Hypertension in Children

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Agenda

- Introduction
- Definition
- Measurement
- Etiology
- Pathophysiology
- Clinical manifestations
- Diagnosis
- Prevention
- Treatment

Introduction

- Hypertension in children is a growing problem.
- Multifactorial in origin.
- Hypertensive children, although usually asymptomatic, already manifest evidence of target organ damage. Up to 40% of hypertensive children have left ventricular hypertrophy and hypertensive children have increased carotid intima–media thickness, a marker of early atherosclerosis.
- Primary hypertension during childhood often tracks into adulthood. Children with BP >90th percentile have a 2.4-fold greater risk of having hypertension as adults. Similarly, nearly half of hypertensive adults had a BP >90th percentile as children. There is also an association between childhood hypertension and early atherosclerosis in young adulthood.
- Early intervention prevents development and progression of target organ damage.

Definition

- The definition of hypertension in adults is BP ≥140/90 mm Hg, regardless of body size, sex, or age. This is a functional definition that relates level of BP elevation with the likelihood of subsequent cardiovascular events.

- The definition of hypertension in children is statistical rather than functional. It includes normal values based on the normative distribution of BP in healthy children and tables with systolic and diastolic values for the 50th, 90th, 95th, and 99th percentile by age, sex, and height percentile.

Definition

- So, **hypertension** is defined as Average systolic blood pressure (SBP) and/or diastolic BP that is ≥95th percentile for age, sex, and height on ≥3 occasions.

Adolescents ≥13 y/o with BP ≥130/80 are considered to be hypertensive.

- **Prehypertension** is defined as average SBP or diastolic BP that are ≥90th percentile but ≤95th percentile in a medical setting but normal BP outside of the office has **white coat hypertension**.

Adolescents ≥13 y/o with BP levels greater than or equal to 120/80 mmHg should be considered to have elevated BP (prehypertension).

Definition

- Studies further recommended that if BP is ≥95th percentile, then the hypertension should be staged.
- Children with BP between the 95th and 99th percentile plus 5 mm Hg are categorized as **stage 1 hypertension**
- And children with BP above the 99th percentile plus 5 mm Hg have **stage 2 hypertension**.
- Stage 1 hypertension, if asymptomatic and without target organ damage, allows time for evaluation before starting treatment, whereas stage 2 hypertension calls for more prompt evaluation and pharmacologic therapy.

Classification of blood pressure in children and adolescents

Table 166-1	Classification of Blood Pressure
BLOOD PRESSURE CATEGORY	**BLOOD PRESSURE PERCENTILE (%)**
Normal	<90th
Prehypertension	*90th to 95th
Stage 1 hypertension	95th to (99th + 5 mm Hg)
Stage 2 hypertension	>99th + 5 mm Hg

*If 90th % is >120/80, use 120/80 as the lower limit.

Classification of blood pressure in Pediatrics up to 12 years old

Age	SBP(mm of Hg)	DBP(mm of Hg)
Newborn	50-70	25-45
6mths-1 yr	60-90	50-70
1-6 yrs	70-100	40-50
7-12yrs	90-110	50-70

Classification of blood pressure in Children 13 years and older

For Children Aged ≥13 y
Normal BP: <120/<80 mm Hg
Elevated BP: 120/<80 to 129/<80 mm Hg
Stage 1 HTN: 130/80 to 139/89 mm Hg
Stage 2 HTN: ≥140/90 mm Hg

Classification of blood pressure in Boys

Blood Pressure Levels for Boys by Age and Height Percentile

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	80	81	83	85	87	88	89	34	35	36	37	38	39	39
	90th	94	95	97	99	100	102	103	49	50	51	52	53	53	54
	95th	98	99	101	103	104	106	106	54	54	55	56	57	58	58
	99th	105	106	108	110	112	113	114	61	62	63	64	65	66	66
2	50th	84	85	87	88	90	92	92	39	40	41	42	43	44	44
	90th	97	99	100	102	104	105	106	54	55	56	57	58	58	59
	95th	101	102	104	106	108	109	110	59	59	60	61	62	63	63
	99th	109	110	111	113	115	117	117	66	67	68	69	70	71	71
3	50th	86	87	89	91	93	94	95	44	44	45	46	47	48	48
	90th	100	101	103	105	107	108	109	59	59	60	61	62	63	63
	95th	104	105	107	109	110	112	113	63	63	64	65	66	67	67
	99th	111	112	114	116	118	119	120	71	71	72	73	74	75	75
4	50th	88	89	91	93	95	96	97	47	48	49	50	51	51	52
	90th	102	103	105	107	109	110	111	62	63	64	65	66	66	67
	95th	106	107	109	111	112	114	115	66	67	68	69	70	71	71
	99th	113	114	116	118	120	121	122	74	75	76	77	78	78	79
5	50th	90	91	93	95	96	98	98	50	51	52	53	54	55	55
	90th	104	105	106	108	110	111	112	65	66	67	68	69	69	70
	95th	108	109	110	112	114	115	116	69	70	71	72	73	74	74
	99th	115	116	118	120	121	123	123	77	78	79	80	81	81	82
6	50th	91	92	94	96	98	99	100	53	53	54	55	56	57	57
	90th	105	106	108	110	111	113	113	68	68	69	70	71	72	72
	95th	109	110	112	114	115	117	117	72	72	73	74	75	76	76
	99th	116	117	119	121	123	124	125	80	80	81	82	83	84	84
7	50th	92	94	95	97	99	100	101	55	55	56	57	58	59	59
	90th	106	107	109	111	113	114	115	70	70	71	72	73	74	74
	95th	110	111	113	115	117	118	119	74	74	75	76	77	78	78
	99th	117	118	120	122	124	125	126	82	82	83	84	85	86	86
8	50th	94	95	97	99	100	102	102	56	57	58	59	60	60	61
	90th	107	109	110	112	114	115	116	71	72	72	73	74	75	76
	95th	111	112	114	116	118	119	120	75	76	77	78	79	79	80
	99th	119	120	122	123	125	127	127	83	84	85	86	87	87	88
9	50th	95	96	98	100	102	103	104	57	58	59	60	61	61	62
	90th	109	110	112	114	115	117	118	72	73	74	75	76	76	77
	95th	113	114	116	118	119	121	121	76	77	78	79	80	81	81
	99th	120	121	123	125	127	128	129	84	85	86	87	88	88	89
10	50th	97	98	100	102	103	105	106	58	59	60	61	61	62	63
	90th	111	112	114	115	117	119	119	73	73	74	75	76	77	78
	95th	115	116	117	119	121	122	123	77	78	79	80	81	81	82
	99th	122	123	125	127	128	130	130	85	86	86	88	88	89	90

Classification of blood pressure in Girls

Blood Pressure Levels for Girls by Age and Height Percentile

Age (Year)	BP Percentile ↓	Systolic BP (mmHg) ← Percentile of Height →							Diastolic BP (mmHg) ← Percentile of Height →						
		5th	10th	25th	50th	75th	90th	95th	5th	10th	25th	50th	75th	90th	95th
1	50th	83	84	85	86	88	89	90	38	39	39	40	41	41	42
	90th	97	97	98	100	101	102	103	52	53	53	54	55	55	56
	95th	100	101	102	104	105	106	107	56	57	57	58	59	59	60
	99th	108	108	109	111	112	113	114	64	64	65	65	66	67	67
2	50th	85	85	87	88	89	91	91	43	44	44	45	46	46	47
	90th	98	99	100	101	103	104	105	57	58	58	59	60	61	61
	95th	102	103	104	105	107	108	109	61	62	62	63	64	65	65
	99th	109	110	111	112	114	115	116	69	69	70	70	71	72	72
3	50th	86	87	88	89	91	92	93	47	48	48	49	50	50	51
	90th	100	100	102	103	104	106	106	61	62	62	63	64	64	65
	95th	104	104	105	107	108	109	110	65	66	66	67	68	68	69
	99th	111	111	113	114	115	116	117	73	73	74	74	75	76	76
4	50th	88	88	90	91	92	94	94	50	50	51	52	52	53	54
	90th	101	102	103	104	106	107	108	64	64	65	66	67	67	68
	95th	105	106	107	108	110	111	112	68	68	69	70	71	71	72
	99th	112	113	114	115	117	118	119	76	76	76	77	78	79	79
5	50th	89	90	91	93	94	95	96	52	53	53	54	55	55	56
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	95th	107	107	108	110	111	112	113	70	71	71	72	73	73	74
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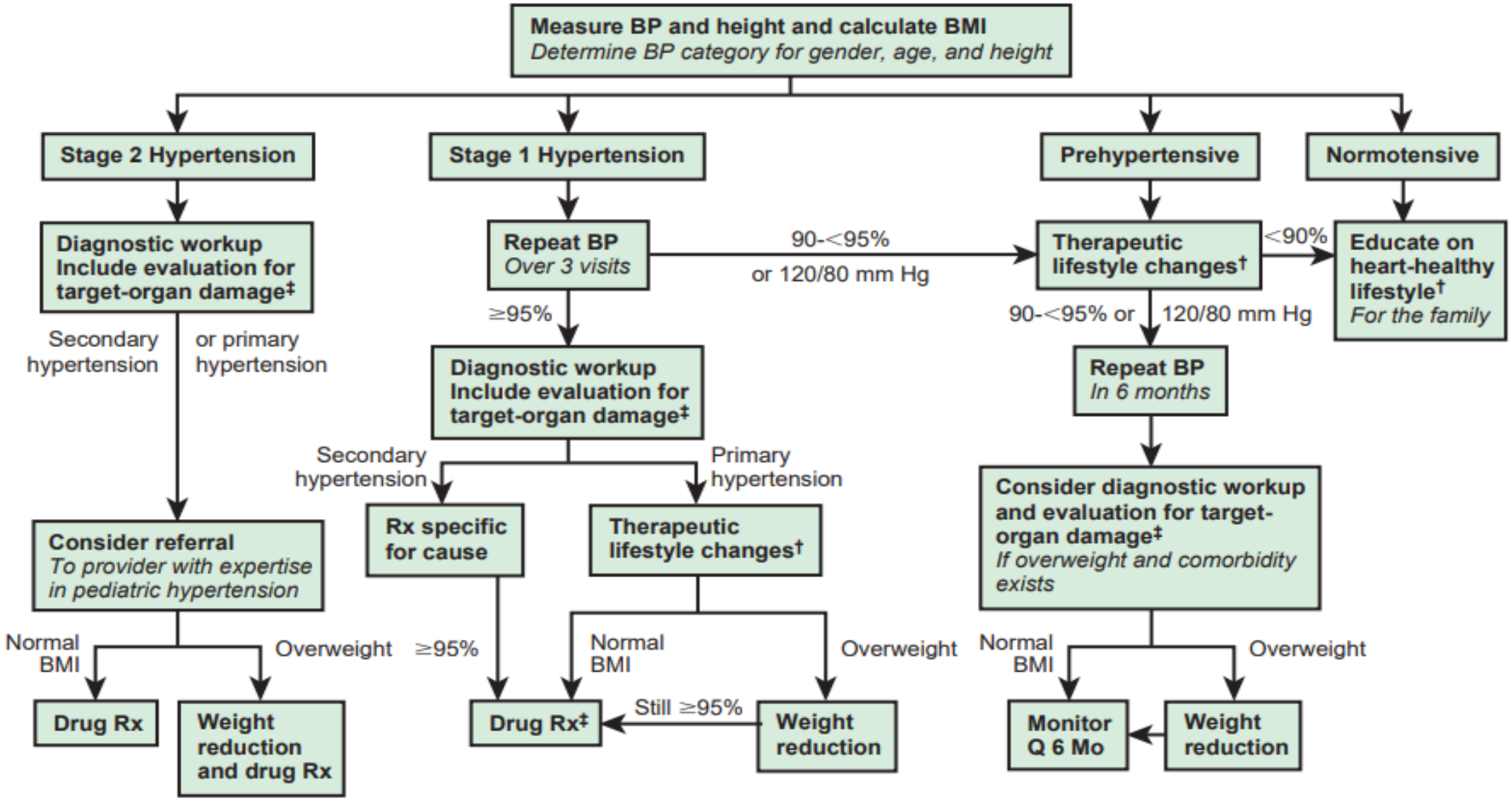
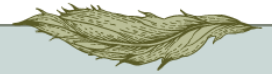
	99th	117	118	119	120	122	123	124	81	81	82	82	83	84	84
8	50th	95	95	96	98	99	100	101	57	57	57	58	59	60	60
	90th	108	109	110	111	113	114	114	71	71	71	72	73	74	74
	95th	112	112	114	115	116	118	118	75	75	75	76	77	78	78
	99th	119	120	121	122	123	125	126	82	82	83	83	84	85	86
9	50th	96	97	98	100	101	102	103	58	58	58	59	60	61	61
	90th	110	110	112	113	114	116	116	72	72	72	73	74	75	75
	95th	114	114	115	117	118	119	120	76	76	76	77	78	79	79
	99th	121	121	123	124	125	127	127	83	83	84	84	85	86	87
10	50th	98	99	100	102	103	104	105	59	59	59	60	61	62	62
	90th	112	112	114	115	116	118	118	73	73	73	74	75	76	76
	95th	116	116	117	119	120	121	122	77	77	77	78	79	80	80
	99th	123	123	125	126	127	129	129	84	84	85	86	86	87	88



