

Review Article

Age and Hypertension: Special Considerations in Children and Adolescents

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Funding statement: No funding.

Conflicts of interest: None

Author contributions: All authors contributed a segment/topic of the manuscript. Khumalo is the first author, and Petersen is the senior author.

Abstract

Hypertension (HTN) is an increasingly recognized public health concern in children and adolescents, with the rising prevalence being driven by obesity and lifestyle factors. While primary HTN is becoming more common, secondary causes remain important and potentially reversible. Early diagnosis, through routine office blood pressure measurements and clinical evaluation, is critical in preventing target organ damage. Management includes lifestyle modifications, with pharmacological therapy necessary in some cases. This review highlights the diagnosis, investigation, and management of paediatric HTN, with an emphasis on several high-risk groups relevant to South Africa.

INTRODUCTION

Hypertension (HTN) in children and adolescents is often diagnosed late after the onset of target organ damage. In contrast to adult-onset HTN, secondary causes of HTN remain more common in children. Early identification will prevent long-term target organ damage.(1–3)

This review aims to highlight the importance of routine blood pressure (BP) measurement in paediatric practice and provides a nuanced guide to investigations, management, and referral of children with HTN, with an emphasis on special populations relevant to South Africa (SA).

DEFINITIONS AND CLASSIFICATION

Paediatric BP measurement and interpretation use age-, sex-, and height-percentile ranges to classify paediatric HTN. A BP reading consistently above the 95th percentile

(on three or more occasions) is defined as HTN, see Table 1.(4) For children 1- ≤13 years of age, the American Academy of Paediatrics (AAP) 2017 clinical practice guidelines is one of the most widely used; the other is the 2016 European Society of Hypertension (ESH).(1,2) The percentiles for AAP are preferred since obese children were excluded in generating the normative tables, though there has been no difference noted in the comparison of outcomes.(1,5) For neonates, postmenstrual age is used, with classification being the same as young children.(6,7)

Mobile phone-based applications offer a less cumbersome means to obtain BP centiles than plotting on charts. Useful applications include MDCalc (developed in partnership with the AAP) and Anthro Calc 3.0.2 (which utilises the AAP 2017 as a reference). Other applications include Pediatric Blood Pressure Guide and Pediatric Blood Pressure AAP (PAPed).

Table 1: Classification of Paediatric Hypertension

	0 to 1 year*	1 to < 13 years**	≥ 13 years**
Normal BP	< 90 th percentile	< 90 th percentile	< 120/<80 mmHg
Elevated BP	90 th - < 95 th percentile	≥ 90 th - < 95 th percentile	120 - 129/<80mmHg
Stage 1 Hypertension	≥ 95 th percentile	≥ 95 th - < 95 th + 12mmHg	130-139/80-89mmHg
Stage 2 Hypertension	≥ 99 th percentile	≥ 95 th percentile + 12mmHg	≥ 140/90 mmHg
White coat hypertension**	BP ≥ 95 th percentile in the clinical setting but <95 th percentile outside the clinical setting		
Masked hypertension**	Normal BP in the clinical setting but elevated BP during ambulatory BP monitoring		
Ambulatory hypertension**	Systolic and/or diastolic BP load of ≥25% above 95 th percentile Nocturnal dipping below 10%		

* Infant blood pressure definitions based on "Report of the Second Task Force on Blood Pressure Control in Children-1987" (6)

** AAP 2017 clinical practice guidelines (1)

BLOOD PRESSURE MEASUREMENT

Most guidelines recommend that BP be measured at least annually in children ≥3 years of age and more often in those with risk factors.(1,2,4) Measuring BP in children requires patience and skill to obtain an accurate reading. In paediatric patients, it is important to ensure that the patient is

comfortable with the healthcare worker, has not had any stimulant for at least 30 minutes, has rested for 5 minutes, and is not crying. (Box 1)

