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Hypertension in children and adolescents

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■ **BACKGROUND** Children have lower blood pressure than adults do, and normal values for children have been established based on age and also on height and weight. Blood pressures in childhood correlate with blood pressures in adulthood, although weakly; a stronger correlation has been established between obesity in childhood and adulthood. Further, obese people are more likely to have high blood pressure than are slender people, both as children and adults. In hypertensive children, the higher the blood pressure and the earlier hypertension appears, the more likely is a secondary cause.

■ **KEY POINTS** Physicians should measure and record children's blood pressure, just as they do their height and weight. An algorithm can help physicians decide whether a child with high blood pressure needs further workup and treatment. Nonpharmacologic therapy includes dietary sodium restriction, weight reduction (if the child is overweight), aerobic exercise, and relaxation. In some cases pharmacologic therapy may be necessary. In general, all children should be encouraged to be physically active and to eat healthy foods.

■ **INDEX TERMS:** HYPERTENSION; CHILD; ADOLESCENT
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HYPERTENSION, usually viewed as a disease of middle age and the elderly, may actually begin much earlier: children at the 90th percentile or above for blood pressure are more likely to have hypertension as adults than are other children. Obesity in children is also worrisome, as overweight children remain so as adults, and overweight people of any age are more likely to have hypertension. Pediatricians and family practitioners are beginning to measure and record the blood pressure of all their young patients at all visits—and to ponder what to do when a reading is high. This article reviews what we currently know about hypertension in children and what experts recommend to do about it.

PREVALENCE AND SIGNIFICANCE

Hypertension in children and adolescents is not uncommon. Sinaiko et al¹ found the prevalence of “significant” hypertension to be 2% in 14 000 school children age 10 to 15; other reported frequencies range from 1.2% to 13%.² Thus, the extent of hypertension in the pediatric

TABLE 1
THRESHOLD HYPERTENSIVE BLOOD PRESSURE
VALUES IN THE YOUNG BY AGE GROUP*

Age	High normal (90th percentile)	Significant hypertension (95th percentile)	Severe hypertension (> 99th percentile)
0–7 days	—	96/—	106/—
8–30 days	—	104/—	110/—
≤ 2 years	104/70	112/74	118/82
3–5 years	108/70	116/76	124/84
6–9 years	114/74	122/78	130/86
10–12 years	122/78	126/82	134/90
13–15 years	130/80	136/86	144/92
16–18 years	136/84	142/92	150/98

*Adapted from the Second Task Force on Blood Pressure Control in Children, reference 3; values are in mm Hg

LONGITUDINAL STUDIES

Children with hypertension risk future cardiovascular, renal, and nervous-system complications. Further, sustained hypertension or even isolated high blood pressure readings may be associated with future risk of hypertension itself, as longitudinal studies suggest that children with high blood pressure will continue to have it as adults.

Lauer and Clarke⁵ observed 2445 subjects who had their blood pressures measured every 2 years between ages 7 through 18 and once between ages 20 and 30. The longitudinal correlation coefficients for systolic blood pressure ranged from .21 to .39, from −.01 to .50 for diastolic blood pressure, and from .45 to .74 for the Quetelet index (a measure of obesity; weight/[height squared]). In addition, the investigators used logistic regression to calculate the risk of future high blood pressure and obesity based on a single childhood observation (Figure 2).

Other studies have confirmed these findings^{6–8}: the correlation coefficients ranged from .2 to .5 for systolic pressure, from .2 to .3 for diastolic pressure, from .4 to .6 for cholesterol concentration, and from .6 to .8 for height and weight. Although blood pressures “track” poorly in childhood, these studies have shown that children with blood pressures less than the 50th percentile have little risk of having pressures in the 90th percentile or higher as young adults.

Gillman et al⁹ found these relatively weak correlations for blood pressure could be increased with repeated testing. The investigators measured the blood pressure of 333 children ages 8 through 15 for 4 successive years, on four weekly visits in each year, with three measurements at each visit; they used the mean blood pressure for each visit to calculate the mean annual blood pressure for each child. The correlation coefficients for systolic blood pressure at the 3-year follow-up for the first, second, third, and fourth weekly visits were .45, .55, .64, and .69, respectively; for diastolic pressure the cor-

relation coefficients for systolic blood pressure ranged from .21 to .39, from −.01 to .50 for diastolic blood pressure, and from .45 to .74 for the Quetelet index (a measure of obesity; weight/[height squared]). In addition, the investigators used logistic regression to calculate the risk of future high blood pressure and obesity based on a single childhood observation (Figure 2).

Age may not tell the whole story, however. Recently, Rosner et al⁴ reanalyzed eight studies cited in the Second Task Force report and one additional study to establish normal values by age, sex, and height. There were 56 108 children, age 1 to 17, seen at 76 018 visits. When height is taken into account, more short children and fewer tall children are classified as hypertensive than when the Task Force criteria are used.