Management algorithm. BMI, body mass index; BP, blood pressure; Q, every; Rx, prescription; † diet modification and physical activity; ‡ especially if younger, very high BP, little or no family history, diabetic, or other risk factors. (From National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents.

When blood pressure should be measured and for who?



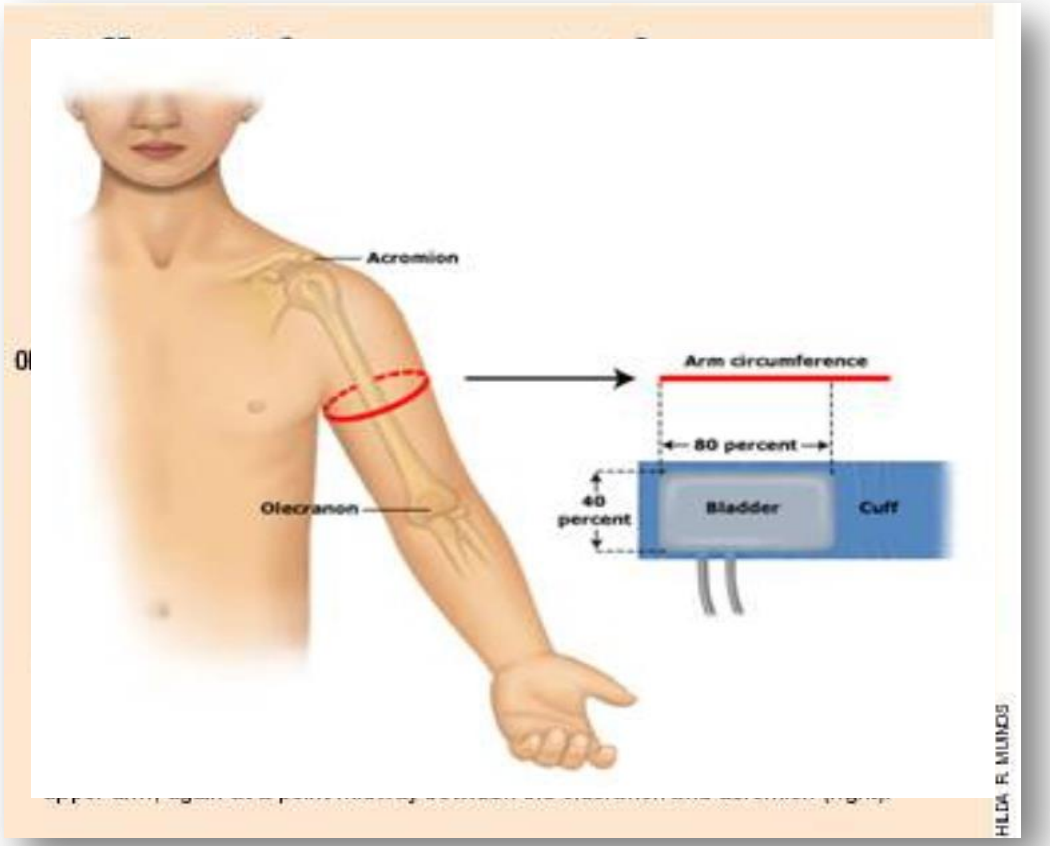
- The American Heart Association recommends that children **3 yrs or older** should have their BP checked during every healthcare episode (the AHA recommends **annual** BP checks).
- Selected children **<3 yrs** old should also have their BP checked under **special circumstances,** including those with a history of prematurity, congenital heart disease, renal disease, solid-organ transplant, cancer, treatment with drugs known to raise BP, other illnesses associated with hypertension (neurofibromatosis, tuberous sclerosis, others), or evidence of increased intracranial pressure.
- The preferred method is by auscultation and a BP cuff appropriate for the size of the child's arm should be used.

How should blood pressure be measured in children?

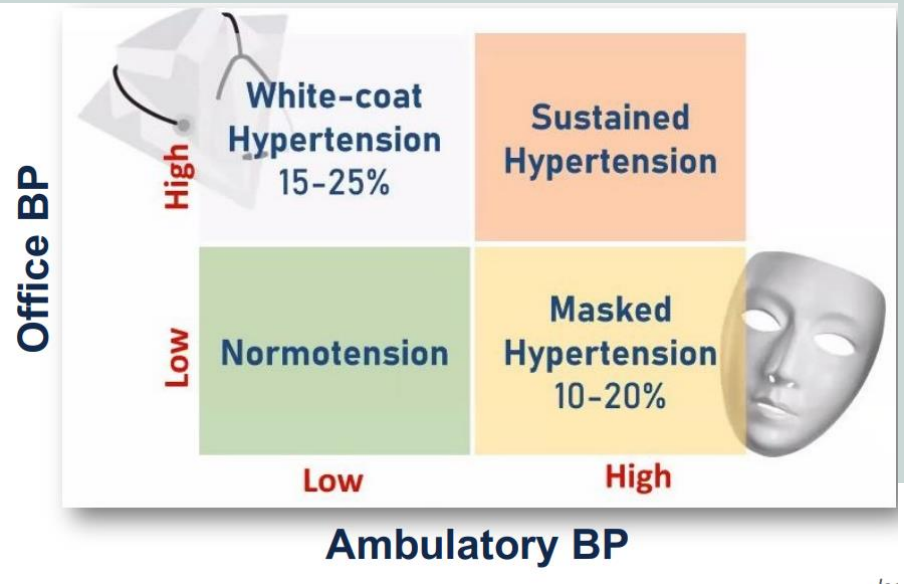
- Elevated readings should be confirmed on repeat visits before determining that a child is hypertensive.
- The BP should be measured with the child:
  - in the sitting position
  - back supported
  - feet on ground
  - after a period of quiet for at least 5 min.

Proper Cuff Size

- Careful attention to cuff size is necessary to avoid over diagnosis, as a cuff that is too short or narrow artificially increases BP readings.
- A wide variety of bladder sizes should be available in any medical office where children are routinely seen.
- An appropriate sized cuff has an inflatable bladder that is at least 40% of the arm circumference at a point midway along the upper arm. The inflatable bladder should cover at least two thirds of the upper arm length and 80-100% of its circumference.



Ambulatory blood pressure monitoring (ABPM)



- Ambulatory blood pressure monitoring (ABPM) is a procedure where the child wears a device that records BP frequently, usually every 20-30 min, throughout a 24 hr period while the child goes about usual daily activities, including sleep.
- This allows calculation of the mean daytime BP, sleep BP, and mean BP over 24 hr.
- The physician can also determine the proportion of BP measurements that are in the hypertensive range (BP load) and whether there is an appropriate decrease in BP during sleep (nocturnal dip).
- ABPM is particularly useful in the evaluation for white coat hypertension and may also be useful for determining risk of hypertensive target organ damage, evaluating resistance to pharmacologic therapy, and evaluating patients with hypotensive episodes on antihypertensive medication.
- ABPM is also useful for certain special populations, such as children with chronic kidney disease, kidney transplant, and diabetes mellitus where it may provide important information on cardiovascular risk that cannot be determined as well by office measurements.

Case 1

- An asymptomatic 16-year-old boy has elevated blood pressure documented on several visits, with an average blood pressure of 144/92 mm Hg.
- His height and weight are above the 97th percentile for age.
- His father has hypertension and takes antihypertensive medication.

What is the most appropriate approach for this boy?

1. Have the boy return for a repeat blood pressure measurement in 6 months.

2. Provide lifestyle counseling to increase physical activity and lower dietary salt and repeat blood pressure measurement in 6 months.

