Hypertension in children: new trends and challenges

Janusz FEBER and Maheen AHMED
Division of Nephrology, Department of Pediatrics, Children's Hospital of Eastern Ontario, 401 Smyth Road, Ottawa, Ontario, Canada K1H 8L1

ABSTRACT
Childhood HTN (hypertension) has become a widely investigated topic within the last decade due to its increasing prevalence. In the present review, we examine new developments and trends that have significantly contributed to aetiology, diagnosis, evaluation and management of childhood HTN. Many recent reports document an increasing prevalence of HTN, mainly essential HTN, in children worldwide. This is probably related to the increase of childhood obesity, although obesity is not the only factor. Evidence has been accumulating to suggest a rather complex interplay between obesity, uric acid level, dietary sodium intake, inflammation, inheritance and other factors, which lead to increased risk of developing HTN in childhood and adulthood. The detection and monitoring of HTN has significantly improved with the use of ABPM (ambulatory blood pressure monitoring), which allows not only for a more accurate classification and staging of HTN, but also for the calculation of more sophisticated parameters such as the AASI (ambulatory arterial stiffness index). Measurement of arterial stiffness enables assessment of arterial dysfunction, which may precede structural vascular changes evaluated by carotid intima media thickness. Sustained HTN eventually leads to end-organ damage [LVH (left ventricular hypertrophy), central nervous system], which in turn increases the risk of cardiovascular morbidity and mortality. New developments in childhood HTN, as outlined in the present review, will hopefully contribute to better screening and management of HTN in children.

INTRODUCTION
Arterial HTN (hypertension) in children has become an important health issue due to its rising prevalence and associated sequelae. Historically, HTN in children, particularly in the younger age group, was thought to be rare and secondary in origin [1]. However, recent reports suggest an increased prevalence of childhood HTN, particularly essential, also known as primary HTN [2].

HTN in children, regardless of the aetiology, results in significant end-organ damage [3,4], which in turn may lead to significant cardiovascular morbidity and mortality later on in life. An early diagnosis and proper management of childhood HTN may therefore prevent HTN-related complications in adulthood.

Despite increased awareness of the importance and severity of childhood HTN among paediatricians and health care workers, a significant proportion of children with HTN are still not diagnosed [5] and thus not treated.

The aim of the present review is to discuss new trends and developments in childhood HTN, which may help to improve the diagnostic and therapeutic interventions in children with suspected or confirmed HTN. The therapy of HTN was recently summarized by Flynn [6,7]; it is therefore not discussed in detail in the present review.

Key words: blood pressure, children, end-organ damage, hypertension, obesity, prevalence.
Abbreviations: AASI, ambulatory arterial stiffness index; ABPM, ambulatory blood pressure monitoring; BMI, body mass index; BP, blood pressure; CIMT, carotid intima media thickness; CRP, C-reactive protein; DBP, diastolic blood pressure; HTN, hypertension; ICAM-1, intercellular cell adhesion molecule-1; IL, interleukin; LVH, left ventricular hypertrophy; LVMi, left ventricular mass index; PWV, pulse wave velocity; SBP, systolic blood pressure.
Correspondence: Dr Janusz Feber (email jfeber@cheo.on.ca).
Table 1 Worldwide prevalence of abnormal BP in children
The results are from published population studies, in alphabetical order by country. N/A., not available.

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DEFINITION OF HTN

HTN in children is defined as average SBP [systolic BP (blood pressure)] and/or DBP (diastolic BP) that is $\geq 95$th percentile for gender, age and height on $\geq 3$ occasions [8]. Prehypertension in children is defined as average SBP or DBP levels that are $\geq 90$th percentile but <95th percentile or if the BP exceeds 120/80 mmHg even if <90th percentile up to <95th percentile [9]. This is consistent with the definition in adults in whom prehypertension is defined as BP level $\geq 120/80$ mmHg [10]. Stage 1 HTN in children is then defined as SBP or DBP $\geq 95$th percentile up to 99th percentile plus 5 mmHg. In stage 2 HTN, the SBP or DBP exceeds the 99th percentile plus 5 mmHg [8]. This definition of the BP threshold has gained worldwide support and has been used in the vast majority of studies on paediatric HTN.