As paediatric BP readings vary, at least three measurements are necessary if the first reading is above the 95th centile. Transient elevated BP may occur with acute

Box1. Best practice for BP measurement in paediatric patients (1,2,4)

Technique	Notes
Oscillometric automated device	Useful for screening BP and 4-limb BP Measures MAP; calculates SBP and DBP algorithm Overestimates BP Needs to be validated for use in paediatrics Should be calibrated regularly Inaccurate in cases of wide pulse pressure
Cuff size	Bladder length >80% MUAC Bladder width >40% MUAC The mid-upper arm circumference (MUAC) is measured at the midpoint between the acromion and olecranon, with the shoulder in a neutral position and the elbow flexed to 90°
Patient position	Quiet setting with child rested for 3-5min Back supported, feet on the ground and right arm at level of heart (supine if unable or in the arms of caregiver) Measurement in right arm if possible BP is usually 10- 20% higher in the legs due to increased peripheral resistance
Aneroid/ manual device	Should be used for confirmation of elevated BP or to measure pulsus paradoxus and pulse pressure. First Korotkoff sound = SBP Fifth Korotkoff sound = DBP (Fourth if fifth absent) Auscultation over the brachial artery, 2-3cm above the antecubital fossa Three readings 1-2 minutes apart (use mean of the last 2)
Ambulatory BP monitor (ABPM)	24- hour reading Diagnose white coat HTN/ masked HTN/ abnormal diurnal BP rhythm

SBP: Systolic blood pressure, DBP: diastolic blood pressure

illness, fear, medication use, or sympathetic stimulation.⁽¹⁾ Diagnosis should therefore be based on repeated measurements in a stable, rested child.

Office/outpatient BP measurement

Screening of HTN may be performed with an automated oscillometric machine (AOM). If the BP is > 90th percentile, confirmation with an aneroid sphygmomanometer is essential, as it provides the most accurate BP measurement. (7) In neonates and infants, dopplers may be used to measure systolic BP. Home BP machines should not be used for diagnosis or dose adjustments, as most have not been validated for paediatric patients. There is limited validated data on wrist and finger BP measurements in children.

Healthcare establishments providing paediatric care, including paediatric rooms, should have a wide range of cuff sizes. Paediatric-validated AOMs or calibrated aneroid sphygmomanometers should be sizes available.

Ambulatory BP measurement

The use of ambulatory BP monitoring (ABPM) helps confirm HTN and diagnose white-coat hypertension and masked HTN. It is also recommended in patients with the following risk factors for HTN: obesity, advanced chronic kidney disease (CKD), premature infants, cardiac disease, and diabetes mellitus (T1DM).^(1,2,8) Correlations between ambulatory HTN and adverse cardiac outcomes have been established in adults, but research is still needed to confirm the same in children. Although ABPM is available in some tertiary centres in SA, it is limited by paediatric-appropriate cuff sizes as supplied by the manufacturer.

AETIOLOGY

The cause of primary HTN is multifactorial. There is a strong genetic link, with heritability being 30-50%. Perinatal risk factors are those that impair nephrogenesis, putting low birth weight and preterm children at higher risk. Primary HTN is more prevalent in those with a low socioeconomic status and food insecurity.^(9,10) Other factors such as ethnicity, psychological stress, tobacco smoking, and rapid urbanisation have also been implicated.⁽⁹⁾

Obesity is a major risk factor, with overweight and obese children having an increased risk of HTN and becoming hypertensive at a younger age. Physical activity is inversely related to obesity and counteracts these mechanisms. Dietary sodium is closely related to BP, and those on a high-salt diet, as well as those with salt sensitivity, are at risk. Children and adolescents with HTN are more likely to have HTN as adults. Salt reduction, increased activity, improved socioeconomic status, and weight loss improve this.⁽⁴⁾

Secondary causes of HTN are more common in the paediatric population and should be excluded based on age, risk factors, and the patient's clinical presentation. (Table 2)