Whatever criteria are used, physicians and nurses should document and graph children's blood pressure, just as they record their height and weight. The American Academy of Pediatrics recommends that blood pressures be routinely measured starting at age 3 years.

It is important that the blood pressure be measured as accurately as possible. As noted by Rosner et al,⁴ accuracy may be influenced by a number of factors, including size of the bladder and cuff, time of day, season of the year, subject position, type and number of observers, arm position, whether the subject is fasting, and whether the fourth or fifth Korotkoff sound is recorded. In infants, for whom the accuracy of measurements by auscultation may be uncertain, the Doppler technique can be used.

Figure 1 outlines the recommended approach to children with documented or suspected hypertension (blood pressure in the 90th percentile or higher).

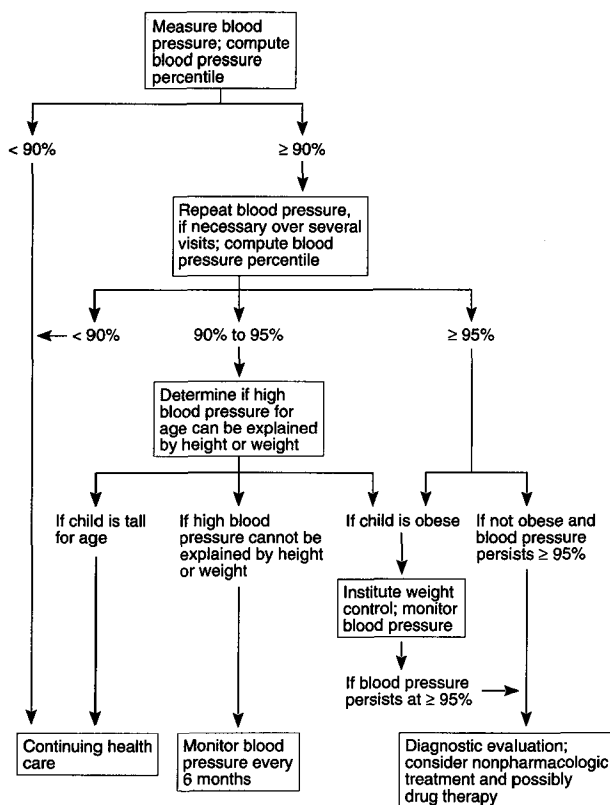


FIGURE 1. Algorithm for detecting and treating hypertension in children. Adapted from the Second Task Force on Blood Pressure Control in Children, reference 3.

responding values were .28, .41, .47, and .54. In another study, Gillman et al¹⁰ measured the blood pressure of 337 children and remeasured it 8 to 12 years later in 317 (94%). They found the correlation coefficient to be .55 for systolic pressure and .44 for diastolic pressure.

Although these correlations from childhood to early adulthood are higher than previously reported, taken together, the data cast doubt on the usefulness of routinely measuring blood pressure to identify children at high risk of developing essential hypertension as adults. Perhaps of more importance is the high longitudinal correlation of the Quetelet index; since hypertension is correlated with obesity in adults, preventing or reducing obesity in childhood may be a useful strategy for preventing hypertension in adults.

Nelson et al¹¹ reported on 221 subjects who had

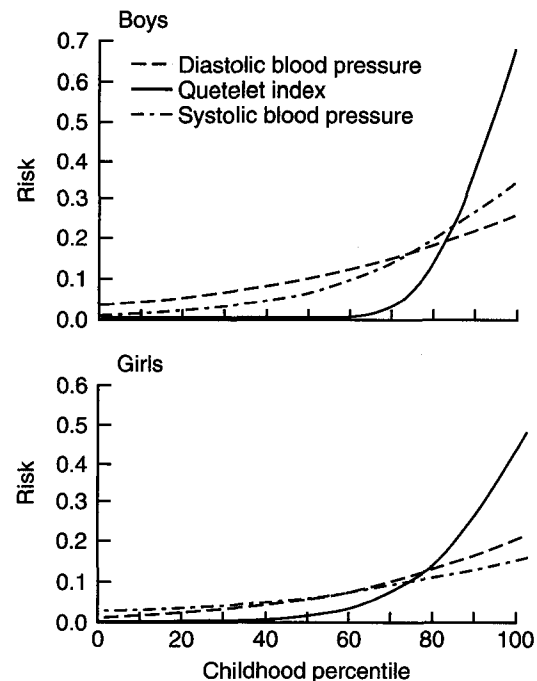


FIGURE 2. The risks of having high blood pressure or being obese (ie, at the 90th percentile or above for systolic pressure, diastolic pressure, or Quetelet index) at ages 20 to 25 based on measurements at age 16 in 2445 subjects. From Lauer and Clarke, reference 5.

their blood pressures measured between ages 3 and 18 years, at age 30, and at age 50. The systolic blood pressure at age 30 was significantly and positively correlated with systolic pressures from age 5 onward in female subjects ($r = .18$ to $.46$) and age 6 onward in male subjects ($r = .22$ to $.42$). The correlation of blood pressure at age 50 was much lower for both sexes. The correlations for diastolic blood pressures were generally weaker than for systolic blood pressures. Height and body mass index were found to be independent predictors of adult blood pressure. The investigators concluded that juvenile blood pressure is one of several predictors of adult blood pressure.