3. Begin diagnostic evaluation for stage 2 hypertension.

4. Admit to the hospital for immediate blood pressure reduction.

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Case 1- Initial Diagnostic Evaluation

- ABPM is done, demonstrating sustained hypertension while awake and asleep, with only 7% SBP dipping.
- Urinalysis is normal. Creatinine is 0.7 mg/dL (62 µmol/L)
- Random glucose elevated. Triglycerides and LDL cholesterol elevated. HDL cholesterol low

The most likely explanation for HTN in this boy is:

1. Excess dietary sodium intake
2. Primary hypertension, based on a parent with hypertension.
3. Secondary to pre-diabetes
4. Secondary to obesity

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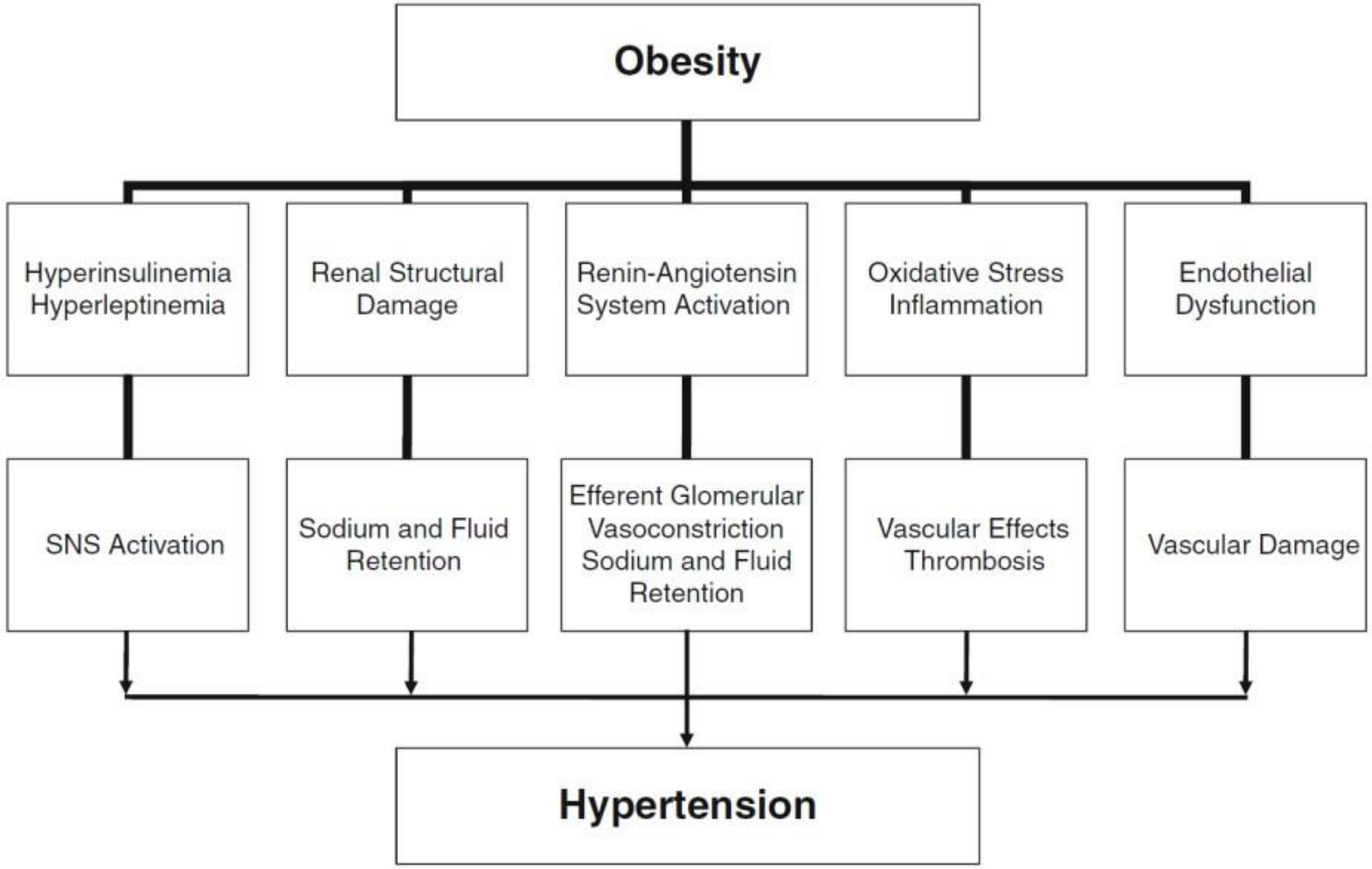
Pathophysiology of Obesity HTN



Case 1 Therapy: Initial Approach

- Weight loss is primary therapy but difficult to achieve

- Increased Physical Activity
  - 2017 AAP CPG: “Vigorous” physical activity 3-5 d/wk, 30-60 min/session
  - Aerobic exercise or combination of aerobic exercise plus resistance training
  - Try to find an activity child is already participating in and intensify it

- Nutritional Counseling
  - 2017 AAP CPG: Provide advice on the DASH diet
  - DASH eating plan: increased fruits and vegetables, low-fat dairy products ± sodium restriction (www.dashdiet.org)
  - AHA: Reduce sodium intake to 1500-2300 mg/day

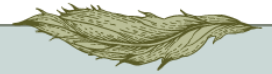
Family-based intervention improves success

Case 1: Outcome

- He met with a nutritionist who taught him about healthy eating
  - Reduced sodium intake
  - Cut down on snacks and portion sizes at meals
- His father started taking him to the gym 4 days per week
  - He used the treadmill and did weight training

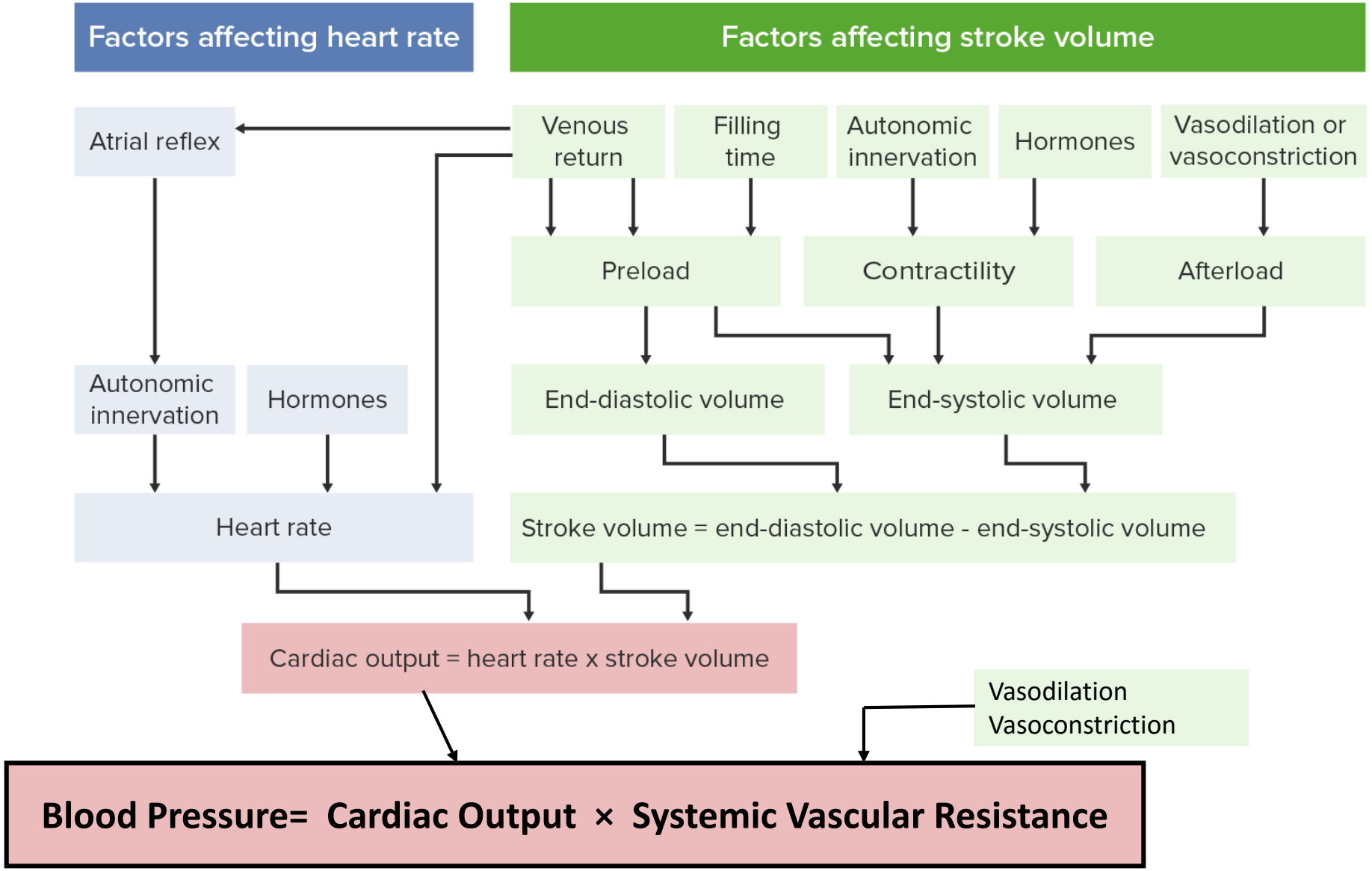
- Over a 2-year period he lost 15 lbs., and his BMI dropped from >97th percentile to the 93rd percentile
- His blood pressure fell to the elevated BP range – 120's/70s

Etiology and Pathophysiology



- Blood pressure is the product of cardiac output (CO) and systemic vascular resistance (SVR).
- An increase in either CO or PVR results in an increase in BP.
- If either of these factors increases while the other decreases, BP may not increase.
- When hypertension is the result of **another disease** process, it is referred to as ***secondary*** *hypertension.*
- When **no identifiable cause** can be found, it is referred to as ***primary*** *hypertension.*

Pathophysiology



https://www.lecturio.com/concepts/cardiac-mechanics/

Etiology and Pathophysiology

- Secondary hypertension is most common in infants and younger children.
- It is most often caused by renal abnormalities(90%); additional etiologies include cardiovascular disease and endocrinopathies.

- **Younger age**, **severely elevated BP**, and **symptomatic** hypertension make a **secondary cause** of hypertension more **likely**.

- Many childhood diseases can be responsible for *chronic hypertension* (**Table 1**) or *acute/intermittent hypertension* (**Table 2**).

- The most likely cause varies with age.
- Hypertension in the **premature infant** is sometimes associated with:
  umbilical artery catheterization, renal artery thrombosis, or bronchopulmonary dysplasia.
- Hypertension during **early childhood** may be caused by:
  renal disease, coarctation of the aorta, endocrine disorders, or medications.

Causes of Hypertension

PRIMARY HYPERTENSION	• Essential hypertension • Metabolic syndrome
RENAL CAUSES	• Congenital anomalies (renal dysplasia, obstructive uropathy) • Structural disorders (Wilms tumor, polycystic kidney disease) • Glomerulonephritis • Acquired injury (renal scarring, acute tubular necrosis)
ENDOCRINE CAUSES	• Catecholamine-secreting tumors (pheochromocytoma, neuroblastoma) • Hypercortisolism (Cushing syndrome) • Hyperaldosteronism • Hyperthyroidism
NEUROLOGICAL CAUSES	• Increased sympathetic activity (stress, anxiety, pain) • Dysautonomia • Increased intracranial pressure
VASCULAR CAUSES	• Coarctation of the aorta • Renal artery stenosis • Renal artery embolism (from umbilical artery catheter) • Renal vein thrombosis • Vasculitis
OTHER CAUSES	• Obstructive sleep apnea • Medications, illicit drugs

Table:1 Conditions Associated With Chronic Hypertension in Children

Renal

- Recurrent pyelonephritis/renal scarring
- Chronic glomerulonephritis
- Prematurity
- Congenital dysplastic kidney
- Polycystic kidney disease
- Vesicoureteral reflux nephropathy
- Segmental hypoplasia (Ask-Upmark kidney)
- Obstructive kidney disease
- Renal tumors
- Renal trauma
- Systemic lupus erythematosus (other connective tissue diseases)

Vascular

- Coarctation of thoracic or abdominal aorta
- Renal artery lesions (stenosis, fibromuscular dysplasia, thrombosis, aneurysm)
- Umbilical artery catheterization with thrombus formation
- Neurofibromatosis (intrinsic or extrinsic narrowing for vascular lumen)
- Renal vein thrombosis
- Vasculitis (ANCA associated, polyarteritis nodosa, Takayasu arteritis)
- Arteriovenous shunt
- Williams-Beuren syndrome
- Moyamoya disease