ASSESSMENT AND SPECIAL INVESTIGATION

A complete history is essential to identify risk factors for HPT. Examination of the patient will also guide the investigation. The image below is an algorithm for the stepwise investigation of hypertension. (Figure 1)

This algorithm outlines a structured approach to evaluating children with suspected or confirmed hypertension. Following confirmation of elevated blood pressure, patients undergo clinical assessment and initial screening investigations. Features suggestive of secondary hypertension guide targeted investigations, while children without such features are managed as primary hypertension. All patients should be assessed for target organ damage and managed with lifestyle modification and pharmacological therapy where indicated. Specialist referral is recommended for those with severe, resistant, or secondary hypertension.^(1,2,4)

TARGET ORGAN DAMAGE

Target organ damage (TOD) may occur early in paediatric HTN, even in those with mildly elevated BPs.^(1,2,10) Although few longitudinal studies in children exist, observational studies provide evidence of damage to the heart, kidneys, brain, retina, and vasculature.⁽⁴⁾

Left ventricular hypertrophy is the most common cardiac manifestation in children.^(2,4,10) It reflects chronic pressure overload and is associated with adverse cardiac outcomes. Echocardiography is the preferred mode of assessment for those with sustained or symptomatic hypertension.^(1,2) Early identification is important as recovery is possible in those who achieve BP targets, highlighting the importance of effective BP control.⁽¹⁾ Paediatric HTN is linked to cardiovascular disease, and target organ damage starts early in hypertension.⁽¹¹⁻¹³⁾ The link is seen even with elevated BP, masked HTN, and WCH.⁽⁴⁾

Kidney disease both contributes to and results from HTN, needing stricter control in these patients. Screening for proteinuria and kidney function is essential in all newly diagnosed hypertensive patients.^(2,4) A guide to investigation is in Table 3.

SPECIAL POPULATIONS

This section explores HTN within selected paediatric populations, focusing on management considerations relevant to SA.

Chronic kidney disease

Children with chronic kidney disease (CKD) are at increased risk of early TOD damage. The targeted BP is lower (50th percentile) for patients with CKD.⁽⁸⁾ There are multiple factors associated with HPT in patients with CKD. Uraemia and kidney failure can cause increased cardiac output and/or total peripheral resistance and thus lead to hypertension. The renin-angiotensin-aldosterone system (RAAS) is a known cause of HTN in CKD and in

Table 2: Causes of secondary hypertension

Cause of hypertension	Specific diagnoses and associated conditions
Kidney disease	Chronic kidney disease Nephritis e.g. lupus nephritis, IgA vasculitis with nephritis, acute postinfectious glomerulonephritis Autosomal recessive polycystic kidney disease (ARPKD) Autosomal dominant polycystic kidney disease (ADPKD) Haemolytic uraemic syndrome and other causes of acute kidney injury Congenital abnormality of the kidney and urinary tract (CAKUT) Kidney scarring post pyelonephritis
Renovascular disease	Renal artery stenosis Fibromuscular dysplasia Neurofibromatosis Type1 Williams syndrome Arteritis (Takayasu, Kawasaki, Moyamoya) Renal arterial or venous thrombosis
Cardiovascular	Coarctation of the aorta
Neurological causes	Increased intracranial pressure Spinal injury Guillain-Barre syndrome
Tumour	Phaeochromocytoma Wilms tumour Neuroblastoma
Endocrine	Diabetes mellitus Congenital adrenal hyperplasia Thyroid disorders
Other	Obstructive sleep apnoea
Drug mediated	Glucocorticoids Calcineurin inhibitors Oral contraceptives Erythropoietin Non-steroidal anti-inflammatory drugs (NSAID) Caffeine, tobacco, ethanol Cocaine, amphetamines

end-stage kidney disease (ESKD).(14) Other mechanisms include sodium and water retention leading to volume expansion, overactivity of the sympathetic nervous system, endothelial cell dysfunction, arterial stiffness, and iatrogenic causes from medications such as erythropoietin.(14) Additionally, children with certain underlying diseases, e.g., glomerulopathies and polycystic kidney disease, are especially prone to hypertension.(15)