However, it is further recommended that the elevated BP (one or two SBP and/or DBP readings at or above the 90th percentile for age, gender and height) is confirmed on repeated visits before characterizing a child as having HTN. Measures obtained by oscillometric devices that exceed the 90th percentile should be repeated by auscultation [8]. The number of BP measurements (one compared with repeated measurements) and methodology (auscultatory compared with oscillometric) varies significantly in reported studies, which makes the comparison/pooling of results rather difficult. In clinical studies, the diagnosis of HTN requires repeated measurements, whereas epidemiological studies are mostly based on one or few BP measurements on one occasion.

SCREENING FOR HTN

Given the complexity of definition of paediatric HTN based on a child’s gender, age and height percentile and the need for repeated measurements as discussed above, it may not be easy to use in routine clinical practice or screening. Kaelber and Pickett [11] developed a simplified table, which has only one threshold value of abnormal SBP and DBP, by gender, for each year of life (ages 3 to 18). This is very useful when BP is measured in a setting when the height percentile may not be easily obtainable. Therefore it may be used as a screening tool that quickly and easily identifies children and adolescents whose BP readings merit further evaluation.

INCREASED PREVALENCE OF CHILDHOOD HTN

The worldwide prevalence of abnormal BP (elevated BPs on less than three measurements on different occasions, prehypertension or HTN) in children is shown in Table 1 [12–22]. The exact prevalence of childhood HTN is difficult to assess, as the results vary significantly depending on age, selection of children for the survey (population compared with school-based survey), BP measurement methods (auscultatory compared with oscillometric), number of BP readings, number of office visits and ethnic differences around the world. It is therefore difficult to compare the data from different studies from around the world.

Given the BP variability and difficulties with the BP measurement in children, the reported numbers...
for paediatric HTN may be lower with repeated BP measurements on more than one office visit [23–25]. Recently, Moore et al. [22] reported abnormal BP in the hypertensive range in 13.8% of the students at first measurement, in 4.5% at second measurement, but only 2.3% had sustained abnormal BP measurement in the hypertensive range at third measurement.

This implies that elevated BP measurements found at a single screening are not diagnostic of sustained HTN in children but are useful for examining population trends, as most epidemiological studies use the single BP screening approach to calculate the prevalence of HTN. Therefore the prevalence of sustained, ‘true’ HTN, confirmed with three office visits, may be, in fact, lower than that found in the population studies. However, a significant proportion of hypertensive children may still remain undiagnosed as suggested by Hansen et al. [5].

**CHANGES IN PREVALENCE OVER TIME**

Regardless of the accuracy of the prevalence estimation, childhood HTN is on the rise [2,26,27]. Elevated BPs in the prehypertensive range (SBP or DBP ≥ 90th percentile but <95th percentile or BP ≥ 120/80 mmHg but below 95th percentile) and elevated BPs in the hypertensive range (SBP or DBP ≥ 95th percentile) have increased by 2.3% ($P = 0.0003$) and 1% ($P = 0.17$) respectively, between 1988 and 1999 [2]. To compare historical groups (trend analysis), all available BP measurements were considered, and cut-off points from the Task Force [8] were applied. Additionally, an ethnic and gender gap appeared; non-Hispanic blacks and Mexican Americans had a greater prevalence of elevated BPs in the hypertensive and prehypertensive range than non-Hispanic whites, with males having a greater prevalence over females [2]. The effect of gender on HTN prevalence remains, however, unclear, as the most recent study showed that controlling all covariates including BMI, BP has increased among girls but decreased among adolescent boys aged 13–17 during 2003–2006 when compared with 1988–1994 [26].

The increase in elevated BPs in the hypertensive range was attenuated by ≈44% when standardized by BMI (body mass index) or waist circumference distribution suggesting that the recent increase in the prevalence of elevated BP in children is explained, at least in part, by the increase in BMI and waist circumference [2]. The other potential culprits such as sodium intake and physical inactivity were not consistently measured over time. Hence, their potential impact on the trend of HTN cannot be evaluated.

These recent reports contradict earlier suggestions by Chiolero et al. [28,29] that the prevalence of childhood HTN has remained stable over time. Similarly, Watkins et al. [30] reported substantial decreases in SBP and DBP over the past decade in adolescents from Northern Ireland.

Therefore we may witness some ethnic/regional differences in secular trends of HTN and its association with obesity. HTN and obesity in children may be on the rise in the Western world, but not necessarily so in other countries. Further studies are needed to analyse these trends and differences.

