Sociedad Argentina de Pediatría

Por un niño sano en un mundo mejor

HIPERTENSIÓN ARTERIAL EN NIÑOS Y ADOLESCENTES

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SAP

Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents

American Academy of Pediatrics

DEDICATED TO THE HEALTH OF ALL CHILDREN™

….. to address the following key questions:

Flynn JT, Kaelber DC, Baker-Smith CM, et al.
Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents.
*Pediatrics.***2017**;140(3):e20171904

….. to address the following key questions:

- What is the **optimal approach to diagnose HTN** in children & adolescents?
- How do we best identify the **underlying etiologies of secondary HTN** in children?
- What is **the optimal goal** systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) for children and adolescents?
- In children 0 to 18 years of age, **how does treatment** with lifestyle versus antihypertensive agents **influence indirect measures of cardiovascular disease risk**?

Epidemiology and Clinical Significance

Prevalence of HTN in Children

- The actual **prevalence** of clinical HTN in children and adolescents is **~3.5%.**
- The prevalence of **persistently elevated BP** (formerly termed "prehypertension" including BP values from the 90th to 94th percentiles or between 120/80 and 130/80 mm Hg in adolescents) is also **~2.2% to 3.5%,** with **higher rates** among children and adolescents who have **overweight and obesity.**
- Data on BP **tracking from childhood to adulthood** demonstrate that higher BP in childhood correlates with higher BP in adulthood & the onset of HTN in young adulthood.
- The data confirm the **association of elevated BP in adolescence with HTN in early adulthood** & that normal BP in childhood is associated with a lack of HTN in midadulthood.

Epidemiology and Clinical Significance

Prevalence of HTN Among Children With Various Chronic Conditions

Children With Obesity

- HTN **prevalence** ranges from **3.8% to 24.8%** in youth with **overweight and obesity**.
- **Rates of HTN increase** in a graded fashion with **↑adiposity & waist circumference.**
- **Obesity** is also associated with **a lack of circadian variability of BP**, with up to 50% of children who have obesity not experiencing the expected nocturnal BP dip.
- Studies shown that **childhood obesity is related to the development of future HTN**. This risk appears to increase with obesity severity.
- The data underscore the **importance of monitoring BP** in all children with **overweight** and/or **obesity at every clinical encounter.**
- **Obesity in children with HTN may be accompanied by additional cardiometabolic risk factors** (eg, dyslipidemia and disordered glucose metabolism) that may have their own effects on BP or may represent comorbid conditions arising from the same adverse lifestyle behaviors.

Epidemiology and Clinical Significance

Prevalence of HTN Among Children With Various Chronic Conditions

Children With Sleep-Disordered Breathing

- SDB occurs on a **spectrum** that includes:
  - primary snoring,
  - sleep fragmentation
  - obstructive sleep apnea syndrome (OSAS)
- Numerous studies have identified an **association between SDB and HTN** in the pediatric population. Small studies of youth with sleep disorders have found the prevalence of high BP to range between **3.6% and 14%.**
- Even **inadequate duration of sleep and poor-quality sleep** have been associated with elevated BP. Studies suggest that children who sleep **≤7 hours / night are at increased risk for HTN.**
- The **more severe** the OSAS, the **more likely** a child is to have **HTN.**

Epidemiology and Clinical Significance

Prevalence of HTN Among Children With Various Chronic Conditions

Children With Chronic Kidney Disease

- There are well-established **pathophysiologic links** between childhood HTN and CKD.
- **Untreated HTN can lead to CKD in adults**, although evidence for the latter in pediatric patients is lacking.
- **Among children and adolescents with CKD, ~50% are known to be hypertensive.**
- In children and adolescents with **end-stage renal disease** (either those on dialysis or after transplant), **48% to 79% are hypertensive**, **20% to 70%** having **uncontrolled HTN.**
- **Almost 20% of pediatric HTN may be attributable to CKD.**

Epidemiology and Clinical Significance

Prevalence of HTN Among Children With Various Chronic Conditions

Children With History of Prematurity

- Abnormal birth history—including **preterm birth and low birth weight**— has been identified as a **risk factor for HTN and other CVD in adults**.
- **Only low birth weight** has been **associated with elevated BP in the pediatric age range.**
- One **retrospective cohort** study showed a prevalence of **HTN of 7.3% among 3 year olds who were born preterm.**
- Researchers in another **retrospective case series** noted a **high prevalence of HTN in older children with a history of preterm birth.**
- It appears that **preterm birth** may result in **abnormal circadian BP patterns in childhood**.
- **These data are intriguing but limited. Further study is needed to determine how often preterm birth results in childhood HTN.**

Definition of HTN
(1–18 Years of Age)

BP levels should be interpreted on the basis of sex, age, and heigh

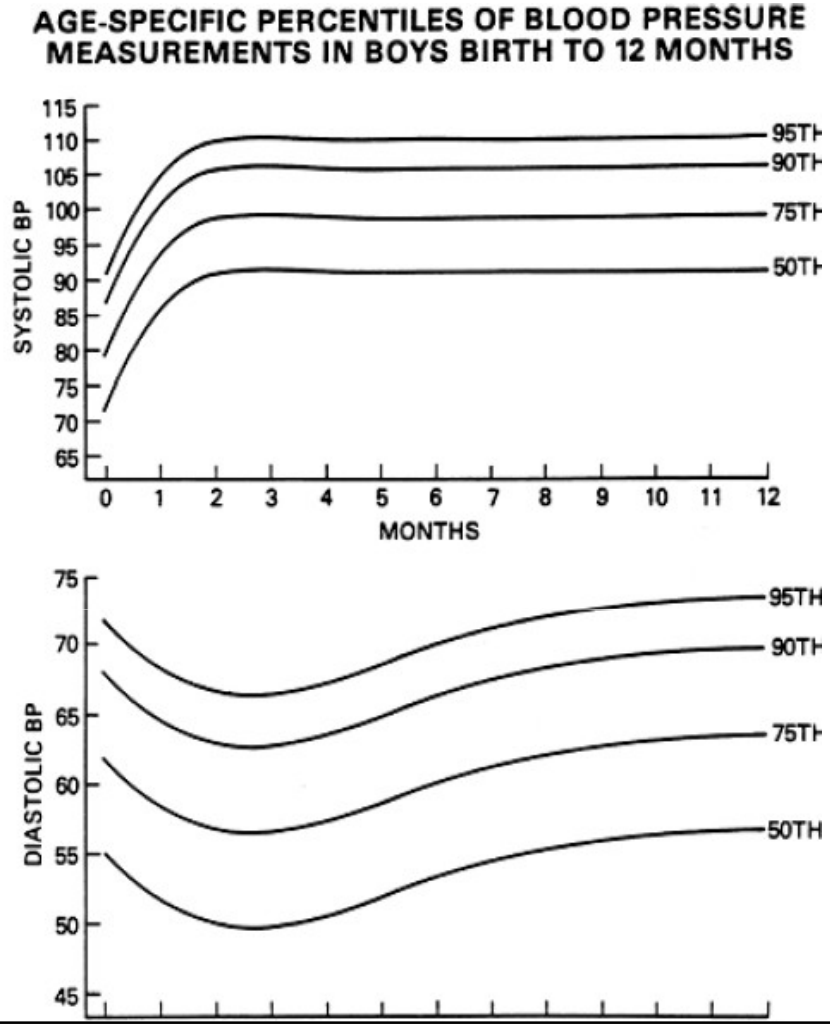
Updated Definitions of BP Categories and Stages

For Children Aged 1–13 y	For Children Aged ≥13 y
Normal BP: <90th percentile	Normal BP: <120/<80 mm Hg
Elevated BP: ≥90th percentile to <95th percentile or 120/80 mm Hg to <95th percentile (whichever is lower)	Elevated BP: 120/<80 to 129/<80 mm Hg
Stage 1 HTN: ≥95th percentile to <95th percentile + 12 mmHg, or 130/80 to 139/89 mm Hg (whichever is lower)	Stage 1 HTN: 130/80 to 139/89 mm Hg
Stage 2 HTN: ≥95th percentile + 12 mm Hg, or ≥140/90 mm Hg (whichever is lower)	Stage 2 HTN: ≥140/90 mm Hg