Endocrine

- Hyperthyroidism
- Congenital adrenal hyperplasia (11β-hydroxylase and 17-hydroxylase
- defect)
- Cushing syndrome
- Primary hyperaldosteronism
- Apparent mineralocorticoid excess
- Glucocorticoid remedial aldosteronism (familial aldosteronism type 1)
- Glucocorticoid resistance (Chrousos syndrome)
- Pseudohypoaldosteronism type 2 (Gordon syndrome)
- Pheochromocytoma
- Other neural crest tumors (neuroblastoma, ganglioneuroblastoma, ganglioneuroma)
- Liddle syndrome
- Geller syndrome

Central Nervous System

- Intracranial mass
- Hemorrhage
- Residual following brain injury
- Quadriplegia (dysautonomia)
- Sleep disordered breathing

Table:2 Conditions Associated With Transient or Intermittent Hypertension in Children

Renal

- Acute postinfectious glomerulonephritis
- Henoch-Schönlein purpura with nephritis
- Hemolytic-uremic syndrome
- Acute kidney injury
- After renal transplantation (immediately and during episodes of rejection)
- Hypervolemia
- Pyelonephritis
- Renal trauma
- Leukemic infiltration of the kidney

Drugs and Poisons

- Cocaine
- Oral contraceptives
- Sympathomimetic agents
- Amphetamines
- Phencyclidine
- Corticosteroids and adrenocorticotropic hormone
- Cyclosporine, sirolimus, or tacrolimus treatment after transplantation
- Licorice (glycyrrhizic acid)
- Lead, mercury, cadmium, thallium
- Antihypertensive withdrawal (clonidine, methyldopa, propranolol)
- Vitamin D intoxication

Central and Autonomic Nervous System

- Increased intracranial pressure
- Guillain-Barré syndrome
- Burns
- Familial dysautonomia
- Stevens-Johnson syndrome
- Posterior fossa lesions
- Porphyria
- Poliomyelitis
- Encephalitis
- Spinal cord injury (autonomic storm)

Miscellaneous

- Preeclampsia
- Pain, anxiety
- Hypercalcemia
- After coarctation repair
- White blood cell transfusion
- Extracorporeal membrane oxygenation (ECMO)

Secondary Hypertension

- **Renal disease** (e.g., chronic glomerulonephritis, reflux or obstructive nephropathy, hemolytic-uremic syndrome, polycystic kidney disease, congenital anomalies of the kidney and urinary tract) and **renovascular hypertension** account for approximately **90%** of children with secondary hypertension.
- Renal parenchymal disease and renal artery stenosis lead to water and sodium retention thought to be, in part, secondary to increased renin secretion.
- Coarctation of the aorta must always be considered.
- Several endocrinopathies are associated with hypertension, usually those involving the thyroid, parathyroid, and adrenal glands.
- Systolic hypertension and tachycardia are common in hyperthyroidism; DBP is not usually elevated.
- Hypercalcemia , whether secondary to hyperparathyroidism or other causes, often results in mild elevation in BP because of an increase in vascular tone.
- Adrenocortical disorders (e.g., aldosterone-secreting tumors, sodium-retaining congenital adrenal hyperplasia, Cushing syndrome) may produce hypertension in patients with increased mineralocorticoid secretion.
- It is important to consider conditions associated with real or apparent mineralocorticoid excess and thus a suppressed renin level (with or without hypokalemia) form of secondary hypertension **(Table 3).**
- hypercortisolism → stimulation of aldosterone receptors in high concentrations and ↑ potassium excretion → ↑ blood pressure
- ↑ Aldosterone → ↑ Na+ reabsorption and retention → water retention → hypertension

Table:3 Clinical Findings in Patients With Mineralocorticoid Excess

CONDITION	CLINICAL PRESENTATION
▪ CAH: 11β-hydroxylase deficiency	Early growth spurt initially, then short adult stature, advanced bone age, premature adrenarche, acne, precocious puberty in males, amenorrhea/ hirsutism/ virilism in females (autosomal recessive)
▪ CAH: 17α-hydroxylase deficiency	Pseudohermaphroditism (male), sexual infantilism (female) (autosomal recessive)
▪ Apparent mineralocorticoid excess	Growth retardation/short stature, nephrocalcinosis (autosomal recessive)
▪ Liddle syndrome	Severe hypertension, hypokalemia, and metabolic alkalosis, muscle weakness (autosomal dominant)
▪ Geller syndrome (exacerbated by pregnancy)	Early onset of hypertension (before age 20 yr), exacerbated in pregnancy
▪ Glucocorticoid-remediable aldosteronism (GRA) (familial aldosteronism type 1)	Early onset of hypertension, presence of family history of mortality or morbidity from early hemorrhagic stroke (autosomal dominant)
▪ Pseudohypoaldosteronism type 2 (Gordon syndrome)	Short stature, hyperkalemic and hyperchloremic metabolic acidosis, borderline blood pressure (autosomal dominant))
▪ Glucocorticoid resistance (children) (Chrousos syndrome)	Ambiguous genitalia, precocious puberty; women may have androgen excess: acne, excessive hair, oligo/anovulation, infertility (familial or sporadic)

Secondary Hypertension

- Pheochromocytomas are catecholamine-secreting tumors that give rise to hypertension because of the cardiac and peripheral vascular effects of epinephrine and norepinephrine.
- Children with pheochromocytoma usually have sustained rather than intermittent or exercise-induced hypertension.
- Pheochromocytoma develops in approximately 5% of patients with neurofibromatosis and can also be seen in certain genetic disorders such as von Hippel–Lindau disease.
- Rarely, secondary hypertension can be caused by pseudohyperaldosteronism , which leads to elevated BP in the face of a suppressed renin level. Such disorders include Liddle syndrome, apparent mineralocorticoid excess, and glucocorticoid-remediable aldosteronism.
- Altered sympathetic tone can be responsible for acute or intermittent elevation of BP in children with Guillain-Barré syndrome, poliomyelitis, burns, and Stevens- Johnson syndrome. Intracranial lesions also affect sympathetic outflow from the central nervous system.

Secondary Hypertension

- A number of **drugs of abuse , therapeutic agents , and toxins** may cause hypertension.
- Cocaine may provoke a rapid increase in BP and can result in seizures or intracranial hemorrhage.
- Phencyclidine causes transient hypertension that may become persistent in chronic abusers.
- Tobacco use may also increase BP.
- Sympathomimetic agents used as nasal decongestants, appetite suppressants, and stimulants for attention-deficit disorder produce peripheral vasoconstriction and varying degrees of cardiac stimulation. Individuals vary in their susceptibility to these effects.
- Oral contraceptives should be suspected as a contributor to elevated BP in adolescent girls, although the incidence is lower with the use of low-estrogen preparations.
- Immunosuppressant agents such as cyclosporine and tacrolimus cause hypertension in organ transplant recipients, and the effect is exacerbated by the co-administration of corticosteroids.
- BP may be elevated in patients with poisoning by a heavy metal (lead, cadmium, mercury).

Primary Hypertension

- In older school-age children and adolescents, **primary hypertension becomes increasingly common**.
  These patients often are overweight, have a strong family history of hypertension, and have BP values at, or only slightly above, the 95th percentile for age.

- Isolated systolic hypertension is also more consistent with primary hypertension, whereas diastolic hypertension may suggest a secondary cause.

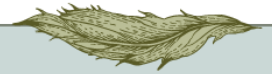
- The **cause of primary hypertension** is likely to be **multifactorial**;
  obesity, genetic alterations in calcium and sodium transport, vascular smooth muscle reactivity, the renin-angiotensin-aldosterone system (RAAS), sympathetic nervous system over activity, and insulin resistance have been implicated in this disorder.

- Elevated uric acid levels may play a role in the pathophysiology of primary hypertension, and proof-of-concept studies have confirmed that lowering of uric acid levels results in lower BP in overweight youth with hypertension or prehypertension.

Primary Hypertension

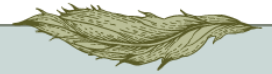
- Some children and adolescents demonstrate salt-sensitive hypertension , a factor that is ameliorated with weight loss and sodium restriction.
- Normotensive children of hypertensive parents may show abnormal physiologic responses that are similar to those of their parents. When subjected to stress or competitive tasks, the offspring of hypertensive adults, as a group, respond with greater increases in heart rate and BP than do children of normotensive parents.
- Similarly, some children of hypertensive parents may excrete higher levels of urinary catecholamine metabolites or may respond to sodium loading with greater weight gain and increases in BP than do those without a family history of hypertension.
- The abnormal responses in children with affected parents tend to be greater in the black population than among white individuals.

Clinical Manifestations



- Children and adolescents with **primary** hypertension are **usually asymptomatic**.
- the **BP elevation** is usually **mild** and is detected during a routine examination or evaluation before athletic participation. These children may also be obese.
- Children with **secondary** hypertension can have **BP elevations** ranging from **mild** to **severe**.
- Unless the BP has been sustained or is rising rapidly, hypertension does not usually produce symptoms.
- Therefore, clinical manifestations may instead reflect the underlying disease process, such as growth failure in children with CKD.
- Children and adolescents with **acute severe hypertension** , in contrast, present with BP elevation well **above stage 2 (>99th +5mmhg)** hypertension and **severe symptoms** that may represent acute target-organ injury.
- Subclinical hypertensive **target-organ injury** is a common clinical manifestation in children with primary hypertension. Using echocardiography with pediatric normative data, left ventricular hypertrophy is detected in up to 40% of hypertensive children.
- Other markers of target-organ damage that have been demonstrated in hypertensive children include: hypertensive retinopathy, increased carotid intima-to-media thickness, and increased vascular stiffness.
- Children with prehypertension also have evidence of target-organ damage, often at a magnitude intermediate between that of normotensive and hypertensive children.

Goals of the evaluation



- Distinguish between primary and secondary HTN
- Uncovering potential underlying causes of the hypertension
- Evaluating for comorbidities
- Identify patients for whom antihypertensive drug therapy is warranted
- Screening for evidence of target organ damage
- The extent of the evaluation for underlying causes of hypertension depends on the type of hypertension that is suspected.

History

Age

- Secondary HTN is more likely in younger children, especially those less than 6 years of age . While older children and adolescents are more likely to have primary HTN.

Onset

- Acute, severe onset is caused by drug toxicity , coarctation of aorta or hypertensive encephalopathy.

Associated symptoms :

- Abdominal pain, dysuria, frequency, nocturia, enuresis, hematuria, and edema may indicate a renal cause
- In infants, growth failure, irritability, and feeding problems may be symptoms of HTN
- Joint pain or swelling may be due to collagen vascular diseases
- Weight loss, sweating, and pallor may be due to a catecholamine- secreting tumor.