**AETIOLOGY/RISK FACTORS OF HTN**

**Obesity**

Obesity, defined as a BMI at or above the 95th percentile for children of the same age and sex, has become an epidemic that affects all age groups including children. The recent report by the CDC (Center for Disease Control) indicates that obesity among low-income, preschool-aged children, increased steadily from 12.4% in 1998 to 14.5% in 2003 and remained at 14.6% in 2008 [31]. Similar numbers were reported from Canada, with obesity prevalence in children more than doubling from 1981 to 1996, from 5% to 13.5% for boys and 11.8% for girls [32]. Increased prevalence of childhood obesity has been reported from Mexico [33], the Netherlands [34], Hungary [35], Turkey [36] and Norway [37]. Interestingly, only a slight increase in childhood obesity was reported from Australia, where the prevalence rates have settled around 21–25% for overweight and obesity together, and 5–6% for obesity alone [38]. Furthermore, a recent study from Switzerland shows that over the past 5 years, adiposity has decreased in Swiss children [39].

Regardless of the actual prevalence of childhood obesity in a given country, it seems logical that there is a link between obesity and HTN. In fact, the association of higher BP trends paralleling the rise in obesity has been documented in numerous reports [13,23,26,33,40–44]. Obesity was a risk factor for girls and boys with elevated BP (adjusted odds ratio = 4.01 and 4.33 respectively) [22]. Similarly, Rebelo et al. [45] reported that overweight boys had an odds ratio for being hypertensive of 2.26% and 3.36%, corresponding with values for girls at 1.58% and 2.31%.

On the other hand, the association between obesity and abnormal BP has been questioned by Chiolero et al. [28] who, in combined data from published BP surveys, demonstrated that BP levels were, in fact, only moderately increasing or even decreasing over time despite substantial increases in childhood obesity in the same surveys. It is, however, difficult to assess the true association between obesity and BP trends from these population-based surveys due to the presence of numerous variables.

It seems that obesity may not be the only factor contributing to the aetiology and severity of HTN. Children at risk for obesity, deemed to have mostly...
primary HTN, may still suffer from secondary HTN as recently documented in a multicentre study of the Midwest Pediatric Nephrology Consortium [46]. This study emphasizes the importance of following current recommendations for evaluation of secondary HTN [8] even in obese patients in whom primary HTN is suspected.

Obesity combined with other factors such as low birth weight may significantly increase the risk of development of HTN [47]. Low birth weight children who became obese had the highest SBP values. Moreover, obesity and HTN may be preprogrammed prenatally as suggested by Filler et al. [48], who showed that higher prepregnancy BMI increases the risk for increased BMI z score and BP z score in children.

It seems that several factors including race/ethnicity, elevated SBP, obesity, low birth weight and inflammation interplay in the development of HTN.

Inflammation
Growing evidence indicates that HTN may be a proinflammatory condition [42,49,50]. Elevated serum concentrations of CRP (C-reactive protein), IL (interleukin)-6, IL-1β and ICAM-1 (intercellular cell adhesion molecule-1) correlated significantly with the ambulatory BP in obese children and adolescents suggesting that low-grade inflammation may play a role in the modulation of arterial BP relatively early in life [51]. Similarly, subjects with essential HTN had higher levels of 8-iso-prostaglandin (2α), CRP, ICAM-1 and VCAM-1 (vascular adhesion molecule-1), and TNF (tumour necrosis factor)-α, when compared with control subjects [52].

Of all the plasma markers of vascular inflammation, CRP has been the most intensively investigated in children. CRP has been also related to increased intima media thickness [53], increased arterial stiffness [54], LVH (left ventricular hypertrophy) [55], BP variability [56] and cardiovascular risk in obese children [57].

The inflammatory markers may also be increased because of obesity alone, independent of HTN [58]. However, the highest concentrations of these molecules were found in obese children with co-existing HTN [49].

It appears that inflammation may play a significant role in the development of HTN, especially in obese children. Furthermore, CRP is elevated in the offspring of parents with essential HTN [59]. It may be therefore speculated that a proinflammatory condition in hypertensive mothers may lead to inflammation-induced HTN in their offspring.

Uric acid
Hyperuricaemia is an independent predictor of arterial stiffness in hypertensive patients [60] and may modestly increase the risks of both stroke incidence and mortality [61]. The mechanisms by which HTN is induced by hyperuricaemia are still unclear, but endothelial dysfunction, induction of sodium sensitivity, stimulation of the renin–angiotensin system and pro-inflammatory markers, vascular smooth muscle proliferation and alteration of scavengers of reactive oxygen species have been proposed [62].