Screening BP Values Requiring Further Evaluation

Age,	BP, mm Hg			
	Boys		Girls	
	Systolic	DBP	Systolic	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

Definition of HTN in the Neonate and Infant (0–1 Year of Age)



90TH PERCENTILE													
SYSTOLIC BP	87	101	106	106	106	105	105	105	105	105	105	105	105
DIASTOLIC BP	68	65	63	63	63	65	66	67	68	68	69	69	69
HEIGHT CM	51	59	63	66	68	70	72	73	74	76	77	78	80
WEIGHT KG	4	4	5	5	6	7	8	9	9	10	10	11	11

Report of the second task force on blood pressure control in children–1987.Task force on blood pressure control in children. National Heart, Lung, and Blood Institute, Bethesda, Maryland.*Pediatrics*. 1987;79(1):1–25

Measurement of BP

BP Measurement Technique

- **BP in childhood may vary considerably** between visits & during the same visit.
- There are many potential etiologies for **isolated elevated BP** in children and adolescents, including such factors as **anxiety and recent caffeine intake.**
- **BP generally decreases with repeated measurements during a single visit**, although the variability may not be large enough to affect BP classification.
- Important to obtain **multiple measurements over time before diagnosing HTN.**

BP measurement are illustrated in an AAP video available at
https://www.youtube.com/watch?v=JLzkNBpqwi0&feature=youtu.be

Best BP Measurement Practices

- The child should be **seated** in a quiet room for 3–5 min before measurement, with the back supported and **feet uncrossed** on the floor.
- BP should be measured in the **right arm** for consistency, for comparison with **standard tables**, and to **avoid a falsely low reading from the left arm in** the case of **coarctation of the aorta**.
- The **arm** should be **at heart level**, **90 supported**, and uncovered above the cuff.
- The patient and observer should **not speak** while the measurement is being taken.
- The **correct cuff size** should be used. The bladder **length** should be 80%–100% of the arm, and the **width** should be at least 40%.

Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation*. 2005;111(5):697–716.

Best BP Measurement Practices

- For an auscultatory BP, the **bell** of the stethoscope should be placed **over the brachial artery** in the antecubital fossa, and the lower end of the cuff should be 2–3 cm above the antecubital fossa.
- The **cuff should be inflated to 20–30 mm Hg above the point at which the radial pulse disappears.** Overinflation should be avoided.
- The cuff should be deflated at a rate of 2–3 mm Hg per second. **The first (phase I Korotkoff) and last (phase V Korotkoff) audible sounds should be taken as SBP and DBP.** If the Korotkoff sounds are heard to 0 mm Hg, the point at which the sound is muffled (phase IV Korotkoff) should be taken as the DBP, or the measurement repeated with less pressure applied over the brachial artery. The measurement should be read to the nearest 2 mm Hg.
- **To measure BP in the legs**, the patient should be in the **prone position**, if possible. An appropriately sized **cuff should be placed mid thigh** and the stethoscope placed over the **popliteal artery**. The SBP in the legs is **usually 10%–20% higher than the brachial artery pressure.**

Pickering TG, Hall JE, Appel LJ, et al. Recommendations for blood pressure measurement in humans and experimental animals: part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation*. 2005;111(5):697–716.

BP Measurement Frequency

- It is **unclear what age is optimal to begin routine BP measurement** in children
- Recommendation: **beginning** measure BP in ambulatory setting **at 3 years of age**.
- For otherwise healthy children, **BP need only be measured annually** rather than during every health care encounter.
- Some children should have BP measured **at every health encounter**, specifically those with **obesity** (BMI ≥95 percentile), **renal disease, diabetes, aortic arch obstruction or coarctation**, or those who are taking **medications** that increase BP.
- Children **younger than 3 years** should have BP measurements taken at well-child care visits **if they are at increased risk for developing HTN**.

Conditions Under Which Children Younger Than 3 Years Should Have BP Measured

History of prematurity <32 week's gestation or small for gestational age, very low birth weight, other neonatal complications requiring intensive care, umbilical artery line
Congenital heart disease (repaired or unrepaired)
Recurrent urinary tract infections, hematuria, or proteinuria
Known renal disease or urologic malformations
Family history of congenital renal disease
Solid-organ transplant
Malignancy or bone marrow transplant
Treatment with drugs known to raise BP
Other systemic illnesses associated with HTN (neurofibromatosis, tuberous sclerosis, sickle cell disease,[114] etc)
Evidence of elevated intracranial pressure

Patient Management on the Basis of Office BP

Normal BP

If BP is normal or normalizes after repeat readings (ie, BP <90th percentile)

- no additional action is needed
- practitioners should measure the BP at the next routine well-child care visit

Patient Management on the Basis of Office BP

Elevated BP

- If the BP reading is **at the elevated BP level lifestyle interventions should be recommended** (ie, healthy diet, sleep, and physical activity); the **measurement should be repeated in 6 months** by auscultation. **Nutrition &/or weight management referral should be considered**
- **If BP remains at the elevated BP level after 6 months**, upper and lower extremity BP should be rechecked (**right arm, left arm, and 1 leg**), **lifestyle counseling** should be repeated, and **BP should be rechecked in 6 months**.
- **If BP continues at the elevated BP level after 12 months** (after 3 auscultatory measurements), **ABPM** should be ordered and diagnostic evaluation should be conducted . **Consider subspecialty referral** (ie, cardiology or nephrology) .
- **If BP normalizes at any point, return to annual BP screening at well-child care visits.**

Patient Management on the Basis of Office BP

Stage 1 HTN

- If the BP reading is at the **stage 1** HTN level and the patient is **asymptomatic**, provide **lifestyle counseling** and **recheck the BP in 1 to 2 weeks** by auscultation.
- If the BP reading is **still at the stage 1 level**, upper and lower extremity BP should be checked (**right arm, left arm, and 1 leg**), and BP should be **rechecked in 3 months** by auscultation. **Nutrition and/or weight management referral should be considered** .
- If BP continues to be at the **stage 1 HTN level after 3 visits**,
  - **ABPM** should be ordered
  - **diagnostic evaluation** should be conducted
  - **treatment** should be initiated
  - **subspecialty referral** should be considered

Patient Management on the Basis of Office BP

Stage 2 HTN

- upper and lower extremity BP should be checked (**right arm, left arm, and 1 leg**),
- **lifestyle recommendations** given,
- the **BP measurement should be repeated within 1 week**.
- alternatively, the patient could be referred to **subspecialty care** within 1 week.

- If the BP reading is **still at the stage 2** HTN level when repeated:
  - **diagnostic evaluation**, including **ABPM**, should be conducted
  - **treatment** should be initiated,
  - or the patient should be **referred to subspecialty care** within 1 week.

- If the BP reading is at the **stage 2** HTN level and the patient is **symptomatic**, or the **BP is >30 mm Hg above the 95th percentile (or >180/120 mm Hg in an adolescent)**,
  - **refer to an immediate source of care, such as an emergency department**

- Trained health care professionals **in the office setting** should **make a diagnosis** of HTN if a child or adolescent has **BP readings ≥95th percentile on 3 different visits.**

Ambulatory BP Monitor

- An ambulatory BP monitor **consists of a BP cuff attached to a box** slightly larger than a cell phone, **which records BP periodically (usually every 20–30 minutes) throughout the day and night;** these data are later **downloaded to a computer for analysis.**
- ABPM has been **recommended** by the US Preventive Services Task Force for the confirmation of HTN **in adults** before starting treatment. Although a growing number of pediatric providers have access to ABPM, **there are still gaps** in access and knowledge regarding the **optimal application of ABPM to the evaluation of children's BP**.
- However, **sufficient data exist** to demonstrate that **ABPM is more accurate for the diagnosis of HTN than clinic-measured BP**, **is more predictive of future BP**, & **can assist in the detection of 2*HTN.** Furthermore, **increased LVMI and LVH correlate >strongly with ABPM parameters than casual BP**, & ABPM is **>reproducible** than casual or home BP measurement.
  - **for these reasons, the routine application of ABPM is recommended.**
- For **technical reasons**, ABPM may need to be limited to **children ≥5 years of age** who can tolerate the procedure and those for whom reference data are available.