Children with CKD and uncontrolled HTN have been shown to progress to ESKD faster than normotensive patients or those whose BPs were kept below the 50th percentile.(3,16) Intensified BP control with diuretics should be cautioned in patients at risk of dehydration and acute kidney injury (AKI).(8) Higher levels of proteinuria are most

strongly associated with poor BP control. Angiotensin-converting enzyme inhibitors (ACEIs) or Angiotensin receptor blockers (ARBs) are recommended as first-line treatment for HTN in children with proteinuric CKD.(8)

Neonatal period

Neonatal HTN is an increasingly recognised condition. This reflects national trends reporting improved detection and monitoring of neonatal complications as part of quality-of-care initiatives.(17–19)

In SA, neonatal HTN is common in preterm infants with significant comorbidities, including pulmonary dysplasia, acute kidney injury, sepsis, or prolonged exposure to invasive

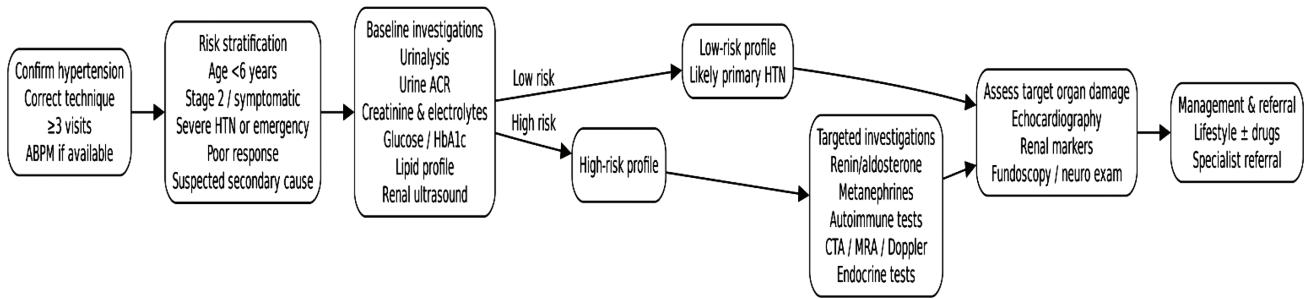


Figure 1: Stepwise approach to the investigation and management of paediatric hypertension

Table 3: Target organ damage and recommended screening

	Findings	Investigation	Who to screen	Frequency
Heart	Left ventricular hypertrophy, diastolic dysfunction	Echocardiography Chest x-ray ECG	Sustained & stage 2 HTN, TOD symptoms	At diagnosis; annually or as required
Kidneys	Proteinuria, reduced eGFR	Urine ACR serum creatinine	All children with HTN	At diagnosis; annually unless abnormal
Vasculature	Increased arterial stiffness, carotid IMT	Carotid ultrasound (specialist centres)	Selected cases / research settings	Not routine
Brain	Encephalopathy, seizures, cognitive impact	Clinical assessment ± neuroimaging	HTN emergency or neurological symptoms	Symptom driven
Eyes	Hypertensive retinopathy	Fundoscopy	Severe or long-standing HTN	At diagnosis
Other cardiovascular risk	Obesity, dyslipidaemia, insulin resistance	BMI, lipid profile, glucose	Primary HTN, obese children	At diagnosis; follow-up per risk