History

- Muscle cramps or weakness and constipation may be seen with the hypokalemia associated with hyperaldosteronism
- Menstrual disorders, hirsutism, and virilization may indicate forms of congenital adrenal hyperplasia (CAH) associated with HTN
- A neonatal history of umbilical artery line placement can result in renal artery embolization, leading to HTN
- History of prolonged loud snoring may identify sleep- related causes of HTN
- Hypertensive encephalopathy may occur as nausea, vomiting, altered mental status, visual disturbances, seizures, or stroke.
- Intermittent HTN may be present in patients with autonomic instability (e.g., Guillain-Barré syndrome, burns, poliomyelitis, Stevens-Johnson syndrome, porphyria)
- Family history of hypertension , early deaths or renal diseases .
- History of drug intake

History in the child or adolescent with elevated blood pressure

History	Possible cause of hypertension
CNS: Head trauma, headache, visual disturbance lethargy. seizures, tremors morning vomiting	Elevated intracranial pressure
Hearing: Hearing loss	Renal disease (ie, Alport syndrome)
	Lead poisoning
Cardiovascular: Palpitations, irregular pulse	Catecholamine excess
Renal: Edema, history of UTI or unexplained fever, abnormal urine color, enuresis, flank pain, dysuria	Renal disease or condition (eg. pyelonephritis, acute glomerulonephritis, acute kidney injury. and chronic kidney disease)
Skin: Rash, sweating pallor	Catecholamine excess
	Thyroid dysfunction
	Renal vasculitis
Recent medical history: Recent pharyngitis or	Post-infectious glomerulonephritis
impetigo, exposure to sources of enterohemorrhagic E. coll	Hemolytic uremic syndrome
Medications: Sympathomimetics oral contraceptives, corticosteroids	Side effect of medication
Substance use: Cocaine, amphetamines anabolic steroids phencyclidine, ephedra- containing alternative medications caffeine	Drug-mediated effects
Family history: Hypertension early MI. diabetes, stroke	Essential hypertension
Sexual history: Postmenarchal female actively engaged in sexual intercourse	Preeclampsia
Neonatal history: Use of umbilical artery catheters	Renovascular hypertension
Growth history: Excessive weight gain or loss, change in growth percentiles	Obesity, thyroid dysfunction
Dietary history: Types and amount of food ingested; salt craving	Obesity, essential hypertension
Social history: Stress factors at home and school	Stress

Physical Examination :

Physical Examination Finding	Possible Etiology
General	
Obesity	Essential Hypertension
Truncal Obesity	Cushing syndrome, Corticosteroid therapy
Growth Retardation	Chronic Kidney Disease
Vital Signs	
Tachycardia	Catecholamine excess (PCC or neuroblastoma) or Hyperthyroidism
BP difference in Extremeties	If upper extremity BP> Lower extremity BP, coarctation of aorta
Head and Neck	
Elfin face	Williams Syndrome
Moon Face	Cushing Syndrome, Corticosteroid therapy
Thyroid enlargement or goiter	Hyperthyroidism
Webbed Neck	Turner Syndrome
Tonsillar Hypertrophy	Sleep-disordered breathing, Sleep apnea

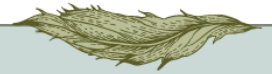
Physical Examination :

Physical Examination Finding	Possible Etiology
Eye	
Retinal changes	Suggest severe hypertension and secondary etiology
Papilledema	Increase intracranial pressure
Skin	
Pallor, flushing	Catecholamine excess (PCC and neuroblastoma)
Acne, hirsutism, striae	Cushing syndrome, corticosteroid therapy
Café-au-lait spots and/or neurofibromas	Neurofibromatosis
Ash leaf spots and/or adenoma sebaceum	Tuberous sclerosis
Rash	Lupus nephritis, Henoch-Schönlein purpura (IgA vasculitis)
Acanthosis nigricans	Type 2 diabetes
Chest	
Widely spaced nipples	Turner syndrome
Murmur	Coarctation of the aorta
Apical heave	Left ventricular hypertrophy
Abdomen	
Abdominal bruit	Renovascular disease
Mass	Hydronephrosis, polycystic kidney disease, renal tumors, neuroblastoma

Physical Examination :

Physical Examination Finding	Possible Etiology
Extremities	
Traction/casts	Orthopedic Manipulation
Asymmetry of limbs	Beckwith-Weidemann syndrome
Arthritis	Henoch-Schonlein purpura (igA vasculitis), Collagen vascular disease (systemic lupus erythematous)
Neurologic	
Muscle Weakness	Liddle syndrome, hyperaldosteronism
Diminished pain response	Familial dysautonomia
Genitalia	
Ambiguous/ virilization	Adrenal Hyperplasia
Advanced puberty	Intracranial tumors

Investigations



Initial evaluation

- CBC
- BUN/ creatinine
- Electrolytes, calcium
- Urinalysis
- Renal ultrasound

Consider

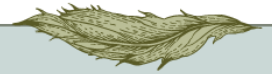
- Evaluation for co-morbidity
  - –Fasting lipid panel
  - –Fasting glucose
  - –Polysomnography (sleep study)
- Evaluation for target-organ damage
  - –Echocardiogram (LVH)
  - –Retinal exam

Investigations

Further evaluation as indicated
(stage 2, prepubertal age, findings specific to underlying condition)

- Free T4, TSH
- Ambulatory BP monitoring
- Plasma renin
- Renovascular imaging
- Plasma and urine catecholamines
- Plasma and urinary steroids
- Urine pregnancy test (if suspected)
- Cranial imaging (should be considered to rule out an intracranial mass in children with H and P indicating raised ICP)

Case 2



- 5 y/o boy, presents for routine well-child visit
- Not seen in > 2 years.
- BP's 137/85, 129/90, confirmed by you with manual sphygnomanometer
- What's the next step ?

Case 2 – DIAGNOSIS

History :

- Has had intermittent headaches without any accompanying symptoms
- Was a term baby with no neonatal complications and no prior hospitalizations or surgeries
- No medication or supplement use
- FH of HTN affecting father, 3 of 4 grandparents, mother has T2DM. No FH of kidney disease

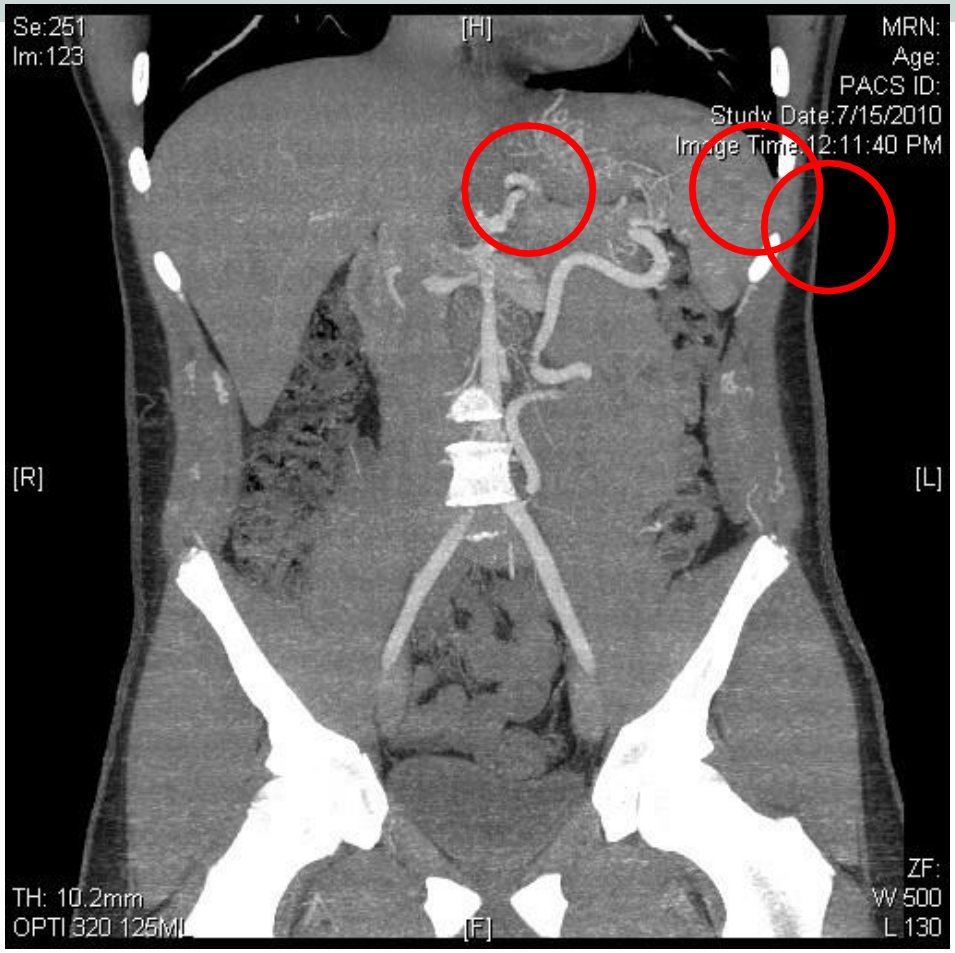
Physical examination

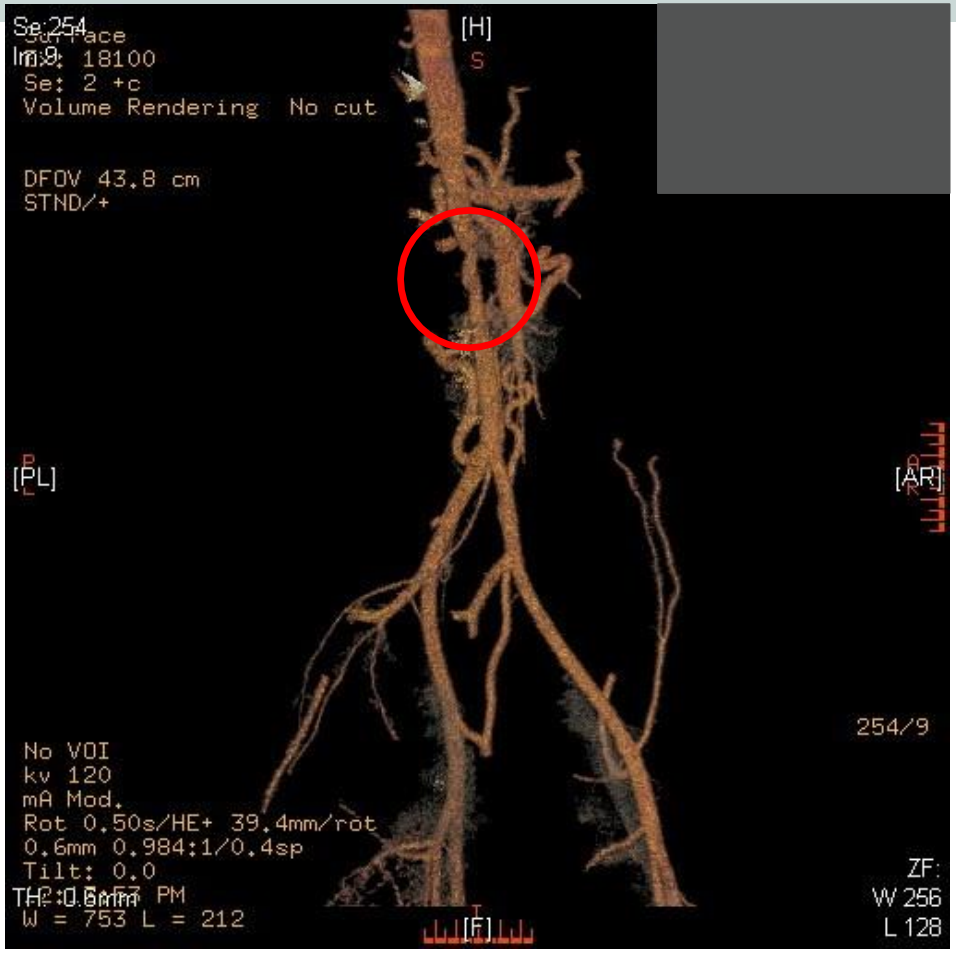
- Normal appearance
- Weight 33.9 kg (>97%tile)
- Height 118.1 cm (50%tile)
- BMI 24 kg/m2 (>97%tile)
- HEENT, cardiac, abdominal, GU exams all normal
- Referred to HTN Clinic
  - Referral BP's 137/85, 129/90
  - UE BP's in our office: 132/92, 128/88, 140/89
  - Mean BP: 133/80
    - 90th percentile: 107/68
    - 95th percentile: 111/71
    - 95th percentile + 12 mmHg: 123/83
  - LE BP's done: 102/55, 108/70
- **Thus he has stage 2 HTN**