Ambulatory BP Monitor

- **The routine performance of ABPM should be strongly considered in children & adolescents with high-risk conditions to assess HTN severity and determine if abnormal circadian BP patterns are present, which may indicate increased risk for target organ damage.**
- ***Masked Hypertension*** occurs when patients have **normal office BP but elevated BP on ABPM,** and it has been found **in 5.8% of unselected children** studied by ABPM. There is growing evidence that compared with those with normal 24-hour BP, these patients have significant risk for end organ hypertensive damage. Patients who are at risk of MH include patients with **obesity** and **secondary forms of HTN,** such as CKD or repaired aortic coarctation. MH is **particularly prevalent** in patients **with CKD and is associated with target organ damage.**
  - **Children with CKD should be periodically evaluated using ABPM for MH as part of routine CKD management.**
- ***White Coat Hypertension*** WCH is defined as BP ≥95th percentile in the office or clinical setting but <95th percentile outside of the office or clinical setting. WCH is diagnosed by ABPM when the mean SBP and DBP are <95th percentile and SBP and DBP load are <25%; load is defined as the percentage of valid ambulatory BP measurements above a set threshold value (eg, 95th percentile) for age, sex, and height. It is estimated that up to half of children who are evaluated for elevated office BP have WCH.
  - **Children and adolescents with WCH should have screening BP measured at regular well-child care visits with consideration of a repeat ABPM in 1 to 2 years.**

High-Risk Conditions for Which ABPM May Be Useful

Condition	Rationale
Secondary HTN	Severe ambulatory HTN or nocturnal HTN indicates higher likelihood of secondary HTN[161,167]
CKD or structural renal abnormalities	Evaluate for MH or nocturnal HTN,[168–172] better control delays progression of renal disease[173]
T1DM and T2DM	Evaluate for abnormal ABPM patterns,[174,175] better BP control delays the development of MA[176–178]
Solid-organ transplant	Evaluate for MH or nocturnal HTN, better control BP[179–188]
Obesity	Evaluate for WCH and MH[23,189–192]
OSAS	Evaluate for nondipping and accentuated morning BP surge[43,46,193,194]
Aortic coarctation (repaired)	Evaluate for sustained HTN and MH[58,112,113]
Genetic syndromes associated with HTN (neurofibromatosis, Turner syndrome, Williams syndrome, coarctation of the aorta)	HTN associated with increased arterial stiffness may only be manifest with activity during ABPM[58,195]
Treated hypertensive patients	Confirm 24-h BP control[155]
Patient born prematurely	Evaluate for nondipping[196]
Research, clinical trials	To reduce sample size[197]

At-Home Measurement

- Home measurement (or selfmonitoring) of BP has **advantages over both office and ambulatory monitoring**:
  - **convenience** & **ability to repeat measurements over time.**
- Furthermore, **automated devices with memory capacity** are straightforward to use.
- **Home BP monitoring should not be used to diagnose HTN, MH, or WCH** but may be a **useful** adjunct to office and ambulatory BP measurement **after HTN has been diagnosed.**

Primary and Secondary Causes of HTN

Primary HTN

- Primary HTN is now the **predominant diagnosis for hypertensive children and adolescents** seen **in referral centers in the United States**, although **single-center studies from outside the United States still find primary HTN to be uncommon.**
- **General characteristics of children with primary HTN** include:
  - **older age (≥6 years)**
  - **positive family history** (in a parent and/or grandparent) of HTN,
  - **overweight** &/or **obesity**
- **Severity** of BP elevation **has not differed** significantly between children with primary and secondary HTN in some studies, but **DBP elevation appears to be more predictive of secondary HTN,** whereas **systolic HTN appears to be more predictive of primary HTN.**
- **Children and adolescents ≥6 years of age do not require an extensive evaluation for secondary causes of HTN if :**
  - they have a positive family history of HTN
  - are overweight or obese
  - don't have history or physical examination findings suggestive of a 2* cause of HTN

Secondary Causes: Renal and/or Renovascular

- **Renal disease and renovascular disease:**
  - among the **most common secondary causes of HTN in children**.
- **Renal parenchymal disease and renal structural abnormalities** accounted for **34% to 79% of patients with secondary HTN** in 3 retrospective, single-center case series, and **renovascular disease** was present in **12% to 13%.**
- **It is appropriate to have a high index of suspicion for renal and renovascular disease in hypertensive pediatric patients, particularly in those <6 years of age.**

Secondary Causes: Aortic Coarctation

- **Coarctation of the aorta** is a congenital abnormality of the aortic arch characterized by discrete narrowing of the aortic arch, generally at the level of the aortic isthmus, usually associated with HTN & **right arm BP is ≥20 mm Hg greater than the lower extremity BP.**
- **Long-segment narrowing of the abdominal aorta** can also cause HTN and should be considered in children with **refractory HTN and a gradient between the upper and lower extremities** . Of note, children with abdominal aortic obstruction may have **Neurofibromatosis, Williams syndrome, Alagille syndrome, or Takayasu arteritis.**
- Patients with **coarctation can remain hypertensive or develop HTN even after early and successful repair**. **HTN can be a manifestation of recoarctation**.
- The **prevalence of HTN** increases over time after successful coarctation repair.
- **Children and adolescents who have undergone coarctation repair should undergo ABPM** for the detection of HTN, **at most, 12 years after coarctation repair**.
  Earlier screening may be considered on the basis of risk factors and clinician discretion.

Hypertension in Coarctation of the Aorta: Challenges in Diagnosis in Children

Pediatr Cardiol (2018) 39:1–10

Systemic arterial hypertension following coarctation repair is common &often observed even in patients with a successful COA repair with minimal or no residual gradient in the aortic arch

Prevalence

- during **childhood** following early repair of COA is ± **30%**
- up to **68%** in long-term follow-up studies **in young adults**