HTN: Hypertension, TOD: Target organ damage, eGFR: estimated glomerular filtration rate
IMT: intima-media thickness, ACR: urinary albumin to creatinine ratio, BMI: body mass index

ventilation and vasoactive medications.(20–23) Renal and renovascular causes predominate e.g. renal parenchymal disease, renal vein thrombosis associated with umbilical vessels catheterisation, and congenital anomalies of the kidney and urinary tract (CAKUT).(17–19) Cardiovascular causes, particularly coarctation of the aorta, should be excluded, in keeping with national guidance that prioritises identification of surgically correctable pathology.(22)

Hypertension is often detected during routine vital signs evaluation in neonatal intensive care settings.(20) Severe or sustained HTN may present with cardiac failure, seizures, feeding difficulties, or poor weight gain.(17–19) In the South African context, access to advanced imaging, antihypertensive agents, and sub-specialist follow-up may be variable, and delayed recognition may increase the risk of end-organ damage.(20–22)

Management involves confirmation of true blood pressure elevations, targeted investigations guided by clinical findings, and treatment of reversible causes, with antihypertensive therapy reserved for moderate-to-severe cases or those with evidence of target-organ involvement.(20,22) While many cases resolve during infancy, structured follow-up is recommended for both preterm and term infants.(23,24)

Endocrine

Endocrine causes account for 0.05–6% of all cases of HTN in children.(1,2) Identifying an endocrine cause allows for specific interventions, such as surgical resection or hormone-directed therapy, which can significantly improve HTN outcomes by decreasing TOD.(1)

Primary hyperaldosteronism (Conn's syndrome) occurs when there is excess aldosterone leading to sodium retention, hypokalaemia, and hypertension.(25) Causes include bilateral adrenal hyperplasia, aldosterone-producing adenomas, or rarely, carcinomas or genetic mutations.(25) Similarly, in congenital adrenal hyperplasia due to 11 β -hydroxylase and 17 α -hydroxylase deficiencies, there is elevated BP.(26) Although aldosterone is low, there is an excess of deoxycorticosterone, which exerts mineralocorticoid effects.(26)

Catecholamine-secreting tumours such as pheochromocytomas and paragangliomas should be considered in children presenting with paroxysmal hypertension, headaches, palpitations, and diaphoresis.(27) For these patients, treatment should be initiated with alpha blockers. Thyroid disorders can lead to hypertension.(28) Hyperthyroidism increases systolic BP via enhanced cardiac output, while

hypothyroidism elevates diastolic pressure through increased systemic vascular resistance.(28) Cushing syndrome with increased cortisol promotes HTN through multiple mechanisms.(29) These include increased vascular sensitivity to catecholamines and activation of mineralocorticoid receptors.(29) Other rarer causes include hyperparathyroidism, growth hormone excess, and genetic conditions such as apparent mineralocorticoid excess and primary glucocorticoid resistance.(25,30,31)

Diabetic nephropathy in adolescents is associated with high nocturnal BP, particularly diastolic, due to most albuminuria occurring at night.(32,33) There may also be no nocturnal dipping, which in adults is associated with poor cardiac outcomes.(33) This will not be evident during office BP measurement, and it is important to screen this cohort with ABPM annually if available.(1)

The investigation and management of paediatric hypertension due to endocrine disease requires specialist guidance. These patients should be managed in conjunction with paediatric endocrinology units.

Takayasu arteritis

Takayasu arteritis (TA) is a large-vessel vasculitis primarily affecting the aorta and its branches. Although incidence rates for Takayasu arteritis have been documented in many parts of the world, data from Africa remain limited.(34) Takayasu arteritis is a significant cause of renovascular HTN in SA.(35) Numerous case series describing the epidemiology, clinical types, and treatment outcomes in children are reported from SA.(36–45) Globally, the association with *Mycobacterium tuberculosis* is controversial (MTb).(46,47) Although a causal link has not been established, the prevalence of MTb in TA is high in reports from SA. The clinical diagnosis is challenging and often delayed. In the pre-pulseless phase, systemic symptoms of fever, weight loss, and fatigue predominate. Once the disease progresses to the pulseless phase, symptoms are protean and depend on the extent of vessels involved.(48) The classification of TA in children is based on the EULAR/ PRINTO/ PRES criteria.(49) (Table 4)

