SPECIAL ISSUE ARTICLE

Hypertension in Children and Adolescents: A Review of Recent Guidelines

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ABSTRACT

Pediatric hypertension (HTN) is a growing problem worldwide that can be attributed to various risk factors, including the upward trend in obesity and poor lifestyle choices. Pediatric HTN will eventually lead to adult HTN and cardiovascular disease. There is concern that HTN in children and adolescents is often underdiagnosed. This article highlights important risk factors and chronic conditions associated with HTN along with complications such as end organ damage and cardiovascular disease. This article also outlines cost-effective diagnostic evaluations and step-wise treatment options, including nonpharmacological interventions such as lifestyle modifications as well as medical management based on the most recent American Academy of Pediatrics Clinical Practice Guidelines. **[*Pediatr Ann*. 2020;49(6):e250-e257.]**

The prevalence of hypertension (HTN) in children worldwide is increasing, currently affecting about 5% of children in the general population. It is clear that HTN in adults has its origins in childhood and is a strong contributor to the high prevalence of cardiovascular disease.[1] The most recent American Academy of Pediatrics Clinical Practice Guidelines (AAP CPG) on HTN were released in 2017 to improve screening and management.[2] They urge the need for early screening of the general pediatric population and close monitoring of the patients at risk for and with HTN. They place emphasis on the importance of lifestyle changes in all patients to treat and prevent HTN as well as on pharmacological treatments for children who have persistent or severe HTN.

The definition of HTN according to the AAP CPG[2] is as follows:

- Normal blood pressure (BP) is defined as systolic and diastolic BP ≤90th percentile for age, height, and gender for preadolescents; and ≤120/80 mm Hg for children age 13 years and older.
- Elevated BP has replaced the term prehypertension. It is defined as BP between the 90th and 95th percentiles. For adolescents age 13 years and older, elevated BP is defined as >120/80 mm Hg to 129/80 mm Hg.
- Stage 1 HTN is defined as BP ≥95th percentile but <95th percentile + 12 mm Hg or 130/80 to 139/89 mm Hg (whichever is lower). For children older than age 13 years, Stage 1 HTN is between 130/80 and 139/89 mm Hg. BP needs to be elevated on at least three different occasions.
- Stage 2 HTN is BP ≥95th percentile + 12 mm Hg or ≥140/90 mm Hg (whichever is lower). For children age 13 years and older, stage 2 HTN is ≥140/90 mm Hg.

The European guidelines[3] and the AAP CPG[2] were released in 2016 and 2017, respectively. Some of the differences between them include the following:

- The American guidelines modified the database to only include children of normal weight, whereas the European guidelines used the original database.[4] Including children with excess weight is a bias to underdiagnose HTN.
- The diagnosis of HTN is based on age, height, and gender until age 16 years for the European guidelines but only until age 13 years for the American guidelines. After these respective ages, the adult guidelines apply (≥140/90 mm Hg for the European guidelines and ≥130/80 mm Hg for the American guidelines). There has been

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controversy on both limits because a hypertensive child can become classified as normotensive after turning age 13 years in America or age 16 years in Europe.

• The definitions of Stage 1 and Stage 2 HTN are different.

• The AAP CPG guidelines recommend echocardiogram at the time when pharmacological treatment is considered, whereas the European guidelines recommend echocardiogram early on in the evaluation to consider the need for medications.

PROPER MEASUREMENT OF BLOOD PRESSURE

Children should be comfortably seated in a quiet room for 3 to 5 minutes before BP is taken. Their back should be supported, and their feet should be uncrossed and fully touching the floor. BP should be measured in the right arm as BP can be lower in the left arm in patients with coarctation of aorta. The arm should be at heart level supported in armchair or leg. The screening should ideally happen on a well-child visit to decrease the chances of pain, fever, or other conditions falsely increasing the BP. To choose the correct arm cuff, the bladder length should be 80% to 100% of the circumference of the arm and the width should be at greater than 40%. For auscultatory BP, the stethoscope should be placed over the brachial artery; the cuff should be inflated 20 to 30 mm Hg above the radial pulse disappearance. Inflation above this has no benefit in BP measurement and can cause pain that could elevate the BP. When leg BP is needed, the patient should be in a prone position. The cuff should be placed in mid-thigh and the stethoscope placed on the popliteal artery. Lower extremity BP is 10% to 20% higher than upper extremities.[2]

When to Screen for HTN?