Hypertension in Coarctation of the Aorta: Challenges in Diagnosis in Children

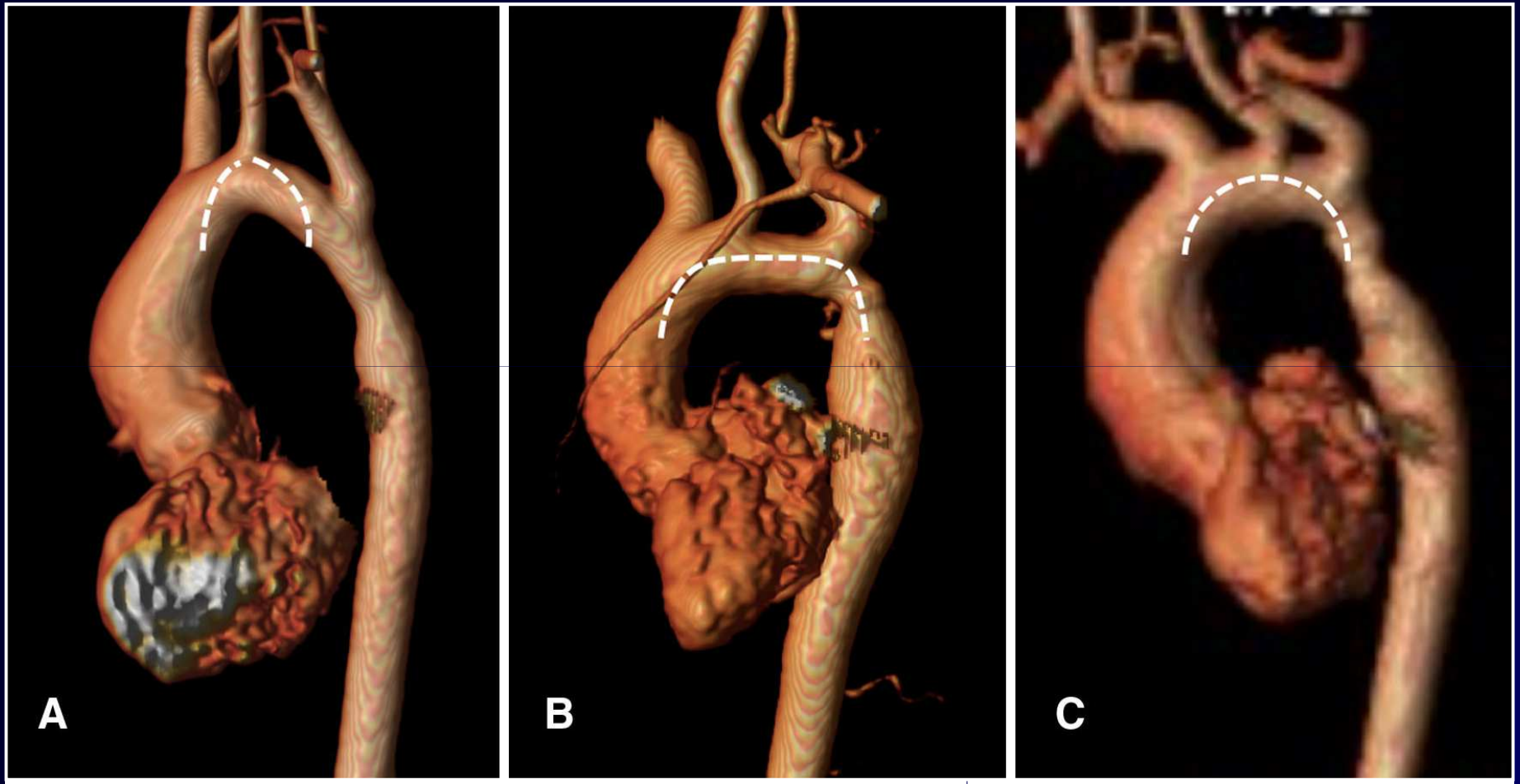
Pediatr Cardiol (2018) 39:1–10

Aetiology → multifactorial

- **Aortic arch morphology**
- **Surgical techniques**
- **Innate abnormalities of the aortic wall**
- **Residual aortic pressure gradients following repair**
- **Altered regulation of renin–angiotens & baroreceptor system**
- **Age at presentation and the timing of surgery**

Hypertension in Coarctation of the Aorta: Challenges in Diagnosis in Children

Pediatr Cardiol (2018) 39:1–10



Gothic aortic arch	Crenel aortic arch	Romanesque aortic arch

Secondary Causes: Endocrine HTN

- HTN resulting from **hormonal excess** accounts for a **relatively small proportion** of children with 2*HTN (prevalence **0.05% to 6%**). An accurate **diagnosis of endocrine HTN provides a unique treatment opportunity** to surgical cure or pharmacologic therapy.

Secondary Causes: Environmental Exposures

- Several environmental exposures (e.g: **lead, cadmium, mercury, and phthalates)** are associated with ↑childhood BP, although most studies are limited to small case series.

Secondary Causes: Neurofibromatosis

- **Neurofibromatosis type 1** (Von Recklinghausen disease) is a rare autosomal dominant disorder with **potential secondary causes of HTN**, most commonly **renal artery stenosis**, and also **coarctation of the aorta, middle aortic syndrome**, and **pheochromocytoma**. Additionally, an ↑incidence of **idiopathic HTN** was documented in NF-1,as high as 6.1%.

Secondary Causes: Medication Related

Over-the-counter drugs	Decongestants
	Caffeine
	Nonsteroidal anti-inflammatory drugs
	Alternative therapies, herbal and nutritional supplements
Prescription drugs	Stimulants for attention-deficit/hyperactivity disorder
	Hormonal contraception
	Steroids
	Tricyclic antidepressants
Illicit drugs	Amphetamines
	Cocaine

Secondary Causes: Monogenic HTN

- Monogenic forms of HTN are **uncommon**, although the **exact incidence** is **unknown**.
- In a study of select hypertensive children without a known etiology, genetic testing for **familial hyperaldosteronism type I** (FH-I), or **glucocorticoid remediable aldosteronism**, confirmed responsible **genetic mutations in 3%** of the population.
- **Other** monogenic forms of HTN : **Liddle syndrome**, **pseudohypo - aldosteronism type II (Gordon syndrome), apparent mineralocorticoid excess, familial glucocorticoid resistance, mineralocorticoid receptor activating mutation, and congenital adrenal hyperplasia**.
- **All manifest as HTN with suppressed plasma renin activity (PRA) and increased sodium absorption in the distal tubule**. Other features may include **serum potassium abnormalities, metabolic acid-base disturbances, and abnormal plasma aldosterone concentrations,** although the **clinical presentations can be highly variable.**
- **Suspect Monogenic forms of HTN in children with a suppressed Plasma Renin Activity or elevated Aldosterone-to-Renin Ratio** , especially with a **family history of early-onset HTN**.

Diagnostic Evaluation : Patient Evaluation

- As with any medical condition, appropriate diagnostic evaluation is critical to determine possible causes &/or comorbidities associated with HTN.
  - **patient & family history, physical examination, laboratory evaluation, & imaging**.
- The **first step** is to obtain a history:
  - **perinatal, past medical, nutritional - activity & psychosocial history.**

Patient Evaluation : *Perinatal History*

- Perinatal factors such as **maternal HTN** and **low birth weight** have been shown to influence later BP, even in childhood. Additionally, a high incidence of **preterm birth** among hypertensive children has recently been reported. Thus, it is appropriate to obtain a history of pertinent prenatal information:
  - maternal pregnancy complications; gestational age; birth weight;
  - complications occurring in the neonatal nursery
  - document pertinent procedures, such as umbilical catheter placement.

Diagnostic Evaluation

Patient Evaluation : *Nutritional History*

- **High sodium intake** is linked to childhood HTN & increased LVMI and is the focus of several population health campaigns.
  - In **NHANES** 2003–2008, among children 8 to 18 years of age (*n* = 6235), higher sodium intake was associated with a **twofold increase in the combined outcome of elevated BP or HTN** & **threefold** in participants with **obesity.** Limited data suggest the same effect is in **younger children.**
- ↑ **intake of total fat /saturated fat** & **adiposity /central obesity**: also predictors of SBP.
- **Nutrition history is an important part of the assessment because it may identify dietary contributors to HTN & detect areas in which lifestyle modification may be appropriate.**
- **Important components to discuss** are:
  - **salt intake** (kitchen /at the table/Na hidden in processed & fast food)
  - consumption of **high-fat foods**, & **sugary beverages**
  - infrequent consumption of **fruits, vegetables, and low-fat dairy products**

Diagnostic Evaluation

Patient Evaluation : *Psychosocial History*

- **Adverse experiences** both prenatally and during childhood (including maltreatment, early onset depression, and anxiety) are associated with adult-onset HTN.
- The psychosocial history should include feelings of depression / anxiety, bullying, & body perceptions. The latter is particularly important for patients with overweight or **obesity** because **~70% of these children report having bullying and body perception concerns.**
- Starting at **11 years of age**, the psychosocial history should include questions about
  - smoking, alcohol, and other drug use.

Patient Evaluation : *Family History*

- **To risk-stratify pediatric patients with an increased risk for HTN**

Diagnostic Evaluation

Patient Evaluation : *Physical Examination*

- **May provide clues to 2*causes of HTN & assess possible hypertensive end organ damage**.
- The child's **height, weight, BMI, and percentiles for age** should be determined at the start of the physical examination. **Poor growth** may indicate an underlying chronic illness.
- At the **2nd visit with confirmed elevated BP or stage 1 HTN**, or the **1st visit** with **stage 2 HTN**,
  - BP should be measured in both arms and in a leg.
  - Normally, BP is 10 to 20 mm Hg higher in the legs than the arms.
- **If the leg BP is <than the arm BP, or if femoral pulses are weak or absent:**
  - coarctation of the aorta may be present.
- **The physical examination in HTN children is frequently normal except for the BP ↑**.