The recommended imaging to determine disease extent is magnetic resonance imaging (MRI), computed tomography (CT), or fluorodeoxyglucose positron emission tomography (FDG-PET), depending on the availability of the modalities.(50) A Systematic review including 21 studies determined the sensitivity (77%) and specificity (71%) of FDG PET for treatment monitoring in large vessel vasculitis.(51) Biomarkers of active inflammation, such as CRP and ESR, demonstrate low sensitivity and specificity, and only moderately reflect activity on FDG PET. Although various classifications of anatomical types are available, the preferred classification, as modified by Pantanowitz et al. (Table 5), is practical for assessing kidney auto-transplantation where indicated.(52)

The treatment of TA requires multidisciplinary specialised teams. The cornerstone of therapy is immunotherapy and BP control (avoiding ACE inhibitors). Invasive procedures should be delayed until the disease is in remission to avoid catastrophic vessel rupture when inflammation is present. Percutaneous angioplasty is preferred where possible. Given the burden of MTb in SA coupled with TA immunosuppressive therapy, it is prudent to consider MTb treatment in paediatric cases of TA.

Acute post-infectious glomerulonephritis

Acute post-infectious glomerulonephritis (PIGN) is an immune-mediated inflammatory disease of the glomeruli that occurs following infections and remains an important cause of hypertension and acute kidney injury (AKI) in children, particularly in developing countries. The disease results from immune complex deposition in the glomeruli with activation of the complement system, leading to glomerular inflammation and impaired renal function. Post-streptococcal glomerulonephritis, caused by infection with Group A β -haemolytic streptococcus, is the most common form of PIGN and typically develops 1–4 weeks after a throat or skin infection.(53–55) Clinically, the condition typically presents with cola-coloured urine, peripheral oedema, and hypertension. Evidence of recent streptococcal infection

Table 4: EULAR/PRINTO/PRES criteria and classification definition of childhood TA

Mandatory criterion plus one of the five listed

Angiography (mandatory)

CT/ MRI aorta and pulmonary arteries showing aneurysmal dilatation, narrowing, occlusion or thickened arterial wall.

1. Pulse deficit or claudication
2. BP discrepancy (4 limb reading)
3. Bruits or thrills over large vessels
4. Hypertension
5. Elevated CRP or ESR

EULAR: European League Against Rheumatism; PRES: Paediatric Rheumatology European Society; PRINTO: Paediatric Rheumatology International Trials Organisation, CT: computed tomography, MR: magnetic resonance imaging, CRP: C-reactive protein, ESR: erythrocyte sedimentation rate

Table 5: Anatomical classification of Takayasu Arteritis

Anatomical type	Vessel involved
I	Ascending aorta and branches
II	Descending aorta and branches
III	Ascending and descending aorta
IV	Pulmonary arteries
V	Iliac arteries

is commonly supported by elevated anti-streptolysin O titres (ASOT) and/or anti-DNase B titres, with the latter being more sensitive for skin infections.(53–56)

Hypertension is a common manifestation of PIGN, occurring in approximately 60–80% of affected children.(54,57,58) It typically develops during the acute phase of the disease and is primarily associated with sodium and water retention secondary to reduced glomerular filtration, resulting in intravascular volume expansion and hypervolemia. Activation of the renin–angiotensin–aldosterone system and increased sympathetic activity may also contribute to elevated BP.(54)

Clinically, HTN ranges from mild elevation of BP to severe HTN or hypertensive emergency. Severe HTN may lead to complications such as hypertensive encephalopathy, seizures, pulmonary oedema, and congestive cardiac failure.(53,54,59) Management focuses on controlling volume overload through salt restriction, fluid restriction, and diuretics, particularly loop diuretics such as furosemide. If HTN persists, calcium channel blockers such as amlodipine may be used, while ACE-Is and ARBs are generally avoided in the acute phase.(53,54) Hypertension is usually transient and resolves as renal function improves, although follow-up is recommended to monitor for persistent HTN or renal complications. Microscopic haematuria may persist after resolution of AKI and HTN and is not an indication of ongoing disease.(53–55)