BP monitoring should start at age 3 years in otherwise healthy children and annually thereafter.

BP should be monitored on every clinic visit for children who are overweight or obese or have any other risk factor for HTN listed in **Table 1**. BP reading should start at the time of diagnosis of that comorbidity.

HYPERTENSION IN CHRONIC CONDITIONS

Obesity

The prevalence of obesity has increased in developed and developing countries in people of all ages. According to the World Health Organization,[5] obesity has nearly tripled since 1975 and by 2016 there were 41 million children age 5 years and younger and 340 million children age 6 to 18 years who were overweight or obese worldwide. The number of adults with HTN increased from 594 million in 1975 to 1.13 billion in 2015.[5] Obesity in children is associated with HTN, dyslipidemia, insulin resistance, and diabetes among other chronic conditions. Abdominal adiposity is associated with HTN, particularly in boys. The severity of HTN also increased as adiposity and waist circumference increased.[6]

Sleep-Disordered Breathing

Sleep-disordered breathing is a well-known risk factor for HTN; however, lack of sleep can also cause HTN. Even without any sleep pathology, short sleep duration has been linked with HTN.[7] The proposed mechanism of sleep-related HTN is overactivation of the sympathetic system, increased cortisol, endothelial dysfunction, and insulin resistance, as well as an increase in inflammatory markers.[8,9] Xu et al.[10] showed that nocturnal BP was elevated in patients with sleep apnea and there was a linear relationship between nocturnal BP and obstructive apnea events. This is explained by the stimulation of the autonomic nervous system, the severity and frequency of oxygen desaturations, increase in respiratory effort, and duration of abnormal breathing that in turn can affect BP.[11]

Acute and Chronic Kidney Disease

Acute kidney injury (AKI) is a transient condition; however, there are strong data suggesting that AKI is associated with long-term HTN, cardiovascular disease, and chronic kidney disease (CKD). AKI was independently associated with a 22% increase in risk to

TABLE 1.

Conditions Associated with Hypertension

- Prematurity ≤32 weeks and low birth weight
- Congenital heart disease (eg, coarctation of the aorta)
- Renal disease including CKD, recurrent UTIs, urological malformation, hematuria, proteinuria
- Family history of congenital renal disease
- Solid organ transplant
- Malignancy and bone marrow transplant
- Increased intracranial pressure
- Abdominal aortic obstruction (neurofibromatosis, Williams syndrome, Alagille syndrome, or Takayasu arteritis)
- Endocrine disorders (catecholamine excess, mineralocorticoid excess, congenital adrenal hyperplasia, familiar hyperaldosteronism, hyperthyroidism)
- Environmental exposure (lead, cadmium, mercury, phalates)
- Systemic illnesses that cause HTN (sickle cell disease, tuberous sclerosis)
- Medications (NSAIDS, corticosteroids, ADHD medications)

ADHD, attention-deficit/hyperactive disorder; CKD, chronic kidney disease; HTN, hypertension; NSAIDS, nonsteroidal anti-inflammatory drugs; UTI, urinary tract infection.
Adapted from Flynn et al.[2]

TABLE 2.

Etiology of Hypertension by Age

Age group	Etiology
Neonates	Renal artery thrombosis Renal artery stenosis Congenital renal abnormality Bronchopulmonary dysplasia Prematurity UAC/UVC lines Low birth weight Congenital heart disease
30 days to 1 year	Renal artery stenosis Renal disease Coarctation of the aorta
1 to 6 years	Renal disease Renal artery stenosis Coarctation of the aorta
6 to 12 years	Renal parenchymal disease Renal artery stenosis Primary hypertension Coarctation of the aorta Endocrinopathies
12 to 18 years	Primary hypertension Renal disease Endocrinopathies Renal artery stenosis

Abbreviations: UAC, umbilical arterial catheter; UVC, umbilical vein catheters.

develop elevated BP, and this risk was higher with more severe renal injury.[12] CKD and HTN are closely associated, as HTN can cause CKD[13] and vice versa. HTN can also promote the deterioration of renal function in CKD patients.

Prematurity and Low Birth Weight

History of prematurity and low birth weight are independent risk factors for the development of HTN in children and adults. The theory behind this is the lower number of nephrons in these patients, but there are also reports of hyperfiltration and hyperactivity of the renin-angiotensin-aldosterone system in adults who have a history of prematurity.[14]

Other conditions associated with HTN are listed in **Table 1** and **Table 2**.