Several reports have demonstrated hyperuricaemia in hypertensive children [63–65] as well as a link between hyperuricaemia and LVH [4]. Interestingly, in a recent randomized trial, Feig et al. [66] demonstrated that in adolescents with newly diagnosed essential HTN, allopurinol treatment alone leads to a substantial decrease in BP compared with placebo. This may represent a new potential therapeutic approach to be confirmed by long-term studies.

Dietary salt intake
Acute and chronic elevations of salt intake produce HTN and direct target-organ damage such as myocardial hypertrophy, vascular hypertrophy and fibrosis in animal studies [67,68]. In humans, sodium reduction, previously shown to lower BP, may also reduce long-term risk of cardiovascular events [69]. Even acute salt loading impaired vascular endothelial function, left ventricular mechanical relaxation and electric repolarization in young healthy normotensives [70].

There is a direct relation between salt intake and cardiovascular risk, and a reduction in salt intake is associated with a reduction in cardiovascular mortality in the population [71].

The effect of salt intake may begin early in life. Babies who were on a low-sodium diet had lower SBP and DBP compared with those who received a normal sodium-containing diet when examined several years later [72]. There is also emerging evidence that a reduced renal mass in growth-restricted children poses a risk for lower renal function and for increased salt sensitivity [73], which may predispose such individuals for the development of HTN later in life.

Therefore it seems that salt reduction is beneficial throughout an individual’s life regardless of age. In addition, there is a significant association between salt intake and total fluid intake, which includes sugar-sweetened soft drinks [74]. A reduction in salt intake could, therefore, play a role in helping to reduce childhood obesity and cardiovascular burden.

BP measurement
Office BP
Office BP measurement has been the preferred method of BP measurement in children. The NHBPEP (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents) published normative BP values based on
Age and height [8]. These normative values were obtained from auscultatory measurements, yet in clinical practice, many centres/physicians use automated, i.e. oscillometric devices for BP measurement, for which the normative auscultatory reference values may not apply. However, the oscillometric devices are widely used in paediatrics mainly due to ease of use and minimization of observer bias [75,76].

In addition, it has been shown that automated devices are equally good or even better than auscultatory devices in predicting long-term BP tracking [77]. However, physicians should be aware that the automated devices slightly overestimate the BP and that the commonly used normative BP values may not apply.

In addition, the normative values published by the Pediatric Task Force [8] were obtained from both normal weight and overweight healthy children, which may potentially overestimate the BP in normal weight children. Indeed, the recalculated normative values from normal weight children are slightly lower than previously developed norms based on Pediatric Task Force data including both normal weight and overweight children [78].

**Number of BP measurements and home/office differences**

Regardless of the type of BP device used for measurement, it is necessary to confirm an elevated BP on repeated visits and/or outside the office before characterizing a child as having HTN. However, the exact number of BP measurements needed for the diagnosis of HTN is not clearly defined. Multiple BP measurements taken over weeks or months are recommended in the guidelines on paediatric HTN [8,9]. In contrast, the classification in adults is based on the average of two or more properly measured, seated BP readings on each of two or more office visits [10].

It is also recommended that multiple BP measurements be obtained in various settings, ideally outside of the medical setting. This includes home BP measurements as well as ABPM (ambulatory blood pressure monitoring). The home BP was superior to the clinic BP in predicting ABPM results, but neither detected HTN with enough sensitivity or specificity to replace ABPM [79]. In a recent report, Stergiou et al. [80] concluded that in children and adolescents, there is reasonable agreement between home and ambulatory BP measurements as diagnostic methods in HTN. However, the positive and negative predictive values were only 74% and 82% respectively. Therefore HTN should be ideally confirmed by ABPM.

**ABPM**

An accurate assessment and management of BP is essential for the prevention of target organ damage [81]. ABPM can more precisely characterize changes in BP throughout daily activities [82] and therefore has been found to be superior to clinic BP monitoring in predicting cardiovascular morbidity and mortality in adults [83]. In children and adolescents, the prevalence of use of ABPM has not been reported; however, ABPM is gaining acceptance as a useful modality for the evaluation of BP levels in both HTN research and in the clinic setting [84,85]. The use of ABPM in children is usually limited to children older than 5 years of age, and a multitude of data obtained from ABPM requires expertise in reading and interpretation of the results. The current research and clinical applications of ABPM in children and adolescents were recently summarized by Urbina et al. [86].

