hg and prevented vascular dysfunction.[31] The 2017 AAP Guideline recommends moderate to vigorous physical activity of 30–60 min/session for 3–5 days/week to control HT in children and adolescents with elevated BP. ACE I or ARB is recommended as initial agents in the treatment of obesity-related HT. The added benefit is being able to target pathways leading to elevated BP, and beneficial effects on comorbidities, diabetes, and dyslipidemia.

Vigilant screening
In addition to screening children who are susceptible to HT as above, ambulatory BP monitoring (ABPM) is a recent tool being used in the pediatric population for confirming HT. According to the 2017 AAP guidelines, ABPM is recommended in children >5 years with the following indications: (a) White-coat HT (WCH), (b) masked HT, (c) to confirm diagnosis and before initiating pharmacologic therapy, (d) assess BP control in children on antihypertensive, and (f) follow-up of secondary HT. The diagnosis of WCH is relevant due to risk for cardiovascular damage. Current knowledge does not recommend treatment. Pharmacological therapy is advised in the presence of LVH, changes in intimal/medial wall thickness of carotid arteries and microalbuminuria. Mark *et al.* did a cross-sectional analysis of BP and cardiac structure in a large population of children with chronic kidney disease (CKD) as a part of the observational CKD cohort study. On the basis of the combination of ambulatory and casual BP assessment (n = 198), 38% of children had masked HT and 18% had confirmed HT. If ABPM was not used, then 38% of the children would have missed the diagnosis of HT. LVH was more common in children with either confirmed (34%) or masked HT (20%). In conclusion, casual BP measurements alone are insufficient, and ABPM should be performed routinely.[32]

Lifestyle modification
The Dietary Approaches to Stop Hypertension (DASH) diet with specific elements is the recommended dietary strategy for HT. These elements include a diet that is high in fruits, vegetables, low-fat milk products, whole grains, fish, poultry, nuts, and lean red meats; it also includes a limited intake of sugar and sweet along with lower sodium intake. The current recommendation for salt intake for normal children is <1.2 g/day for children aged 4–8 years and <2.5 g/day for 8–16 years.

Pharmacologic management
In both primary and secondary HT, if lifestyle modification is unsuccessful in BP control, it is necessary to initiate pharmacologic therapy along with lifestyle modification. The frequency of repeat BP measurement and pharmacologic therapy initiation is shown in Table 3 and Figure 4.[33]

Evidence has emerged that markers of target organ damage, such as increased left ventricular mass, can be detected among some children with BP >90th percentile (or >120/80 mmHg) but <95th percentile. The goals of therapy for the treatment of HT are for achieving a BP level that reduces the risk for target organ damage. In the recent AAP 2017 guidelines, optimal BP level to be achieved with the treatment of childhood HT is <90th percentile or <130/80 in >13 years of age. Children with CKD, HT should be treated to lower 24-h MAP to <50th percentile by ABPM. Alternately guideline recommends, in children with non-dialysis CKD particularly those with proteinuria, BP to be lowered to achieve systolic and diastolic

Table 3: Frequency of BP measurement and timing of pharmacologic therapy initiation[33]

Types of HT	Stage of HT	BP measurement frequency	Pharmacologic treatment
Primary	Elevated BP	Initiate lifestyle changes Repeat in 6–12 months	After 12 months if not controlled with lifestyle changes
	Stage I	Initiate lifestyle changes Recheck in 1–2 weeks (twice) Sooner if the patient is symptomatic Repeat after 3 months and refer to a specialist if high	Symptomatic After 3 months if lifestyle changes fail in asymptomatic children
	Stage II	Initiate lifestyle changes If asymptomatic, repeat in week and evaluate or refer to a specialist	Symptomatic or acute severe HT If repeat values are high
Secondary		Initiate lifestyle changes	Initiate treatment

HT: Hypertension, BP: Blood pressure
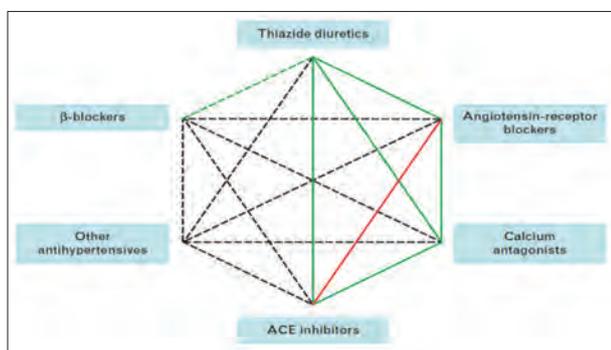
Figure 4: Recommended combination of antihypertensives.[33] Green/continuous: Preferred. Green/dashed: Useful (with some limitations). Black/dashed: Possible but less well tested. Red/continuous: Not recommended. Only dihydropyridines to be combined with beta-blockers. Thiazides and beta-blockers increase risk of new onset diabetes mellitus. angiotensin-converting enzyme inhibitors and angiotensin receptor blockers combination discouraged

readings less than or equal to the 50th percentile for age, sex, and height.[34] First line pharmacologic agents to control BP, in children and adolescents are Angiotensin converting enzyme inhibitor (ACE I) and angiotensin receptor blocker (ARB), long-acting calcium channel blockers or thiazide. Other antihypertensive medications should be reserved for children who fail to achieve adequate BP control with two or more of these preferred agents.[35]

In conclusion, the current focus is early identification of HT in asymptomatic, healthy children and adolescents and those with secondary HT. Barriers for early identification in clinical practice are poor knowledge of normal BP range, lack of awareness of previous BP readings and the need to synthesize multiple BP readings over time to make a diagnosis of HT. The current scenario is around the epidemic of obesity in children, obesity-related HT, and primary HT. The increasing survival of newborns from pregnancy, prenatal and perinatal complications due to improved maternal and NICU care would indicate an increase in childhood HT and its consequences in adulthood. In future, this form of non-communicable disease would be dominant in pediatrics. Hence, physicians who care for children should have before them Table 2 (screening BP values requiring further evaluation of AAP Guidelines 2017). Oscillometric devices may be used for BP screening in children and adolescents. The update recommends increased use of ABPM for diagnosis and for assessing therapeutic response. Treatment goal with non-pharmacologic and pharmacologic therapy should be a reduction in SBP and DBP to 90th percentile for children and <130/80 for adolescents of 13 years and above. Prevention of predisposing causes and early identification of elevated BP in children would serve the foundation for battling the impending storm of HT in children.

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