CONSEQUENCES OF HYPERTENSION: END ORGAN DAMAGE

Hypertension in children causes damage to the heart, kidneys, eyes, and brain. HTN causes left ventricular hypertrophy, which is strongly related to cardiovascular disease in adulthood.[15] In addition, HTN is associated with increased carotid intimal medial thickness (cIMT) and pulse wave velocity, which are measures of arterial stiffness. There is a progressive increase in cIMT, arterial stiffness, and left ventricular mass index and a decrease in diastolic function in patients who progress from normal BP to pre-HTN and then HTN.[16] Leiba et al.[13] found that HTN doubles the risk of end stage renal disease in his cohort of 2 million teenagers.

ETIOLOGY AND DIFFERENTIAL DIAGNOSIS

Essential hypertension is defined as HTN with no underlying secondary cause. Primary HTN is associated with a sedentary lifestyle, a diet high in fat and sodium, obesity, and abnormal sleep patterns. Secondary HTN is when the elevated BP is caused by an underlying medical condition. Causes of secondary HTN are listed in **Table 1** and **Table 2**.

EVALUATION

Normal BP

If BP is normal (BP <90th percentile) after repeat readings, no additional evaluation is needed. Continue to measure BP in the well-child visits.

Elevated BP

If BP is elevated at initial measurement, recommend lifestyle changes in nutrition and exercise. Repeat BP measurements, including BP measurements of the four extremities and lifestyle counseling in 6 months. If BP is persistently elevated at 6 months, consider ambulatory BP monitoring (ABPM), diagnostic evaluation, and subspecialty referral.

Stage 1 HTN

If BP is at stage 1 HTN at initial presentation, recommend lifestyle modifications with BP monitoring measurements of the four extremities and repeat evaluation in 1 to 2 weeks. Re-evaluate BP again in 3 months. If persistently in stage 1 HTN, proceed with ABPM, diagnostic evaluation, pharmacological treatment, and subspecialty referral.

Stage 2 HTN

If BP is at stage 2 HTN at initial presentation, check BP of the four extremities, recommend lifestyle changes, and re-evaluate or refer to subspecialty within a week. If patient is symptomatic, they need emergency department referral for evaluation and management. If BP is still at stage 2 HTN level when measured 1 week later, recommend diagnostic evaluation and ABPM, and initiate pharmacological treatment.

Diagnostic Evaluation

Evaluation should start with a thorough perinatal, birth, and past medi-

cal history. Family history of HTN and related conditions such as early cardiovascular disease is also important. History of diet and physical activity should be obtained. Important risk factors to consider include low birth weight or prematurity, and history of recurrent urinary tract infections leading to renal scarring. Any recent infection such as streptococcal pharyngitis, rash concerning for Henoch-Schönlein purpura, exposure to nephrotoxic medications, or pain may all contribute to HTN. Physical examination should be detailed to include signs of secondary causes of HTN or end organ damage, body metrics, and BP measurement of all four limbs (**Table 3**).

TABLE 3.

Clinical Features in Secondary Hypertension

Body system	Clinical features	Etiology
Vital signs	Tachycardia	Hyperthyroidism, phenochromocytoma, neuroblastoma
	Decreased lower extremity pulses and blood pressures	Coarctation of aorta
Height and weight	Growth stunting	Chronic kidney disease
	Obesity	Cushing syndrome, insulin resistance syndrome
Eyes	Proptosis	Hyperthyroidism
	Papilledema	Increased intracranial pressure
	Retinal hemorrhages, arteriovenous nicking	Hypertensive emergency, hypertensive retinopathy
Ears, nose, and throat	Adenotonsillar hypertrophy	Sleep disordered breathing
Head, neck	Elfin facies	Williams syndrome
	Moon facies	Cushing syndrome
	Thyromegaly, goiter	Hyperthyroidism
	Webbed neck	Turner syndrome
Skin	Pallor, flushing, diaphoresis	Penochromocytoma
	Cafè-au-lait spots, axillary freckling	Neurofibromatosis
	Ash leaf patches, angiofibromas, adenoma sebaceum	Tuberous sclerosis
	Palpable purpura	Henoch-Schönlein purpura, vasculitis
Chest, cardiac	Chest pain/palpitations	Heart disease
	Widely spaced nipples	Turner syndrome
	Heart murmur	Coarctation of aorta
	Apical heave	Left ventricular hypertrophy
Abdomen	Abdominal mass	Wilms tumor, neuroblastoma
	Flank/epigastric bruit	Renal artery stenosis
	Palpable kidneys	Polycystic kidneys, multicystic dysplastic kidneys
Genitourinary	Ambiguous genitalia	Congenital adrenal hyperplasia
Extremities	Joint swelling	Systemic lupus erythematosus
Neurologic, metabolic	Headache, altered mental status	Hypertensive encephalopathy
	Muscle weakness	Monogenic hypertension