As no outcome studies are yet available relating ABPM levels in children to hard outcomes such as myocardial infarction or stroke, these guidelines are expert opinion-driven and not evidence based [86]. The use of ABPM is recommended in situations listed in Table 2.

Owing to the multitude of results obtained from the ABPM, potential technical difficulties and complexity of paediatric reference values for ABPM ([87], but see [87a]), the interpretation of ABPM in children is not straightforward. Therefore the ABPM should be performed by personnel with specific training in the application of the device and interpretation of ABPM data in paediatric patients.

ABPM is particularly useful in children with white-coat and masked HTN as these conditions are not uncommon [88,89] and would not be diagnosed by the office BP. Masked HTN in children and adolescents is associated with a similar risk of end-organ damage as in established HTN [90,91]. Even white-coat HTN may lead to significant end-organ damage [92,93] and therefore represent a prehypertensive state [92]. The prevalence of both masked and white-coat HTN is not

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Adapted and modified from [86].

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negligible, especially in children with chronic conditions, such as chronic kidney disease [94] and diabetes mellitus type 1 [89]. This further underlines the importance of ABPM in children.

**IMPACT OF HTN**

**BP measured in childhood predicts adult BP (BP tracking)**

BP tracking can be described as “a phenomenon when a BP level early in life predicts a BP level later on” [95]. In a recent systematic review and metaregression analysis, the evidence was found to be strong for BP tracking from childhood into adulthood [96]. In addition, an elevated BMI in hypertensive children increases the risk of persistent HTN in adulthood [97]. Thus obesity, in combination with HTN, seems to confer increased cardiovascular risk throughout the life of an individual. Furthermore, other factors such as male gender and family history of essential HTN contribute to a more stable BP tracking [98]. This means that hypertensive boys with family history of HTN are more likely to develop HTN in adulthood. Serum uric acid was also shown to be an independent predictor of longitudinal BP progression [99].

**Impact of the level of BP control**

The BP tracking phenomenon implies that a lower BP level in childhood represents a lower risk of HTN in adult life. In adults, every mmHg increase in BP increases the risk of stroke and cardiovascular mortality [100]. Bo et al. [101] demonstrated that even individuals with high-normal BP had a higher prevalence of cardiovascular and metabolic risk factors than those with optimal BP. In addition, these patients had early signs of endothelial dysfunction and oxidative stress, suggesting that even mild HTN is not benign, especially if it originates early in life. In fact, a significant proportion of children with newly diagnosed HTN already have evidence of end-organ damage [4,102]. Therefore it seems logical that the target BP in children should be lower than the 50th percentile rather than just below the 95th percentile. This is now supported by a very recent paediatric multicentre study showing that aggressive BP control (below the 50th percentile) reduced progression of chronic kidney disease in children [103]. The clinical advantage of an ‘aggressive BP control’ needs to be confirmed in larger longitudinal studies; if confirmed, it would significantly change the approach to the practical management of HTN, at least in children with chronic kidney disease.

**End-organ damage (structural changes)**

LVH has been used as a surrogate marker for HTN-associated cardiovascular damage [104–106]. LVH has been usually assessed by the LVMi (left ventricular mass index); values above 38.6 g/m²⁷ (95th percentile) define LVH, values above 51 g/m²⁷ (adult cut-off) define severe LVH. However, the use of LVMi only may not identify some patients with concentric LVH [107]. In addition, the LVMi increases with decreasing height [108]. The cut-off defining LVH would, therefore, strongly depend on height distribution of the reference group. Foster et al. recently proposed a novel method of expressing left ventricular mass relative to body size and defining LVH by use of centile curves [109]. This approach provides a numeric value on a continuous scale (z score or percentile) and a stable estimate of the proportion of at-risk children with LVH regardless of reference group [109].