Adolescent hypertension

The prevalence of HTN in adolescents is increasing, peaking during the adolescent growth spurt.(10–12) The increase in prevalence is attributed to the increase in rates of obesity and poor lifestyle choices.(10,60) Identified risk factors in adolescence include sedentary lifestyle, reduced physical activity, psychological stress, increased consumption of salty foods and use of electronic devices, poor sleep quality and obstructive sleep apnoea.(12,60,61) In addition to factors leading to HTN in younger children, adolescents have added risks contributing to HTN, including the use of stimulants, caffeine and energy drinks consumption, alcohol use, smoking, use of contraceptives, and teenage pregnancy-related HTN.(12,60,61) Hypertension in adolescence is strongly associated with adulthood HTN, and the tracking into adulthood is stronger in obese teenagers.

(11) Children with persistent HTN have a 7.6 times higher hazard ratio of having hypertension as adults.(10)

Adolescent HTN is associated with the development of ESKF in adulthood. Hypertension in adolescents is also linked to poor neurocognitive function, learning disabilities, and worse executive function.(62,63) Controlling HTN is associated with reductions in LVH, improvements in cognitive function, and delayed progression of CKD.(10,16,63) It is therefore important to prevent, diagnose, and control HTN in adolescents to reduce the risk of adult-onset target organ disease.(13)

The adolescent period and transfer to adult care is a difficult period as young adults assert their autonomy. It is a period associated with non-adherence to medical advice.(64) Adolescents face many challenges, including rapid development, sexual maturation, use of alcohol and smoking, and establishing their independence.(65) To ensure continued BP control, transition into adult care should be approached with care.

Guidelines on transitioning to adult care increase the chances of a successful transition.(66–69) In SA, a dedicated kidney transfer clinic was associated with improved outcomes, reduced mortality, and higher follow-up rates.(70) Transition should aim to empower adolescents in self-management skills, to take responsibility for their health, to engage with the adult team, and ultimately to adhere to treatment recommendations.

MANAGEMENT

Lifestyle and non-pharmacological interventions should be included in all treatment plans. A healthy diet, increased physical activity, and stress management are three areas where patients should receive counselling.(1,2) The Dietary Approach to Stop Hypertension (DASH) recommends increasing fruits, vegetables, legumes, low-fat milk products, whole grains, fish, poultry, nuts, and lean red meats in the diet, limiting sugars and lowering salt intake.(1,2,71) A dietician may assist in drawing up a meal plan. Moderate-to-vigorous physical activity for at least 30 to 60 minutes daily is recommended.(1,2,4) This can be achieved by walking and participation in school sports. In adolescence, stress and anxiety reduction has been seen with the use of breath awareness, meditation, mindfulness, and yoga practice, although the quality and consistency of this evidence vary.(4)

Pharmacological treatment

Treatment is indicated if HTN persists despite lifestyle intervention, those with symptomatic HTN, stage 2 HTN without modifiable factors, or HTN associated with CKD or DM. Pharmacological treatment is individualised to the underlying cause in secondary hypertension. The safest initial agent, in the absence of an ultrasound to exclude possible renal artery stenosis, is a long-acting calcium channel blocker (CCB). Angiotensin-converting enzyme inhibitors

Table 6: Common first-line drugs [2]