Adapted from the Lurbe and Ferrer:[3]

Laboratory Analysis and Imaging

Laboratory analysis should include a urinalysis, electrolytes, blood urea nitrogen and creatinine, and lipid profile in all patients. Renal ultrasound should be done in children younger than age 6 years or those with abnormal urinalysis or renal function. If patient is obese (body mass index >95% percentile), hemoglobin A1C, liver enzymes, and fasting lipid panel should be obtained. Optional additional tests include thyroid-stimulating hormone, drug screen, or a sleep study (**Table 4**). Further imaging should be used to investigate for renal vascular HTN in patients who have Stage 2 HTN, presence of renal bruits, hypokalemia on laboratory analysis, or notable size discrepancy of kidney size and parenchyma on standard ultrasound imaging. Doppler renal ultrasonography can be considered as an initial screening tool for evaluation of renal artery stenosis, but computed tomographic angiography and magnetic resonance angiography have better sensitivity and specificity. Renal arteriography is the gold standard for vascular evaluation.

Assessment of End Organ Damage

Left ventricular hypertrophy is strongly associated with HTN and adverse cardiovascular disease in adults.[17] Echocardiogram is the test to diagnose end organ damage on the left ventricle according to the AAP CPG.[2] As per the 2017 AAP CPG, echocardiogram should

TABLE 4.

Optional Tests for Hypertension

Optional tests	Reason for evaluation
Thyroid studies	Hyperthyroidism
Urine drug screen	Illicit drug use
Sleep study	Obstructive sleep apnea
Plasma metanephrines	Pheochromocytoma
Plasma and urine steroid levels	Steroid-mediated hypertension

Adapted from the National High Blood Pressure Education Program Working Group.[4]

be done at the time of initiation of pharmacological treatment. The definition for left ventricular hypertrophy (LVH) is left ventricular (LV) mass >51 g/m^2 (both boys and girls) when age 8 years and older or LV mass >115 g/body surface area (BSA) for boys and >95 g/BSA for girls.[7] Echocardiography should be repeated every 6 to 12 months to evaluate the changes in LV mass. Electrocardiography is not recommended in patients with hypertension to evaluate for LVH.[18]

MANAGEMENT

The treatment goal for HTN with nonpharmacological and pharmacological therapy is reduction of systolic and diastolic BP to <90% percentile in children younger than age 13 years and <130/80 mm Hg in adolescents age 13 years and older.

Nonpharmacological

According to the new guidelines, lifestyle modifications alone may have a significant impact on the reduction of BP and should be the initial recommendation in the management of HTN.[19] Lifestyle modifications should include incorporating the Dietary Approaches to Stop Hypertension diet with reduced sodium, sugar, fat, and processed foods. In addition, increasing physical activity and improvement in sleep is also beneficial.[20] A team-based approach involving a dietician and education of parents and patient has shown best results.

Physical activity is an important aspect in the management of hypertension. A recent review demonstrated that 30 to 60 minutes of aerobic exercise in children and adolescents will lead to improvement in BP.[21] Static exercises such as weightlifting should be approached cautiously in children with Stage 2 hypertension because weightlifting can increase BP.

Improving the quantity and quality of sleep also decreases the risk of HTN. Although the duration of sleep and the association with HTN is not clearly defined, the American Academy of Sleep Medicine[22] recommends 12 to 13 hours of sleep for children age 1 to 2 years, 11 to 12 hours for children age 3 to 5 years, 9 to 11 hours children age 6 to 12 years, and 8 to 10 hours for adolescents age 13 to 18 years.

Pharmacological Treatment

Antihypertensive medications are recommended when children remain hypertensive despite lifestyle modifications, have evidence of end organ damage, or have Stage 2 HTN. It is also recommended to prescribe antihypertensive medications for children with CKD and diabetes mellitus. Medications should be initiated at the lower end of the dosing spectrum, and combination medications should be avoided for initial therapy in children. Lifestyle modifications should always go along with the medication regimen.