Recently, CIMT (carotid intima media thickness), measured by non-invasive vascular ultrasound, has emerged as a potential marker of cardiovascular structural damage. Normative values are available for patients aged 10 to 20 years of age [110] limiting the use of this technique to children above 10 years. Further studies and determination of normative values of CIMT in younger children (less than 10 years of age) are needed. Nevertheless, CIMT measurement appears as a good method for the assessment of early arterial wall changes, which may predict other cardiovascular sequelae in children with HTN and/or obesity. Indeed, a significant number of children with essential HTN have an increased CIMT suggesting early vascular structure damage [4,111,112]. Although obesity may play a significant role in vascular changes, a recent study showed that hypertensive subjects had increased CIMT compared with BMI-matched controls, providing strong evidence that CIMT is increased in childhood primary HTN, independent of the effects of obesity [113].

**Arterial wall dysfunction (functional changes)**

Structural changes (LVH, increased CIMT) may be preceded by functional changes in the arterial function and elasticity. Aggoun et al. [114] recently demonstrated that impaired brachial endothelial and smooth muscle functions, as well as alteration of wall material of the carotid artery, appear before puberty in obese children without concomitant increase of the CIMT. These functional changes result in decreased arterial cross-sectional compliance (distensibility) or increased arterial stiffness. Arterial dysfunction involving both endothelium and smooth muscles may be considered as the first markers of atherosclerosis [114].

Arterial rigidity or stiffness can be assessed by measurement of the PWV (pulse wave velocity), which proved to be a good marker of cardiovascular risk in adults [115]. An increased PWV has been recently reported in children after renal and cardiac transplantation [116,117] and in children with Type 1
diabetes mellitus [118]. Children with essential and secondary HTN were also studied using another index of arterial stiffness calculated from the ABPM – AASI (ambulatory arterial stiffness index). The AASI is defined as one minus the regression slope of DPB over SBP measurements obtained during a 24-h period and correlates with PWV [119]. As expected, the AASI was found to be elevated in hypertensive children and correlated with the duration and the origin of HTN in childhood [120]. Therefore AASI can potentially be used in clinical practice to assess the severity and duration of HTN in children. However, further longitudinal studies of AASI during treatment of childhood HTN are indicated before it can be recommended for routine use.

This data suggests that children with HTN and other conditions associated with HTN have an increased risk of cardiovascular complications.

**Kidney damage**

There are limited data on kidney damage/renal dysfunction in children with HTN. Lubrano et al. [121] recently showed that children with even mildly elevated BP and higher BP load (>40%) had lower glomerular filtration rate and higher proteinuria, suggesting early renal damage. Moreover, microalbuminuria lowering halts the progression of LVH or induces its regression in children and adolescents with essential HTN [122]. Further studies are needed to explore the impact of HTN on kidney function and structure in children and formulate recommendations for routine follow-up of the renal changes in hypertensive children.

**Cognitive changes**

Long-standing HTN may lead to cognitive impairment and dementia in the elderly [123,124]. Children with an elevated BP >90th percentile had poorer performance on selected tests of cognition compared with normotensive children [125]. In addition, children with both HTN and obesity demonstrate higher rates of clinically significant internalizing problems, and hypertensive children (irrespective of obesity) demonstrate lower parental ratings of executive function compared with normotensive control subjects [126].

This data suggests that hypertensive children are also at risk for central nervous system end-organ damage, which further underlies the importance of early and efficacious treatment of HTN in children.

**MANAGEMENT OF HTN**

The evaluation of children with suspected primary or secondary HTN has been described in detail in recent guidelines [8,9]. Recent reports emphasizing the need for evaluation of secondary causes of HTN even in children with suspected primary HTN [46] have already been discussed.

Similarly to adults, the management of HTN in children has benefited from the availability of new and safer drugs, although the choice of medication approved for use in children remains limited. The advances in the management of paediatric HTN have been summarized in several recent reports [7,9].

**SUMMARY**

HTN in children has been gaining significant attention in the last decade, mainly due to the increased prevalence of primary HTN, most likely linked to the epidemic of childhood obesity. The estimated prevalence of paediatric HTN varies from 1% to 10%, but more importantly, it continues to increase over time. This fact, along with BP tracking, will probably lead to a significant increase in adult HTN within the next decade or so, which may further worsen the cardiovascular morbidity and mortality. This could be theoretically prevented by early diagnosis and management of HTN in children before they even develop end-organ damage. In addition to increased alertness to paediatric HTN in clinical practice, along with careful and perhaps more aggressive monitoring and management of HTN in children, research efforts should continue with the goal to clarify the aetiology, complexity and inheritable factors of childhood HTN. Research efforts should also focus on optimal treatment of these children and on effective preventive measures starting from a young age.

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