Examples of Physical Examination Findings and History Suggestive of Secondary HTN or Related to End Organ Damage

Body System	Finding, History	Possible Etiology
Vital signs	Tachycardia	Hyperthyroidism
		PCC
		Neuroblastoma
	Decreased lower extremity pulses; drop in BP from upper to lower extremities	Coarctation of the aorta
Eyes	Proptosis	Hyperthyroidism
	Retinal changes[a]	Severe HTN, more likely to be associat with secondary HTN
Ear, nose, throat	Adenotonsillar hypertrophy	SDB
	History of snoring	Sleep apnea
Height, weight	Growth retardation	Chronic renal failure
	Obesity (high BMI)	Cushing syndrome
	Truncal obesity	Insulin resistance syndrome
Head, neck	Elfin facies	Williams syndrome
	Moon facies	Cushing syndrome
	Thyromegaly, goiter	Hyperthyroidism
	Webbed neck	Turner syndrome
Skin	Pallor, flushing, diaphoresis	PCC
	Acne, hirsutism, striae	Cushing syndrome
		Anabolic steroid abuse
	Café-au-lait spots	Neurofibromatosis
	Adenoma sebaceum	Tuberous sclerosis
	Malar rash	Systemic lupus
	Acanthosis nigricans	T2DM
Hematologic	Pallor	Renal disease

Examples of Physical Examination Findings and History Suggestive of Secondary HTN or Related to End Organ Damage

Chest, cardiac	Chest pain	Heart disease
	Palpitations	
	Exertional dyspnea	
	Widely spaced nipples	Turner syndrome
	Heart murmur	Coarctation of the aorta
	Friction rub	Systemic lupus (pericarditis)
		Collagen vascular disease
	Apical heave[a]	LVH
Abdomen	Abdominal mass	Wilms tumor
		Neuroblastoma
		PCC
	Epigastric, flank bruit	RAS
	Palpable kidneys	Polycystic kidney disease
		Hydronephrosis
		Multicystic dysplastic kidney

Examples of Physical Examination Findings and History Suggestive of Secondary HTN or Related to End Organ Damage

Genitourinary	Ambiguous or virilized genitalia	Congenital adrenal hyperplasia
	Urinary tract infection	Renal disease
	Vesicoureteral reflux	
	Hematuria, edema, fatigue	
	Abdominal trauma	
Extremities	Joint swelling	Systemic lupus
		Collagen vascular disease
	Muscle weakness	Hyperaldosteronism
		Liddle syndrome
Neurologic, metabolic	Hypokalemia, headache, dizziness, polyuria, nocturia	Reninoma
	Muscle weakness, hypokalemia	Monogenic HTN (Liddle syndrome, GRA, AME)

Diagnostic Evaluation

Laboratory Evaluation

- The purpose of the laboratory evaluation is **to identify underlying secondary causes of HTN** (eg, renal or endocrine disease) that would require specific treatment guided by a subspecialist.
- In general, such testing includes a basic **set of screening tests and additional, specific tests**; the latter are selected on the basis of clues obtained from the history and physical examination and/or the results of the initial screening tests.

Patient Management on the Basis of Office BP

Screening Tests and Relevant Populations

Patient Population	Screening Tests
All patients	Urinalysis
	Chemistry panel, including electrolytes, blood urea nitrogen, and creatinine
	Lipid profile (fasting or nonfasting to include high-density lipoproteina and total cholesterol)
	Renal ultrasonography in those <6 y of age or those with abnormal urinalysis or renal function
In the obese (BMI >95th percentile) child or adolescent, in addition to the above	Hemoglobin A1c (accepted screen for diabetes)
	Aspartate transaminase and alanine transaminase (screen for fatty liver)
	Fasting lipid panel (screen for dyslipidemia)
Optional tests to be obtained on the basis of history, physical examination, and initial studies	Fasting serum glucose for those at high risk for diabetes mellitus
	Thyroid-stimulating hormone
	Drug screen
	Sleep study (if loud snoring, daytime sleepiness, or reported history of apnea)
	Complete blood count, especially in those with growth delay or abnormal renal function

Adapted from Wiesen J, Adkins M, Fortune S, et al. Evaluation of pediatric patients with mild-to-moderate hypertension: yield of diagnostic testing. *Pediatrics*. 2008;122(5). Available at: www.pediatrics.org/cgi/content/full/122/5/e988.

Diagnostic Evaluation

ECG

- **High specificity but poor sensitivity** for identifying LVH.
- ECG **positive predictive value** to identify LVH is **extremely low**.
- **Clinicians should not perform ECG in hypertensive children and adolescents being evaluated for LVH.**

Imaging Evaluation

Echocardiography: Detection of Target Organ Damage

- To measure left ventricular target organ injury related to HTN
- Basis for this assessment:
  - the relationship of LV mass to BP
  - independent strong relation LVH /adverse adults CVD outcomes
- Antihypertensive treatment reduces LVH.
- Observational data suggest that regression of LVH independently predicts outcomes in adults.

- **LVEF** may be ↓↓ in severe or acute onset HTN with associated congestive heart failure.
- Rarely, LVEF may be mildly depressed in chronic HTN

Imaging Evaluation

Echocardiography: *Key Action*

- Performed ECHO to assess for cardiac target organ damage (LV mass, geometry, & function) **at the time of consideration of pharmacologic treatment** of HTN.
- **LVH should be defined** as:
  - LV mass >51 g/m (boys and girls) for children and adolescents >than 8 years ,
  - LV mass >115 g/BSA in boys
  - LV mass >95 g/BSA in girls.
- **Repeat** ECHO to monitor improvement or progression of target organ damage
  - at 6- to 12-month intervals.

Diagnostic Evaluation

Vascular Structure and Function

- Emerging data demonstrate association of **BP higher levels in youth with adverse changes in measures of vascular structure & function**, including **ultrasonography** of the **cIMT, PWV**, a robust measure of central arterial stiffness related to hard CV events in adults (eg, stroke, myocardial infarction, etc), & **FMD**.
- **Insufficient normative data** are available to define clinically actionable cut-points between normal and abnormal for these vascular parameters **in children**.
- **The routine measurement of vascular structure and function to stratify risk in hypertensive youth cannot be recommended at this time.**

Diagnostic Evaluation

Imaging for Renovascular Disease

- **There are no evidence-based criteria for the identification of children and adolescents who may be more likely to have RAS.**
- Some experts will do a more extensive evaluation for RAS in children and adolescents with **stage 2 HTN**, those with **significant diastolic HTN** (especially on ABPM), those with **HTN and hypokalemia** on screening laboratories, and those with a **notable size discrepancy between the kidneys** on standard ultrasound imaging.
- **Bruits** over the renal arteries are also suggestive of RAS but are not always present.
- **Consultation with a subspecialist is recommended to decide which patients warrant further investigation & to aid in the selection of the appropriate imaging modality.**

Diagnostic Evaluation: Imaging for Renovascular Disease

Renal Ultrasonography

- **Sensitivity** : 64% to 90%. **Specificity** of 68% to 70%.
- Factors that may affect the accuracy of Doppler ultrasonography include patient cooperation, the technician's experience, the age of the child,and the child's BMI.
- Best results are obtained in older (**≥8 years**), **nonobese** (BMI ≤85th percentile), cooperative children and adolescents who are examined in a facility with extensive pediatric vascular imaging experience.

Key Action

- **Doppler renal ultrasonography may be used as a noninvasive screening study for the evaluation of possible RAS in normal-weight children and adolescents ≥8 years of age who are suspected of having renovascular HTN & will cooperate with the procedure.**

Computed Tomographic Angiography (CTA) & Magnetic Resonance Angiography (MRA)

Diagnostic Evaluation

Uric Acid

- Cross-sectional data have suggested a **relationship** between elevated serum uric acid (UA) levels and HTN.
- These findings suggest that the **↑UA levels** may be viewed
  - as **1 component of CV risk assessment**, especially in those with **obesity**.
- A **causative** role for ↑UA in childhood HTN has not been established.
- **There is currently not sufficient evidence to support the routine measurement of serum UA in the evaluation and management of children with elevated BP.**