Antihypertensive doses should be titrated every 2 to 4 weeks with home BP measurements until BP control is achieved. During this time, it is recommended that they follow up with their providers every 4 to 6 weeks while titration to the medication dose is being made. Once BP is controlled, follow-up appointments can be every 3 or 4 months.

Choice of Antihypertensive Agent

The new guidelines recommend initial treatment with angiotensin-converting enzyme inhibitors (ACEI), angiotensin II receptor blockers (ARB), long-acting calcium channel blockers, or a thiazide diuretic. See **Table 5** for more information about schedules and doses.

ACEIs improve proteinuria and HTN, and can slow the progression of CKD. It is the antihypertensive preferred for people with diabetes, people with proteinuria, and people with left ventricular dysfunction. ARBs have a similar profile but they carry a lower risk of the chronic dry cough that can be an uncomfortable side effect of ACEIs. Calcium channel blockers are frequently used due to their safety and lack of side effects in the pediatric population. Beta-blockers are not recommended for initial antihypertensive regimen in children, and because they can cause a decrease of cardiac ability to modulate heart rate they should be avoided in athletes. Beta-blockers should also be avoided in children with asthma and diabetes. Diuretics are the first line of treatment for adults. Thiazides are most frequently used as antihypertensive agents; however, patients need to be monitored for electrolyte abnormalities. Clonidine is a central alpha-agonist that is efficacious and has the advantage of transdermal weekly application; however, it may have a sedative effect in children.

TABLE 5.

Pharmacological Treatment of Hypertension

Class	Mechanism of action	Agent	Dose	Side effects
Angiotensin-converting enzyme inhibitor[a]	Degradation of bradykinin through the blockade of kinin-kallikrein system Efferent artery dilation	Captopril Enalapril Lisinopril (in patients >6 years)	Initial: 0.3 to 0.5 mg/kg/dose (tid-qid) Maximum: 6 mg/kg/day Initial: 0.08 mg/kg/day (qd-bid) Maximum: 0.6 mg/kg/day to 40 mg/day Initial: 0.07 mg/kg/day (qd) Maximum: 0.6 mg/kg/day to 40 mg/day	Cough Hyperkalemia Acute renal failure Angioedema Neutropenia Thrombocytopenia
Angiotensin receptor blockers[a]	Block the binding of angiotensin II to type I angiotensin II receptors	Losartan	Initial: 0.7 mg/kg/day Maximum: 1.4 mg/kg/day to 100 mg/day	Hyperkalemia Acute renal failure
Calcium channel blockers	Block the influx of calcium into smooth muscle, resulting in arteriole dilatation	Amlodipine Isradipine Nifedipine XL Nifedipine (short-acting)	Initial: 0.1 mg/kg/day Maximum: 0.6 mg/kg/day to 10 mg/day Initial: 0.05 mg/kg to 0.1 mg/kg/day Maximum: 0.6 mg/kg/day to 10 mg/day Initial: 0.2 mg/kg/day to 0.5 mg/kg/day Maximum: 3 mg/kg/day (up to 120 mg/day) Initial: 0.04 mg/kg/day to 0.25 mg/kg/dose Maximum: 1 mg/kg/day to 2 mg/kg/day	Tachycardia Peripheral Edema Headaches Flushing Gingival hyperplasia
Beta-blockers	Competitively inhibiting catecholamines from binding to B1, B2, and B3 receptors	Atenolol Metoprolol Propranolol Labetalol	Initial: 0.5 to 1 mg/kg/dose (qd- bid) Maximum: 2 mg/kg/day to 100 mg/day Initial: 1 mg/kg/day to 2 mg/kg/day (bid) Maximum: 6 mg/kg/day to 200 mg/day Initial: 1 mg/kg/day to 2 mg/kg/day (bid-tid) Maximum: 4 mg/kg/day to 640 mg/day Initial: 1 mg/kg/day to 3 mg/kg/day (bid) Maximum: 10-12 mg/kg/day to 1200 mg/day	Decrease cardiac contractility Bronchospasm Fatigue Insomnia
Diuretic	Thiazides inhibit the Na^+ Cl-transporter in the early distal convoluted tubule Loop diuretics: inhibit Na-K-2Cl carrier in the thick ascending limb of the loop of Henle	Hydrochlorothiazide Furosemide Amiloride Spironolactone	Initial: 1 mg/kg/day (qd) Maximum: 3 mg/kg/day up to 50 mg Initial: 0.5 mg/kg/day to 2.0 mg/kg/day (qd-bid) Maximum: 6 mg/kg/day Initial: 0.4 mg/kg/day to 0.6 mg/kg/day (qd) Maximum: 20 mg/day Initial: 1 mg/kg/day (qd-bid) Maximum 3.3 mg/kg/day to 100 mg/day	Electrolyte abnormalities Hyponatremia Hypokalemia Hypochloremia Hypercalciuria/stones[b] Hypocalciuria[c] Ototoxicity Dehydration Renal failure