Investigations

- Labs, imaging studies ordered
- Started on propranolol
- Normal UA, creatinine, electrolytes, elevated renin
- Echocardiogram: structurally normal heart with LVH
- Complete kidney US: kidneys of normal appearance and size bilaterally
- CT- angiogram performed

Case 2 - CT ANGIOGRAM

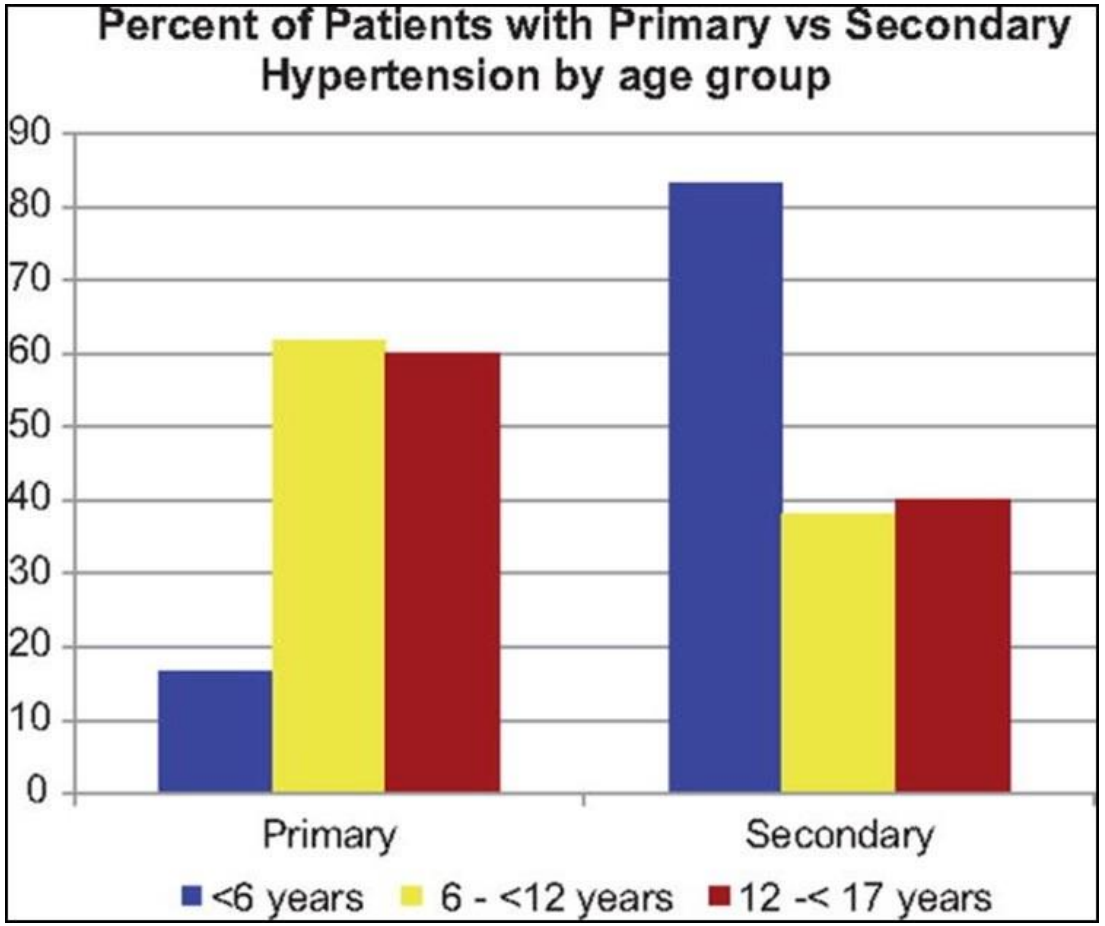




Relevant Guidance from the 2017 AAP CPG

- 16. Doppler renal ultrasonography may be used as a noninvasive screening study for the evaluation of possible RAS in normal wt children and adolescents ≥8 y of age who are suspected of having renovascular HTN and who will cooperate with the procedure.
  - C, moderate
- 17. In children and adolescents suspected of having RAS, either CTA or MRA may be performed as noninvasive imaging studies.
  - D, weak
- 11. Children and adolescents ≥6 y 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
  - -and/or do not have history or physical examination findings suggestive of a secondary cause of HTN.
  - C, Moderate

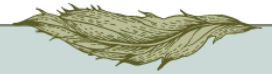
Distribution of HTN Causes by Age



Case 2: Follow-up

- Propranolol and amlodipine needed to control BP
- Repeat echo 6 mo later – improved LVH
- Followed with repeat kidney ultrasounds to monitor kidney growth
- Underwent surgical reconstruction of abdominal aorta and reimplantation of renal arteries bilaterally
- Now off antihypertensive medications but still being closely followed

Prevention



- Prevention of high BP may be viewed as part of the prevention of cardiovascular disease and stroke, the leading cause of death in adults in the United States.

- Population approaches to prevention of primary hypertension include :
  - A reduction in obesity
  - Reduced sodium intake
  - an increase in physical activity through school- and community-based programs.

Treatment

- Children + Asymptomatic mild hypertension without evidence of target-organ damage:

1. **Lifestyle modification**
2. **Dietary changes:**
   DASH diet (diet increased in fresh fruits, fresh vegetables, fiber, and nonfat dairy and reduced in sodium)
3. **Regular exercise:**
   30-60 min on most days

Treatment

- **Indications for pharmacologic therapy include**
- symptomatic hypertension
- stage 2 hypertension without a modifiable risk factor
- hypertension in patients with comorbidities such as diabetes (types 1 and 2) or CKD
- persistent hypertension despite nonpharmacologic measures.
- **Acceptable initial agents for use in children:**
- Angiotensin-converting enzyme inhibitors (ACEIs)
- Angiotensin receptor blockers (ARBs)
- Thiazide diuretics
- Calcium channel blockers
- The choice of antihypertensive agent for a patient should be tailored to the **etiology of that patient's hypertension** whenever possible.

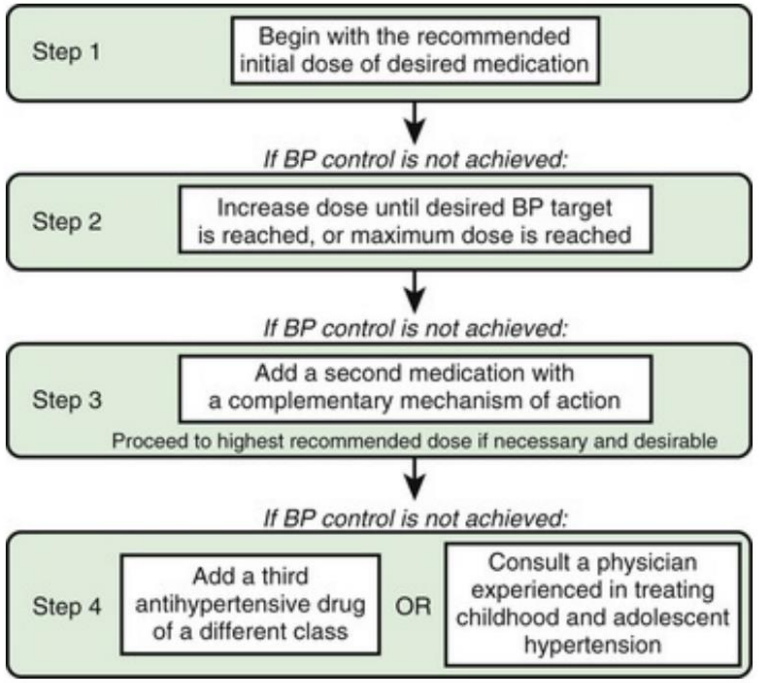


FIG. 472.3 Stepped-care approach to antihypertensive therapy in children and adolescents. BP, Blood pressure. (From Flynn JT, Daniels SR: Pharmacologic treatment of hypertension in children and adolescents, *J Pediatr* 149:746–754, 2006, Fig 2, p