Diagnostic Evaluation

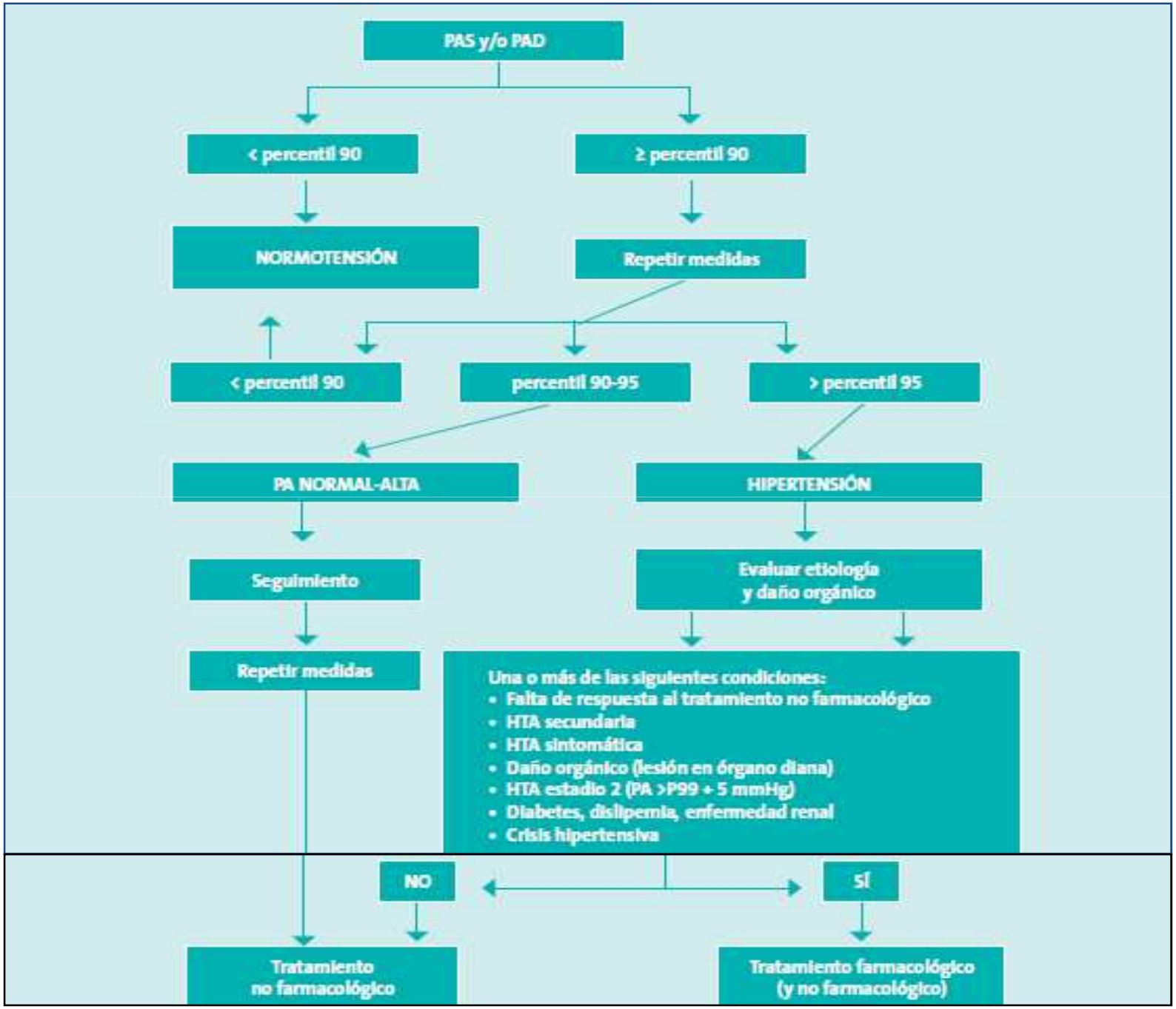
Microalbuminuria

- Microalbuminuria (MA), which should be differentiated from proteinuria in CKD, has been shown to be a **marker of HTN-related kidney injury and a predictor of CVD in adults.**
- MA has been shown to be effectively **reduced via the use of ARBs and ACE inhibitors** in adults. Lowering the degree of MA in adults has been associated with decreased CVD risk.
- **In contrast, data to support a clear relationship between HTN and MA in pediatric patients with primary HTN are limited.**
- MA appears to be a **nonspecific finding in children** that can occur in the absence of HTN; it can occur in children who have obesity, insulin resistance, diabetes,dyslipidemia, and even in those who have recently participated in vigorous physical activity.

Key Action

- **Routine testing for MA is not recommended for children & adolescents with primary HTN**

Algoritmo del manejo diagnóstico y terapéutico de la HTA pediátrica



TREATMENT

Overall Goals

- **To achieve a BP level that not only reduces the risk for target organ damage in childhood but also reduces the risk for HTN and related CVD in adulthood.**
- Several studies have shown that currently **available treatment options can even reverse target organ damage** in hypertensive youth.
- The **previous recommendations** for HTN treatment target in children without CKD or diabetes were **SBP and DBP <95th percentile**. Evidence has emerged that markers of target organ damage, such as increased LVMI, can be detected among some children with BP >90th percentile (or >120/80 mm Hg) but <95th percentile.
- **Therefore, an optimal BP level to be achieved with treatment of childhood HTN is <90th percentile or <130/80 mm Hg in ≥13 years old.**

TREATMENT

Lifestyle and Nonpharmacologic Interventions

- **Lifestyle interventions are recommended to lower BP.**
- There is **good evidence** from studies in **adults** showing that **nutritional interventions lower BP**, including clinical trials demonstrating that:
  - **reducing dietary sodium** results in lower BP and CV mortality
  - a diet high in **olive oil polyphenols** lowers BP.
- Studies of hypertensive **youth** suggest that the relationship between **diet, physical activity**, & BP in childhood is ≅ to adults.

TREATMENT

Lifestyle and Nonpharmacologic Interventions: Diet

- The **Dietary Approaches to Stop Hypertension (DASH**) approach and specific elements of that diet have been the primary dietary strategy tested in the literature.
- These elements include a diet that is **high in fruits, vegetables, lowfat milk products, whole grains, fish, poultry, nuts, and lean red meats; it also includes a limited intake of sugar and sweets along with lower sodium intake .**
- **Cross-sectional studies demonstrate associations between the DASH diet & BP.**
- Population-based data from **NHANES** show correlations between dietary sodium and BP in childhood, particularly with excess weight.
- A high intake of fruits, vegetables, and legumes is associated with lower BP. **A lack of fruit consumption in childhood has been linked to increases in cIMT in young adulthood** in the **Young Finns study**. Higher intake of low-fat dairy products has been associated with lower BP in childhood.
- **Longitudinal, observational, and interventional data – The National Heart Lung & Blood Institute's Growth & Health Study** also support relationships between diet & BP in youth

DASH Diet Recommendations

Food	Servings per Day
Fruits and vegetables	4–5
Low-fat milk products	≥2
Whole grains	6
Fish, poultry, and lean red meats	≤2
Legumes and nuts	1
Oils and fats	2–3
Added sugar and sweets (including sweetened beverages)	≤1
Dietary sodium	<2300 mg per d

Physical activity and sedentary behavior thresholds for identifying childhood hypertension and its phenotypes
The Healthy Growth Study

Journal of the American Society of Hypertension
2018

2473 school children aged 9-13 years

Physical activity is inversely associated with all hypertension phenotypes, whereas sedentary behavior is positively associated with ISH and SDH in boys

TREATMENT

Lifestyle & Nonpharmacologic Interventions: Physical Activity

- Any type of exercise, whether it's aerobic training, resistance training, or combined training, appears to be beneficial.

Key Action

- At the time of diagnosis of elevated BP or HTN in a child or adolescent, clinicians should provide advice on the DASH diet and recommend moderate to vigorous physical activity at least 3 to 5 days per week (30–60 minutes per session) to help reduce BP.