Treatment in Special Conditions

Chronic kidney disease. HTN is frequent in children with CKD. Studies have shown that aggressive treatment of HTN and proteinuria slows the progression of CKD. ACEIs and ARBs are frequently used except when patients have hyperkalemia or advanced renal disease because these medications can further deteriorate renal function. ABPM should be used in patients with CKD to rule out masked HTN that can lead to LVH.[23,24] Accord-

TABLE 5. (*continued*)

Pharmacological Treatment of Hypertension

Class	Mechanism of action	Agent	Dose	Side effects
Central alpha-blockers	Central acting alpha-2 agonist that stimulates these receptors to decrease peripheral vascular resistance and heart rate	Clonidine	Initial: 5-10 mcg/kg/day in divided doses every 8-12 hours Maximum: 0.9 mg/day	Sleepiness Drowsiness Dry mouth Rebound HTN when abruptly discontinued
Vasodilator	Vasodilators directly act on vascular smooth muscle to reduce peripheral vascular resistance	Hydralazine	Initial: IV/IM 0.1 -0.2 mg/kg/dose Maximum IV dose is 20 mg/dose Oral: 0.75 mg/kg/day q4-6 hours	Flushing Headaches Fluid retention Lupus-like syndrome Hypertrichosis Tachycardia

Abbreviations: bid, two times daily; HTN, hypertension; IM, intramuscular; IV, intravenous; qd, once daily; qid, four times daily; tid, three times daily.
[a]Teratogenic.
[b]Loop diuretic.
[c]Thiazide.
Adapted from Flynn et al.[2] and the National High Blood Pressure Education Program Working Group.[4]

ing to the new AAP guidelines[2] (1) BP should be assessed at every medical encounter in children with CKD; (2) BP goal is <50th percentile by ABPM; and (3) ABPM should be done annually to screen for masked hypertension.

Hypertension in diabetes. HTN can be present in both type 1 (T1DM) and type 2 diabetes (T2DM); however, prevalence is higher in children with T2DM (from 12% to 31%).[25] BP higher than 130/90 mm Hg has a strong association with cardiovascular disease.[26] Despite the complications and recommendations by the American Diabetes Association,[27] studies have demonstrated poor awareness and overall lower use of antihypertensive medications in this population. With that said, the recommendations from the recent AAP guidelines recommend that children with T1DM or T2DM should be evaluated for HTN with each medical encounter and treated for HTN if BP ≥95% percentile or >130/80 mm Hg in adolescents.[2]

Hypertension urgency and emergency. Children and adolescents who present with acute severe HTN and signs of end organ damage such as congestive heart failure need to be treated with fast-acting intravenous medications with the goal of reducing BP by 25% in the first 8 hours. The remainder of BP reduction may be achieved in the next 24 hours to achieve the goal of approximately the 95% percentile. Oral agents can be tried if the patient is able to tolerate them and does not have acute symptoms.

Hypertension in the athlete. As mentioned previously, physical activity should be encouraged in children with HTN as it is associated with lower mortality, improvement in BP, and cardiac health. However, children with Stage 2 HTN, even without evidence of end organ damage like LVH, should not be permitted to participate in high-static sports including weightlifting, boxing, or wrestling until their blood pressure is controlled after lifestyle modifications and/or medications. In addition, athletes with LVH due to HTN should be restricted from competitive sports until BP is reduced to normal range.