Treatment

CLASS	DRUG	STARTING DOSE	INTERVAL	MAXIMUM DOSE*
Aldosterone receptor antagonist	Eplerenone	25 mg/day	qd-bid	100 mg/day
	Spironolactone †	1 mg/kg/day	qd-bid	3.3 mg/kg/day up to 100 mg/day
Angiotensin-converting enzyme inhibitors	Benazepril †	0.2 mg/kg/day up to 10 mg/day	qd	0.6 mg/kg/day up to 40 mg/day
	Captopril †	0.5 mg/kg/dose (0.05 mg/kg/dose in infants)	tid	6 mg/kg/day up to 450 mg/day
	Enalapril †	0.08 mg/kg/day	qd	0.6 mg/kg/day up to 40 mg/day
	Fosinopril	0.1 mg/kg/day up to 10 mg/day	qd	0.6 mg/kg/day up to 40 mg/day
	Lisinopril †	0.07 mg/kg/day up to 5 mg/day	qd	0.6 mg/kg/day up to 40 mg/day
	Quinapril	5-10 mg/day	qd	80 mg/day
	Ramipril	1.6 mg/m^2 /day	qd	6 mg/m^2 /day up to 10 mg/day
Angiotensin receptor blockers	Candesartan	1-6 yr: 0.2 mg/kg/day 6-17 yr: <50 kg 4-8 mg qd >50 kg 8-16 mg qd	qd	1-6 yr: 0.4 mg/kg up to 4 mg/day 6-17 yr: <50 kg: 16 mg qd >50 kg: 32 mg qd
	Losartan †	0.75 mg/kg/day up to 50 mg/day	qd	1.4 mg/kg/day up to 100 mg/day
	Olmesartan	20 to <35 kg 10 mg qd; ≥35 kg 20 mg qd	qd	20 to <35 kg: 20 mg qd ≥35 kg: 40 mg qd
	Valsartan †	6-17 yr: 1.3 mg/kg/day up to 40 mg/day	qd	6-17 yr: 2.7 mg/kg/day up to 160 mg/day
α- and β-Adrenergic antagonists	Labetalol †	2-3 mg/kg/day	bid	10-12 mg/kg/day up to 1.2 g/day
	Carvedilol	0.1 mg/kg/dose up to 6.25 mg bid	bid	0.5 mg/kg/dose up to 25 mg bid
β-adrenergic antagonists	Atenolol †	0.5-1 mg/kg/day	qd-bid	2 mg/kg/day up to 100 mg/day
	Bisoprolol/HCTZ	2.5/6.25 mg/day	qd	10/6.25 mg/day
	Metoprolol	1-2 mg/kg/day	bid	6 mg/kg/day up to 200 mg/day
	Propranolol	1 mg/kg/day	bid-tid	8 mg/kg/day up to 640 mg/day
Calcium channel blockers	Amlodipine †	1-5 yr: 0.1 mg/kg/day ≥6 yr: 2.5 mg/day	qd	1-5 yr: 0.6 mg/kg/day up to 5 mg/day ≥6 yr: 10 mg/day
	Felodipine	2.5 mg/day	qd	10 mg/day
	Isradipine †	0.05-0.15 mg/kg/dose	tid-qid	0.6 mg/kg/day up to 10 mg/day
	Extended-release nifedipine	0.2-0.5 mg/kg/day	qd-bid	3 mg/kg/day up to 120 mg/day
Central α-agonist	Clonidine †	5-10 µg/kg/day	bid-tid	25 µg/kg/day up to 0.9 mg/day
Diuretics	Amiloride	5-10 mg/day	qd	20 mg/day
	Chlorthalidone	0.3 mg/kg/day	qd	2 mg/kg/day up to 50 mg/day
	Chlorothiazide	10 mg/kg/day	bid	20 mg/kg/day up to 375 mg/day
	Furosemide	0.5-2.0 mg/kg/dose	qd-bid	6 mg/kg/day
	HCTZ	0.5-1 mg/kg/day	qd	3 mg/kg/day up to 37.5 mg/day
Vasodilators	Hydralazine	0.25 mg/kg/dose	tid-qid	7.5 mg/kg/day up to 200 mg/day
	Minoxidil	0.1-0.2 mg/kg/day	bid-tid	1 mg/kg/day up to 50 mg/day

* The maximum recommended adult dose should never be exceeded.

† Information on preparation of a stable extemporaneous suspension is available for these agents.

bid, Twice daily; HCTZ, hydrochlorothiazide; qd, once daily; qid, 4 times daily; tid, 3 times daily.

Adapted from Flynn JT: Management of hypertension in the young: role of antihypertensive medications, *J Cardiovasc Pharmacol* 58(2)111−120, 2011.

Treatment

- There have been changes in the recommended BP goals for treatment of hypertension in children and adolescents.
- Data from **the SPRINT (SBP intervention)** trial group suggests that stricter goals **(SBP goal of 120 vs 140 mm Hg)** improve cardiovascular outcomes in adults.
- In children with **CKD**, the ESCAPE (Effects of Strict BP Control and Angiotensin-Converting Enzyme Inhibition on the Progress of Chronic Renal Failure in Pediatric Patients) trial group showed slower progression of CKD if the **24 hr MAPs were kept below the 50th percentile on ABPM compared to the 50th-95th percentile**.
- It is now recommended that treatment achieve BP such as headache, dizziness, or nausea/vomiting **(hypertensive urgency) and in more severe cases**, retinopathy, encephalopathy, cardiac failure, renal injury, and seizures**(hypertensive emergency)**

Hypertensive Encephalopathy (generalized or posterior reversible encephalopathy syndrome)

- It is suggested by the presence of

1. Headache
2. Vomiting
3. Temperature elevation
4. Visual disturbances
5. Ataxia
6. Depressed level of consciousness

- it is one of the more common presentations of acute severe hypertension in children and adolescents.

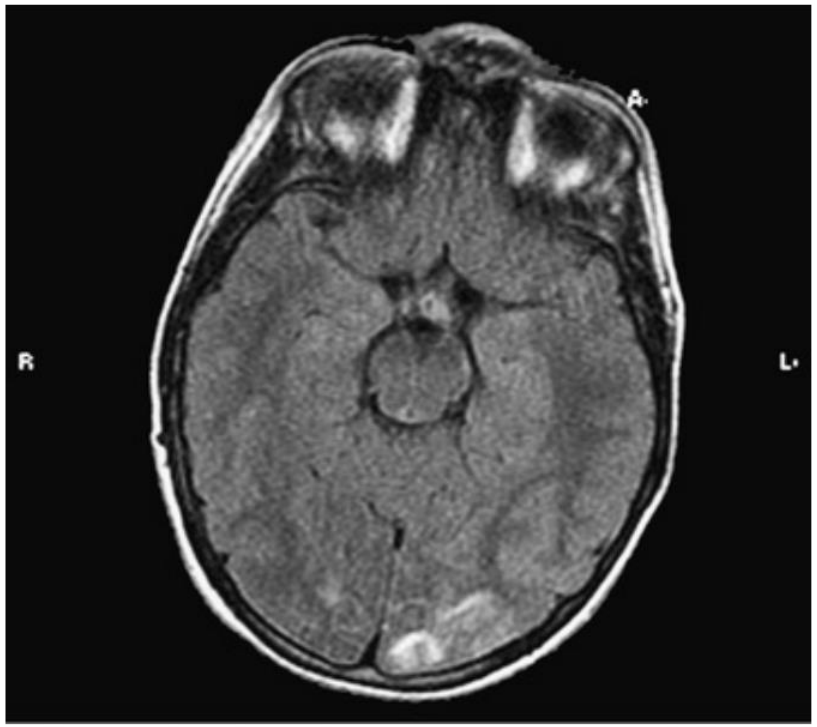


FIG. 472.4 Magnetic resonance image of brain of a 6 yr old boy with end-stage renal disease and hypertensive encephalopathy (i.e., posterior reversible leukoencephalopathy syndrome). Bilateral occipital high signal intensity is more pronounced on the left side. (From Daroff RB, Fenichel GM, Jankovic J, Mazziotta JC, editors, *Bradley's neurology in clinical practice,* ed 6, vol 2, Philadelphia, 2012, Elsevier Saunders, Fig 49B.4, p 924.)

Treatment

Manifest of Acute severe hypertension

- Decreased vision (cortical blindness)
- Papilledema
- Congestive heart failure
- Accelerated deterioration of renal function

Acute severe hypertension and life-threatening symptoms,

- Intensive care unit (ICU) admission
- Intravenous (IV) drug infusion
- Arterial lines should be used for continuous BP monitoring
- It is indicated so that decreases in BP can be carefully monitored and titrated

Drug of choice

- labetalol, nicardipine, and sodium nitroprusside.
- Why ?

1. Rapid a reduction in BP may interfere
2. Adequate organ perfusion

Antihypertensive Drugs for Management of Severe Hypertension in Children Age 1-17 yr.

DRUG	CLASS	DOSE	ROUTE	COMMENTS
USEFUL FOR SEVERELY HYPERTENSIVE PATIENTS WITH LIFE-THREATENING SYMPTOMS				
Esmolol	β-Adrenergic blocker	100-500 µg/kg/min	IV infusion	Very short acting—constant infusion preferred; may cause profound bradycardia
Hydralazine	Direct vasodilator	0.2-0.4 mg/kg/dose	IV, IM	Should be given every 4 hr when given IV bolus
Labetalol	α- and β-Adrenergic blocker	Bolus: 0.20-1.0 mg/kg/dose, up to 40 mg/dose Infusion: 0.25-3.0 mg/kg/hr	IV bolus or infusion	Asthma and overt heart failure are relative contraindications.
Nicardipine	Calcium channel blocker	Bolus: 30 µg/kg up to 2 mg/dose Infusion: 0.5-4 µg/kg/min	IV bolus or infusion	May cause reflex tachycardia
Sodium nitroprusside	Direct vasodilator	0.5-10 µg/kg/min	IV infusion	Monitor cyanide levels with prolonged (>72 hr) use or in renal failure; or co-administer with sodium thiosulfate.
USEFUL FOR SEVERELY HYPERTENSIVE PATIENTS WITH LESS SIGNIFICANT SYMPTOMS				
Clonidine	Central α-agonist	0.05-0.1 mg/dose, may be repeated up to 0.8 mg total dose	PO	Side effects include dry mouth and drowsiness.
Fenoldopam	Dopamine receptor agonist	0.2-0.8 µg/kg/min	IV infusion	Produced modest reductions in blood pressure in a pediatric clinical trial in patients up to age 12 yr
Hydralazine	Direct vasodilator	0.25 mg/kg/dose, up to 25 mg/dose	PO	Extemporaneous suspension stable for only 1 wk
Isradipine	Calcium channel blocker	0.05-0.15 mg/kg/dose, up to 5 mg/dose	PO	Stable suspension can be compounded.
Minoxidil	Direct vasodilator	0.1-0.2 mg/kg/dose, up to 10 mg/dose	PO	Most potent oral vasodilator; long acting

ACE, Angiotensin-converting enzyme; IM, intramuscular; IV, intravenous; PO, oral.

Adapted from Flynn JT, Tullus K: Correction to severe hypertension in children and adolescents: pathophysiology and treatment, *Pediatr Nephrol* 27(3):503–504, 2012.

Treatment

- In general, BP should be reduced by no more than 25% of the planned reduction over the 1st 8 hr., with a gradual normalization of BPs over next 24-48 hr.

- For patients with less severe symptoms, such as headache or nausea/vomiting,

1. Oral medications such as Clonidine or Isradipine can be used
2. Short-acting IV medications such as hydralazine or labetalol are

Treatment

- Treatment of secondary hypertension must also focus on the underlying disease, such as chronic renal disease, hyperthyroidism, pheochromocytoma, coarctation of the aorta, or renovascular hypertension.
- The treatment of renovascular stenosis includes antihypertensive medications, angioplasty, or surgery .
- If bilateral renovascular hypertension or renovascular disease in a solitary kidney is suspected, drugs acting on the RAAS are usually contraindicated because they may reduce glomerular filtration rate and lead to acute kidney injury.