Pharmacologic Treatment

- **Children who remain hypertensive** despite lifestyle modifications
- **Symptomatic** HTN
- **Stage 2 HTN without a clearly modifiable factor (eg, obesity**),
- Any stage of HTN **associated with CKD or diabetes mellitus**

- **Initiate therapy with a single medication at the low end of the dosing range** .
- Depending on repeated BP measurements, **the dose** of the initial medication **can be increased every 2 to 4 weeks** until :
  - BP is controlled (eg, <90th percentile)
  - the maximal dose is reached
  - or adverse effects occur
- **If BP is not controlled with a single agent, a second agent can be added** to the regimen and titrated as with the initial drug.
- Because of the salt and water retention that occurs with many antihypertensive medications, **a thiazide diuretic is often the preferred second agent.**

Recommendations for the Initial Prescription of Antihypertensive Drugs for Outpatient Management of Chronic HTN

Drug	Age	Initial Dose	Maximal Dose	Dosing Interval
ACE inhibitors				
Contraindications: pregnancy, angioedema				
Common adverse effects: cough, headache, dizziness, asthenia				
Severe adverse effects: hyperkalemia, acute kidney injury, angioedema, fetal toxicity				
Benazepril	≥6 y[a]	0.2 mg/kg per d (up to 10 mg per d)	0.6 mg/kg per d (up to 40 mg per d)	Daily
Captopril	Infants	0.05 mg/kg per dose	6 mg/kg per d	Daily to 4 times a day
	Children	0.5 mg/kg per dose	6 mg/kg per d	Three times a day
Enalapril	≥1 mo[a]	0.08 mg/kg per d (up to 5 mg per d)	0.6 mg/kg per d (up to 40 mg per d)	Daily to twice a day
Fosinopril	≥6 y	0.1 mg/kg per d (up to 5 mg per d)	40 mg per d	Daily
	<50 kg			
	≥50 kg[a]	5 mg per d	40 mg per d	
Lisinopril	≥6 y[a]	0.07 mg/kg per d (up to 5 mg per d)	0.6 mg/kg per d (up to 40 mg per d)	Daily
Ramipril	—	1.6 mg/m^2 per d	6 mg/m^2 per d	Daily
Quinapril	—	5 mg per d	80 mg per d	Daily
ARBs				
Contraindications: pregnancy				
Common adverse effects: headache, dizziness				
Severe adverse effects: hyperkalemia, acute kidney injury, fetal toxicity				
Candesartan	1–5 y[a]	0.02 mg/kg per d (up to 4 mg per d)	0.4 mg/kg per d (up to 16 mg per d)	Daily to twice a day
	≥6 y[a]			
	<50 kg	4 mg per d	16 mg per d	
	≥50 kg	8 mg per d	32 mg per d	
Irbesartan	6–12 y	75 mg per d	150 mg per d	Daily
	≥13	150 mg per d	300 mg per d	
Losartan	≥6 y[a]	0.7 mg/kg (up to 50 mg)	1.4 mg/kg (up to 100 mg)	Daily
Olmesartan	≥6 y[a]	—	—	Daily
	<35 kg	10 mg	20 mg	
	≥35 kg	20 mg	40 mg	
Valsartan	≥6 y[a]	1.3 mg/kg (up to 40 mg)	2.7 mg/kg (up to 160 mg)	Daily

Recommendations for the Initial Prescription of Antihypertensive Drugs for Outpatient Management of Chronic HTN

Drug	Age	Initial Dose	Maximal Dose	Dosing Interval
Thiazide diuretics				
Contraindications: anuria				
Common adverse effects: dizziness, hypokalemia				
Severe adverse effects: cardiac dysrhythmias, cholestatic jaundice, new onset diabetes mellitus, pancreatitis				
Chlorthalidone	Child	0.3 mg/kg	2 mg/k per d (50 mg)	Daily
Chlorothiazide	Child[a]	10 mg/kg per d	20 mg/kg per d (up to 375 mg per d)	Daily to twice a day
Hydrochlorothiazide	Child[a]	1 mg/kg per d	2 mg/kg per d (up to 37.5 mg per d)	Daily to twice a day
Calcium channel blockers				
Contraindications: hypersensitivity to CCBs				
Common adverse effects: flushing, peripheral edema, dizziness				
Severe adverse effects: angioedema				
Amlodipine	1–5 y	0.1 mg/kg	0.6 mg/kg (up to 5 mg per d)	Daily
	≥6 y[a]	2.5 mg	10 mg	
Felodipine	≥6 y	2.5 mg	10 mg	Daily
Isradipine	Child	0.05–0.1 mg/kg	0.6 mg/kg (up to 10 mg per d)	Capsule: twice daily to 3 times a day; extended-release tablet: daily
Nifedipine extended release	Child	0.2–0.5 mg/kg per d	3 mg/kg/d (up to 120 mg per d)	Daily to twice a day

Pharmacologic Treatment: *Choice of Agent*

- Pharmacologic treatment of HTN in children and adolescents should be **initiated** with an **ACE inhibitor, ARB, long-acting calcium channel blocker, or a thiazide diuretic**.
- In view of the expanded adverse effect profile and lack of association in adults with improved outcomes compared with other agents, **β-blockers are not recommended as initial treatment in children.**
- **ACE inhibitors and ARBs are contraindicated in pregnancy** because these agents can cause injury and death to the developing fetus. Adolescents of childbearing potential should be informed of the potential risks of these agents on the developing fetus; alternative medications (eg, calcium channel blocker, β-blocker) can be considered when appropriate.
- In children **with HTN and CKD, proteinuria, or diabetes mellitus, an ACE inhibitor or ARB is recommended as the initial antihypertensive agent** unless there is an absolute contraindication.
- **Other antihypertensive medications** (eg,α-blockers, β-blockers, combination α-and β-blockers, centrally acting agents, potassium-sparing diuretics, and direct vasodilators) should be reserved for children who are not responsive to ≥2 of the preferred agents .

TREATMENT: *Follow-Up and Monitoring*

- Treatment of a child or adolescent with HTN requires ongoing monitoring because **goal BP can be difficult to achieve**.
- **If the decision has been made to initiate treatment with medication**, the patient should be seen frequently (**every 4–6 weeks**) for dose adjustments and/or addition of a second or third agent until goal BP has been achieved. After that, the frequency of visits can be extended to **every 3 to 4 months**.
- **If the decision has been made to proceed with lifestyle changes only,** then follow-up visits can occur at longer intervals (**every 3–6 months**) so that adherence to lifestyle change can be reinforced and the need for initiation of medication can be reassessed.
- At each follow-up visit, the patient should be assessed for **adherence** and for any **adverse effects** of the prescribed medication; such assessment may **include laboratory testing** depending on the medication (for example, electrolyte monitoring if the patient is on a diuretic). It is also important to **continually reinforce adherence to lifestyle changes** because effective treatment will depend on the combination of effects from both medication and lifestyle measures.
- Finally, known hypertensive **target organ damage (such as LVH) should be reassessed** according to the recommendations

Treatment-Resistant HTN

- **Resistant HTN** in adults is defined as persistently elevated BP **despite treatment with 3 or more antihypertensive agents of different classes**. All of these drugs should be prescribed **at maximally effective doses**, and **at least 1 should be a diuretic**.
- **Key to the identification** of patients with true resistant HTN is correct office BP measurement, confirmation of adherence to current therapy, and confirmation of treatment resistance by ABPM.
- The **treatment** f patients with resistant HTN includes dietary sodium restriction, the elimination of substances known to elevate BP, the identification of previously undiagnosed secondary causes of HTN, the optimization of current therapy, and the addition of additional agents as needed.
- Recent clinical trial data suggest that an **aldosterone receptor antagonist** (such as spironolactone) is the optimal additional agent in adults with resistant HTN; it helps address volume excess as well as untreated hyperaldosteronism, which is common in adult patients with true resistant HTN.
- **At present, there are no data on if true treatment-resistant HTN exists in pediatric patients.**

Treatment in Patients With CKD

- **Children and adolescents with CKD often present with or develop HTN.**
- **HTN is a known risk factor for the progression of kidney disease** in adults and children. Evidence suggests that the treatment of HTN in children with CKD might slow the progression of or reverse end organ damage.
- When evaluated by **24-hour ABPM**, children and adolescents with CKD often have poor BP control even if BP measured in the clinic appears to be normal.
- **MH** is associated with **end organ damage**, such as LVH.
- Threshold values that define HTN are not different in children with CKD, although there is some evidence that **lower treatment goals might improve outcomes**.

Treatment in Patients With CKD: *Key Action*

- Children & adolescents with **CKD should be evaluated for HTN at each medical encounter.**
- Children or adolescents with both CKD and HTN should be treated to lower 24-hour MAP **to <50th percentile by ABPM**.
- Regardless of apparent control of BP with office measures, children and adolescents with CKD and a history of HTN should have BP assessed by **ABPM at least yearly to screen for MH**

Treatment in Patients With Proteinuria

- **Proteinuric renal disease** is often associated with **HTN** and a **rapid decline in glomerular filtration**.
- Studies in both adults and children have indicated that **both BP control and a reduction in proteinuria are beneficial for preserving renal function**.
- **RAAS blockade therapy** in patients with CKD &HTN has been shown to benefit both BP & proteinuria. The benefit of such therapy may not be sustained, however.