Hypertension in solid organ transplants. There is a high prevalence of HTN seen after solid organ transplant due to various reasons, most frequently secondary to steroids, nephrotoxic medications, and AKI. ABPM is considered the most effective tool in diagnosing, managing, and treating HTN in solid organ transplants such as kidney and heart transplants.[28] Studies demonstrate that good BP control is essential for the well-being and stability of the graft.[29] There may be evidence that ACEI or ARB may be beneficial in achieving BP control and long-term graft survival.[30]

CONCLUSION

The incidence of HTN in children is increasing, and it is clear that childhood HTN is the start of adult HTN and cardiovascular disease. Most causes are due to or exacerbated by poor lifestyle choices that will fur-

ther persist in these patients to adulthood. It is of paramount importance that pediatricians educate patients and families on the importance of a healthy lifestyle, including healthy nutrition with abundant fruits and vegetables, daily exercise, and sleep. If needed, antihypertensive medications should be used to lower BP and prevent end organ damage.

REFERENCES

1. Chen X, Wang Y. Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation*. 2008;117(25):3171-3180. https://doi.org/10.1161/CIRCULATIONAHA.107.730366 PMID:18559702
2. Flynn JT, Kaelber DC, Baker-Smith CM, et al.; Subcommittee on Screening and Management of High Blood Pressure in Children. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. *Pediatrics*. 2017;140(3):e20171904. https://doi.org/10.1542/peds.2017-1904 PMID:28827377
3. Lurbe I, Ferrer E. European Society of Hypertension Guidelines for the management of high blood pressure in children and adolescents. *An Pediatr* (Barc). 2016;85(4):167-169. https://doi.org/10.1016/j.anpedi.2016.08.001 PMID:27692099
4. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The Fourth report on the diagnosis, evaluation and treatment of high blood pressure in children and adolescents. *Pediatrics*. 2004;114(2 suppl 4th Report):555-576. PMID:15286277
5. World Health Organization. Obesity and overweight. https://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight. Accessed May 13, 2020.
6. Skinner AC, Perrin EM, Moss LA, Skelton JA. Cardiometabolic risks and severity of obesity in children and young adults. *N Engl J Med*. 2015;373(14):1307-1317. https://doi.org/10.1056/NEJMoa1502821 PMID:26422721
7. Gupta N, Maranda L, Gupta R. Differences in self-reported weekend catch up sleep between children and adolescents with and without primary hypertension. *Clin Hypertens*. 2018;24(1):7. https://doi.org/10.1186/s40885-018-0092-6 PMID:29636986
8. Gangwisch JE. A review of evidence for the link between sleep duration and hypertension. *Am J Hypertens*. 2014;27(10):1235-1242. https://doi.org/10.1093/ajh/hpu071 PMID:24778107
9. Mullington JM, Haack M, Toth M, Serrador JM, Meier-Ewert HK. Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Prog Cardiovasc Dis*. 2009;51(4):294-302. https://doi.org/10.1016/j.pcad.2008.10.003 PMID:19110131
10. Xu Z, Li B, Shen K. Ambulatory blood pressure monitoring in Chinese children with obstructive sleep apnea/hypopnea syndrome. *Pediatr Pulmonol*. 2013;48(3):274-279. https://doi.org/10.1002/ppul.22595 PMID:22615200
11. Guilleminault C, Khramsov A, Stoohs RA, et al. Abnormal blood pressure in prepubertal children with sleep-disordered breathing. *Pediatr Res*. 2004;55(1):76-84. https://doi.org/10.1203/01.PDR.0000099791.39621.62 PMID:14605262
12. Hsu CY, Hsu RK, Yang J, et al. Elevated BP after AKI. *J Am Soc Nephrol*. 2016;27(3):914-923. https://doi.org/10.1681/ASN.2014111114 PMID:26134154
13. Leiba A, Fishman B, Twig G, et al. Association of adolescent hypertension with future end-stage renal disease. *JAMA Intern Med*. 2019;179(4):517-523. https://doi.org/10.1001/jamainternmed.2018.7632 PMID:30801616
14. Vasylyeva TL, Chennasamudram SP, Okogbo ME. Can we predict hypertension among preterm children? *Clin Pediatr* (Phila). 2011;50(10):936-942. https://doi.org/10.1177/0009922811409918 PMID:21646252