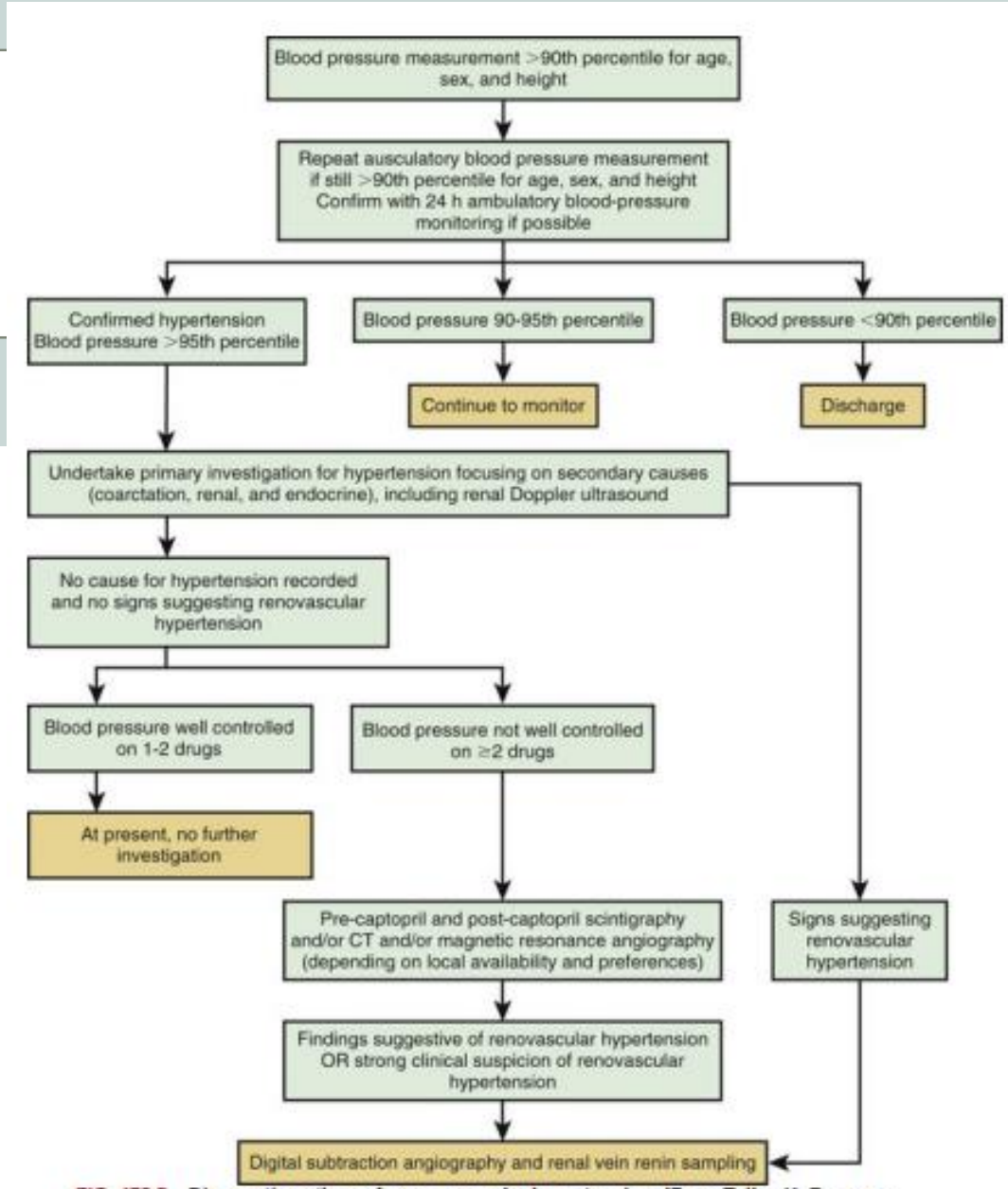


FIG. 472.5 **Diagnostic pathway for renovascular hypertension. (From Tullus K, Brennan E, Hamilton G, et al: Renovascular hypertension in children, *Lancet* 371:1453–1463, 2008, Fig 6, p 1458.)**

Case 3: Initial evaluation

- A14-year-old soccer player referred for evaluation of elevated blood pressure detected at a pre-sports participation screening at her school.
- Blood pressures obtained at the screening ranged from 137– 149/75–80 mmHg.
- Repeat office BP's are similar to the readings at the sports physical
- She denies any symptoms of hypertension.
- She is at the 50th percentile for height and weight and has no other chronic health problems or abnormal physical examination findings. Both parents have hypertension.

Next step should be:

1. Start hydrochlorothiazide 25 mg daily
2. Refer to IR for arteriogram
3. Perform 24-hr ambulatory BP monitoring
4. Request that the school nurse check her BP daily for the next 10 days

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Further Evaluation

- 24-hr ABPM demonstrates sustained ambulatory hypertension with normal nocturnal dipping
- Urinalysis, electrolytes, BUN and Cr are normal.
- Fasting lipids: total cholesterol 195, LDL cholesterol 90, HDL cholesterol 52, triglycerides 165.

What is your diagnosis?

1. Metabolic Syndrome
2. Primary hypertension
3. Renal artery stenosis
4. Polycystic kidney disease

What is your diagnosis?

1. Metabolic Syndrome
2. Primary hypertension
3. Renal artery stenosis
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Goal for Antihypertensive Treatment in Children

- 19. In children and adolescents diagnosed with HTN, the treatment goal with non-pharmacologic and pharmacologic therapy should be a reduction in SBP and DBP to <90th percentile and <130/80 mm Hg in adolescents ≥ 13 years old.
  - C, moderate

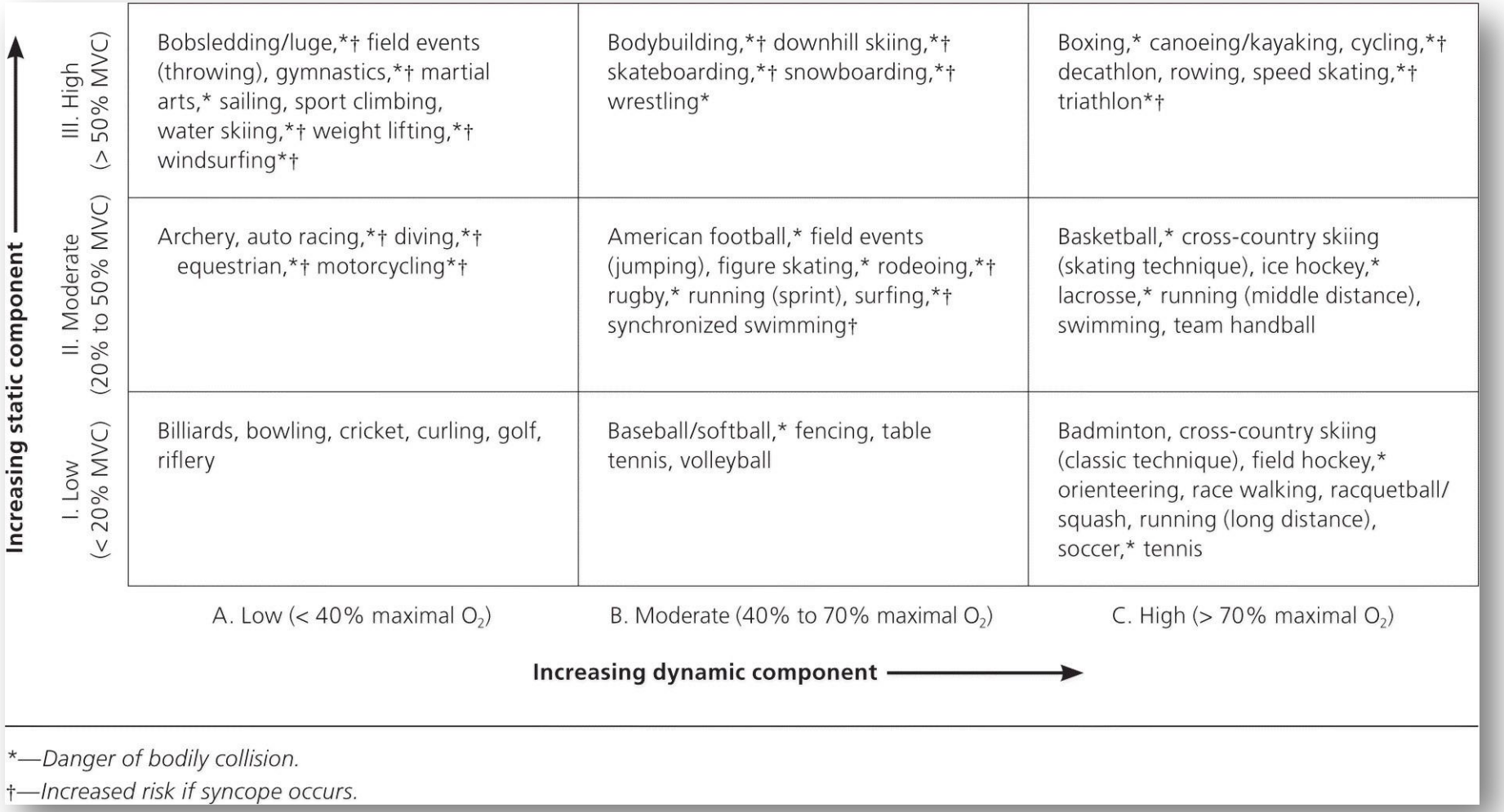
- 23-2. Children or adolescents with both CKD and HTN should be treated to lower 24-hr MAP <50th percentile by ABPM
  - B, strong

Sports Participation and Hypertension

- 28. Children and adolescents with HTN may participate in competitive sports once hypertensive target organ effects and cardiovascular risk have been assessed.
  - C, moderate

- 29. Children and adolescents with HTN should receive treatment to lower BP below stage 2 thresholds before participation in competitive sports.
  - C, moderate

Classification of Various Sports



Increasing static component	A. Low (< 40% maximal O_2)	B. Moderate (40% to 70% maximal O_2)	C. High (> 70% maximal O_2)
III. High (> 50% MVC)	Bobsledding/luge,*† field events (throwing), gymnastics,*† martial arts,* sailing, sport climbing, water skiing,*† weight lifting,*† windsurfing*†	Bodybuilding,*† downhill skiing,*† skateboarding,*† snowboarding,*† wrestling*	Boxing,* canoeing/kayaking, cycling,*† decathlon, rowing, speed skating,*† triathlon*†
II. Moderate (20% to 50% MVC)	Archery, auto racing,*† diving,*† equestrian,*† motorcycling*†	American football,* field events (jumping), figure skating,* rodeoing,*† rugby,* running (sprint), surfing,*† synchronized swimming†	Basketball,* cross-country skiing (skating technique), ice hockey,* lacrosse,* running (middle distance), swimming, team handball
I. Low (< 20% MVC)	Billiards, bowling, cricket, curling, golf, riflery	Baseball/softball,* fencing, table tennis, volleyball	Badminton, cross-country skiing (classic technique), field hockey,* orienteering, race walking, racquetball/ squash, running (long distance), soccer,* tennis

Increasing dynamic component →

*—Danger of bodily collision.

†—Increased risk if syncope occurs.

Mitchell et al, JACC 2005; 45:1366

Case 3: Outcome

- Clinic BP readings remained at stage 2 HTN level
- Allowed to participate in light workouts with team, but restricted from competition
- Echocardiogram done – normal EF, mild concentric LVH
- Started on therapy with amlodipine 5 mg daily
- Dose increase to 10 mg based on home BP readings
- Follow-up clinic BP 132/78
- Allowed to compete in soccer

Resources

- 2017 AAP CPG
  - https://pediatrics.aappublications.org/content/140/3/e20171904.long
- NEJM video on BP measurement
  - https://www.nejm.org/doi/full/10.1056/NEJMvcm0800157
- 2014 AHA Pediatric ABPM statement
  - https://www.ahajournals.org/doi/10.1161/HYP.0000000000000007

Thank you

Resources :

Nelson Textbook of Pediatrics

21st Edition