Key Action

- Children and adolescents with **CKD & HTN should be evaluated for proteinuria** .

Key Action

- Children and adolescents with **CKD, HTN, & proteinuria** should be treated with: an **ACE inhibitor or ARB.**

Treatment in Patients With Diabetes

Key Action

- Children and adolescents with **T1DM or T2DM** should be **evaluated for HTN at each medical encounter**
- **…** and **treated** if BP is **≥95th percentile or >130/80** mm Hg in adolescents ≥13 years of age

Comorbidities

- ***Dyslipidemia***

  Children and adolescents with **HTN** are at **increased risk for lipid disorders** attributable to the "common soil" phenomenon, in which **poor diet, inactivity, and obesity** contribute to both disorders. HTN and dyslipidemias are associated with **subclinical atherosclerosis** and are **risk factors for future CVD.**

- ***OSAS***

  Children with moderate to severe OSAS are at increased risk for HTN.
  However, it is not known whether OSAS treatment with **continuous positive airway** pressure results in improved BP in all children. Furthermore, **adenotonsillectomy** may not result in BP improvement in all children with OSAS.

- **Cognitive Impairment**

  The **central nervous system is a target organ** that can be affected by HTN.

Acute Severe HTN

- **There is a lack of robust evidence** to guide the evaluation and management of children and adolescents with acute presentations of severe HTN. Thus, much of what is known is derived from studies conducted in adults, including medication choice.

- Although children and adolescents can become symptomatic from HTN at lesser degrees of BP elevation, in general, patients who present with acute severe HTN will have BP elevation well **above the stage 2 HTN threshold**.

- The major clinical issue in such children is that this level of BP elevation may produce **acute target organ effects, including encephalopathy, acute kidney injury, & congestive heart failure**. Clinicians should be concerned about these complications when a child's **BP increases 30 mm Hg or more above the 95th percentile**.

- Although a few children with primary HTN may present with features of acute severe HTN, **the vast majority will have an underlying secondary cause of HTN.** Thus, for patients who present with acute severe HTN, an evaluation for secondary causes is appropriate & should be conducted expediently.

Acute Severe HTN

- Given the potential for the development of **potentially lifethreatening complications**, children & adolescents who present with acute severe HTN **require immediate treatment with short-acting antihypertensive medications** that may abort such sequelae.
- Treatment may be initiated with **oral agents** if the patient is able to tolerate oral therapy and **if life-threatening complications have not yet developed**.
- **Intravenous agents** are indicated when oral therapy is not possible because of the patient's clinical status or when a severe complication has developed (such as congestive heart failure) that warrants a more controlled BP reduction.
- **The BP should be reduced by no more than 25% of the planned reduction over the first 8 hours, with the remainder of the planned reduction over the next 12 to 24 hours.**
- **The ultimate short-term BP goal in such patients should be around the 95th percentile.**

Oral & IV Antihypertensive Medications for Acute Severe HTN

Useful for Severely Hypertensive Patients With Life-Threatening Symptoms

Drug	Class	Dose	Route	Comments
Esmolol	β-adrenergic blocker	100–500 μg/kg per min	Intravenous infusion	Short acting, constant infusion preferred. May cause profound bradycardia
Hydralazine	Direct vasodilator	0.1–0.2 mg/kg per dose up to 0.4 mg/kg per dose	Intravenous, intramuscular	Causes tachycardia Give every 4 h when given intravenous bolus
Labetalol	α- and β-adrenergic blocker	Bolus: 0.20–1.0 mg/kg per dose up to 40 mg per dose Infusion: 0.25–3.0 mg/kg per h	Intravenous bolus or infusion	Asthma and overt heart failure are relative contraindications
Nicardipine	Calcium channel blocker	Bolus: 30 μg/kg up to 2 mg per dose Infusion: 0.5–4 μg/kg per min	Intravenous bolus or infusion	May cause reflex tachycardia. Increases cyclosporine and tacrolimus levels
Sodium nitroprusside	Direct vasodilator	Starting: 0–3 μg/kg per min Maximum: 10 μg/kg per min	Intravenous infusion	Monitor cyanide levels with prolonged (>72 h) use or in renal failure; or coadminister with sodium thiosulfate

Useful for Severely Hypertensive Patients With Less Significant Symptoms

Clonidine	Central α-agonist	2–5 Mg/kg per dose up to 10 Mg/kg per dose given every 6–8 h	Oral	Adverse effects include dry mouth and drowsiness
Fenoldopam	Dopamine receptor agonist	0.2–0.5 Mg/kg per min up to 0.8 Mg/kg per min	Intravenous infusion	Higher doses worsen tachycardia without further reducing BP
Hydralazine	Direct vasodilator	0.25 mg/kg per dose up to 25 mg per dose given every 6–8 h	Oral	Half-life varies with genetically determined acetylation rates
Isradipine	Calcium channel blocker	0.05–0.1 mg/kg per dose up to 5 mg per dose given every 6–8 h	Oral	Exaggerated decrease in BP can be seen in patients receiving azole antifungal agents
Minoxidil	Direct vasodilator	0.1–0.2 mg/kg per dose up to 10 mg per dose given Q 8–12 h	Oral	Most potent oral vasodilator, long acting

HTN and the Athlete

Sports participation & increased physical activity should be encouraged in children with HTN

- **In adults, physical fitness is associated with lower all-cause mortality.**
- **On the basis of adult evidence, sports participation should improve BP over time**. Additionally, **exercise itself has a beneficial effect on cardiac structure in adolescents**.
- The **athlete** interested in participating in **competitive sports and / or intense training** presents a special circumstance.

  Recommendations from AHA and ACC include:

  1. limiting competitive athletic participation among athletes with **LVH** beyond that seen with athlete's heart **until BP is normalized** by antihypertensive drug therapy.
  2. r**estricting** athletes with **stage 2 HTN** from participating in **high-static sports** (eg, weight lifting, boxing, and wrestling) **until HTN is controlled**.
- There are **no data linking** the presence of **HTN to sudden death related to sports** participation in children, although many cases of sudden death are of unknown etiology.

HTN and the Posttransplant Patient

HTN is common in children after solid-organ transplants : 50% to 90%

- **Contributing factors** include the use of steroids, calcineurin inhibitors, & mTOR inhibitors.
- In patients with **renal transplants**, the presence of **native kidneys, CKD, and transplant glomerulopathy are additional risk factors for HTN**.
  - HTN rates are higher by 24-hour **ABPM** compared with clinic BP measurements because these populations commonly have **MH & nocturnal HTN**.
  - The **management** of identified HTN in the pediatric transplant patient can be **challenging**. **Rates of control of HTN in renal-transplant patients** generally range from **33% to 55%.**
  - **Limited evidence that ACE inhibitors and ARBs may be superior** in achieving BP control and improving long-term graft survival in renal-transplant patients.
  - The **combination of ACE inhibitors and ARBs in renal-transplant patients has been associated with acidosis & hyperkalemia → not recommended.**

Lifetime HTN Treatment & Transition to Adulthood

Key Action

- Adolescents with elevated BP or HTN should have:
  - **care transitioned to an adult care provider by 22 years of age**
- **Transfer of information** of the patient**:**
  - HTN **etiology**
  - past **manifestations**
  - **complications**

Prevention of HTN : Importance

- **Elevated BP measurements in childhood conferrs an increased risk of adult HTN.**
- **↑BMI** is a major determinant of the development of HTN.
- **In both children and adults, efforts should be made to prevent progression to sustained HTN and to avoid the development of hypertensive CV diseases**.

Prevention of HTN : Strategies

- Prevention efforts to date have focused on **lifestyle modification, especially dietary intervention, exercise, and treatment of obesity.**
- **Appropriate energy balance :** calories eaten balanced by calories expended in physical activity → the best strategy **to maintain an appropriate BMI & avoid obesity.**
- 60 min/d ay of moderate to vigorous **physical activity**
- **Normal sleep habits & avoidance of tobacco** are also strategies to reduce CV risk.

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