15. Armstrong AC, Gidding S, Gjesdal O, Wu C, Bluemke DA, Lima JA. LV mass assessed by echocardiography and CMR, cardiovascular outcomes, and medical practice. *JACC Cardiovasc Imaging*. 2012;5(8):837-848. https://doi.org/10.1016/j.jcmg.2012.06.003 PMID:22897998
16. Urbina EM, Khoury PR, McCoy C, Daniels SR, Kimball TR, Dolan LM. Cardiac and vascular consequences of pre-hypertension in youth. *J Clin Hypertens* (Greenwich). 2011;13(5):332-342. https://doi.org/10.1111/j.1751-7176.2011.00471.x PMID:21545394
17. Armstrong AC, Gidding S, Gjesdal O, Wu C, Bluemke DA, Lima JA. LV mass assessed by echocardiography and CMR, cardiovascular outcomes, and medical practice. *JACC Cardiovasc Imaging*. 2012;5(8):837-848. https://doi.org/10.1016/j.jcmg.2012.06.003 PMID:22897998
18. Grossman A, Prokupetz A, Koren-Morag N, Grossman E, Shamiss A. Comparison of usefulness of Sokolow and Cornell criteria for left ventricular hypertrophy in subjects aged <20 years versus >30 years. *Am J Cardiol*. 2012;110(3):440-444. https://doi.org/10.1016/j.amjcard.2012.03.047 PMID:22534054
19. Chobanian AV, Roccella EJ. The JNC 7 hypertension guidelines. *JAMA*. 2003;290(10):1312b-1312. https://doi.org/10.1001/jama.290.10.1314-c
20. Adler AJ, Taylor F, Martin N, Gottlieb S, Taylor RS, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease. *Cochrane Database Syst Rev*. 2014;(12):CD009217. https://doi.org/10.1002/14651858.CD009217.pub3 PMID:25519688
21. Torrance B, McGuire KA, Lewanczuk R, McGavock J. Overweight, physical activity and high blood pressure in children: a review of the literature. *Vasc Health Risk Manag*. 2007;3(1):139-149. PMID:17583184
22. Paruthi S, Brooks LJ, D'Ambrosio C, et al. Consensus statement of the American Academy of Sleep Medicine on the recommended amount of sleep for healthy children: methodology and discussion. *J Clin Sleep Med*. 2016;12(11):1549-1561. https://doi.org/10.5664/jcsm.6288 PMID:27707447
23. Shatat IF, Flynn JT. Hypertension in children with chronic kidney disease. *Adv Chronic Kidney Dis*. 2005;12(4):378-384. https://doi.org/10.1053/j.ackd.2005.07.002 PMID:16198277
24. Wühl E, Mehls O, Schaefer F; ESCAPE Trial Group. Antihypertensive and antiproteinuric efficacy of ramipril in children with chronic renal failure. *Kidney Int*. 2004;66(2):768-776. https://doi.org/10.1111/j.1523-1755.2004.00802.x PMID:15253732
25. Klingensmith GJ, Connor CG, Ruedy KJ, et al.; Pediatric Diabetes Consortium. Presentation of youth with type 2 diabetes in the Pediatric Diabetes Consortium. *Pediatr Diabetes*. 2016;17(4):266-273. https://doi.org/10.1111/pedi.12281 PMID:25951940
26. Orchard TJ, Forrest KYZ, Kuller LH, Becker DJ; Pittsburgh Epidemiology of Diabetes Complications Study. Lipid and blood pressure treatment goals for type 1 diabetes: 10-year incidence data from the Pittsburgh Epidemiology of Diabetes Complications Study. *Diabetes Care*. 2001;24(6):1053-1059. https://doi.org/10.2337/diacare.24.6.1053 PMID:11375370
27. Nambam B, DuBose SN, Nathan BM, et al.; T1D Exchange Clinic Network. Therapeutic inertia: underdiagnosed and undertreated hypertension in children participating in the T1D Exchange Clinic Registry. *Pediatr Diabetes*. 2016;17(1):15-20. https://doi.org/10.1111/pedi.12231 PMID:25330905
28. Krmar RT, Berg UB. Blood pressure control in hypertensive pediatric renal transplants: role of repeated ABPM following transplantation. *Am J Hypertens*. 2008;21(10):1093-1099. https://doi.org/10.1038/ajh.2008.251 PMID:18704114
29. Suszynski TM, Rizzari MD, Gillingham KJ, et al. Antihypertensive pharmacotherapy and long-term outcomes in pediatric kidney transplantation. *Clin Transplant*. 2013;27(3):472-480. https://doi.org/10.1111/ctr.12125 PMID:23647497
30. Arbeiter K, Pichler A, Stemberger R, et al. ACE inhibition in the treatment of children after renal transplantation. *Pediatr Nephrol*. 2004;19(2):222-226. https://doi.org/10.1007/s00467-003-1317-8 PMID:14673630