Antihypertensive Class	Recommended disease	Contraindication	Drug	Initial Dose	Maximal Dose	Dosing interval
Diuretics						
Potassium sparing	Hyperaldosteronism	CKD (caution), Competitive athletes,	Spironolactone	1mg/kg	3.3mg/kg up to 100mg	Daily – twice daily
Thiazide	CKD, Corticosteroid-induced HTN	Competitive athletes, DM	Hydrochlorothiazide	0.5-1mg/kg	3mg/kg/day	Daily
Loop diuretic	CCF	Dehydration Acute gastro-enteritis	Furosemide	0.5–2mg/kg	6mg/kg	Daily– twice daily
Calcium Channel Blockers	Post-transplant, Migraine, Coarctation of Aorta	CCF	Amlodipine	0.06-0.3mg/kg	5–10mg	Daily
ACE inhibitors	CKD, DM, CCF, Microalbuminuria, Obesity-linked primary HTN	RAS, Hyperkalaemia, Pregnancy	Enalapril Captopril	0.08–0.6mg/kg 0.3-0.5mg/kg/dose	6mg/kg	Daily Twice– thrice daily
ARBs	CKD, DM, CCF, Microalbuminuria, Obesity-linked primary HTN	RAS, Hyperkalaemia, Pregnancy	Losartan	0.7mg/kg up to 50mg	1.4mg/kg up to 100mg	Daily– twice daily

Key: DM -Diabetes Mellitus, CKD- Chronic Kidney Disease, RAS- Renal artery stenosis, CCF- Congestive Cardiac Failure

(ACE-I), Angiotensin II receptor blockers (ARBs), or thiazide diuretics are recommended unless contraindicated. The combination of ACE-I and ARB in paediatric patients is not recommended. Use a single agent, titrated 2-4 times weekly, until the maximum dose is reached before adding another agent. After using 2 or more of these agents, other agents are added, preferably under specialist advice. (Table 6)

Emergency hypertension

A hypertensive crisis is defined as an acute, severe elevation in BP (above stage 2).(72) This can be further classified as a hypertensive emergency or urgency depending on the degree of BP increase and the presence of acute TOD. (72,73) Absolute thresholds in terms of cut-offs for defining hypertensive crises in paediatrics differ. Regardless, a hypertensive emergency is a life-threatening condition associated with severe BP elevation and organ damage, such as neurological, cardiac, or renal dysfunction. In children, these can present as headache, confusion, seizures, nausea and vomiting, or visual symptoms. Symptoms can be very non-specific and easy to miss if BP is not checked routinely. A hypertensive urgency is sudden, severe HTN without organ damage, and is not a life-threatening condition

Management of hypertensive crisis- goals and principles

1. Treat reversible causes – e.g., intravenous (IV) furosemide for fluid overload, hypertonic saline for

raised intracranial pressure, and appropriate seizure management.

2. Reverse and avoid further HTN-induced organ damage and avoid secondary ischaemia-induced end-organ damage caused by rapid lowering of BP.
3. Reduce BP by no more than 25% within 8 hours, then gradually reduce to < 90th percentile over the next 48-72 hours.

There is no robust evidence to guide management. When possible and available, children should be managed in a paediatric intensive care unit with invasive BP monitoring. There is also no clear consensus on IV versus oral medication, or which drug is the most effective and safest as first-line therapy.(73,74)

Commonly used intravenous drugs include Labetalol, Clevidipine, Esmolol, Nicardipine, and Hydralazine. Common oral agents are Nifedipine and Amlodipine. Of importance is knowing what is available in the centre in which you work and in which situations these drugs are contraindicated.

CONCLUSION

Hypertension in children is prevalent across all age groups. It is an early marker of future cardiovascular, renal, and neurocognitive disease. Because target organ damage may begin in childhood and adolescence, routine blood pressure measurement must become a standard part of paediatric

care. Elevated readings should be confirmed with repeated, accurate measurements. Confirmed hypertension, but confirmed hypertension requires a structured approach to identify secondary causes, assess end-organ damage, and initiate timely management. Preventing hypertension-related complications in adulthood begins with detecting and treating hypertension in childhood.

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