ETIOLOGY

The cause of childhood hypertension is often suggested by the age of the child and by the level of blood pressure—the older the child and the lower the blood pressure, the more likely it is that the hypertension is idiopathic; the younger the child and the higher the blood pressure, the more likely is

TABLE 2
COMMON CAUSES OF HYPERTENSION
IN CHILDHOOD

Age 1 to 6	
Renal parenchymal disease	
Renovascular disease	
Coarctation of the aorta	
Endocrine causes [†]	
Iatrogenic [†]	
Essential hypertension	
Age 6 to 12	
Renal parenchymal disease	
Renovascular disease	
Essential hypertension	
Coarctation of the aorta	
Endocrine causes [†]	
Iatrogenic [†]	
Age 12 to 18	
Essential hypertension	
Iatrogenic	
Renal parenchymal disease	
Renovascular disease [†]	
Endocrine causes [†]	
Coarctation of the aorta	

*From Ingelfinger, reference 15

[†]Substantially less common

a secondary cause for the hypertension (Tables 2 and 3).¹² Of the secondary causes, a renal origin is the most common. It is beyond the scope of this article to explore all of these possibilities. However several recent articles^{13,14} and chapters^{2,15,16} provide excellent reviews of these secondary causes of hypertension.

Genetic factors

Idiopathic hypertension in children is multifactorial. Since hypertension tends to run in families, genetic factors have been implicated. Studies in populations and in twins point to important genetic influences on blood pressure in childhood and adolescence. In studies in twins, it has been estimated that genetic influences account for as much as 82% of the variability in systolic blood pressure, but only 64% of the variability in diastolic blood pressure.¹⁷ Investigation of polygenic pathways led to the study of factors such as ion transport, kallikrein excretion, plasma haptoglobin, and sympathetic reactivity, all of which correlate with blood pressure. Such pathways appear to be under strong genetic influence.^{18,19}

Univariate analysis shows that a significant proportion of the variability of systolic and diastolic blood pressure is under genetic control.²⁰ Multivariate analysis demonstrates that in adolescents, genetic paths shared with body mass index appear to

TABLE 3
SECONDARY CAUSES OF HYPERTENSION

Renal	
Acute glomerulonephritis	
Hemolytic uremic syndrome	
Obstructive uropathy	
Congenital abnormalities (polycystic kidneys, multicystic kidneys, Ask-Upmark kidney)	
Renal arterial disease	
Renal parenchymal disease	
Perirenal masses	
Anaphylactoid purpura	
Renal transplantation	
Acute renal failure	
Renal tumors	
Collagen vascular disease	
Endocrine	
Pheochromocytoma	
Congenital adrenal hyperplasia	
Hyperthyroidism (systolic only)	
17-Hydroxylase deficiency	
Aldosteronism (primary)	
Neuroblastoma	
Cushing's disease	
Hyperparathyroidism	
Vascular system	
Polycythemia	
Anemia (systolic only)	
Takayasu's arteritis	
Patent ductus arteriosus (systolic only)	
Coarctation of the aorta	
Metabolic	
Diabetes mellitus (renal involvement)	
Acute intermittent porphyria	
Hypercalcemia	
Neurologic	
Dysautonomia (Riley-Day syndrome)	
Neurofibromatosis	
Increased intracranial pressure	
Guillain-Barré syndrome	
Anxiety	
Drug-related	
Steroid administration	
Heavy metals	
Amphetamine overdose	
Following sympathomimetic drugs	
Birth control pills	
Miscellaneous	
Essential hypertension	
Burns	
Stevens-Johnson syndrome	
Cyclic vomiting with dehydration	

influence systolic but not diastolic blood pressures.¹⁷ More direct evidence of the involvement of specific genes have been provided by Caulfield et al²¹ and Jeunemaite et al,²² who reported linkage between the angiotensinogen-gene locus on chromosome 1 and essential hypertension. However, these studies found linkages only—they did not identify a causative gene.

Obesity

Obesity is a modifiable risk factor strongly associated with hypertension. Many studies of children and adolescents have demonstrated the association of body size (measured by body mass indices or skin-fold thickness) with blood pressure.¹⁸ As in adults, the distribution of fat also may be important. Shear et al²³ reported that central deposition of body fat may be more strongly related to hypertension than is peripheral body fat. Additional evidence of the role of obesity comes from longitudinal studies, which demonstrate that children who have an increase in relative body size may also have more of an increase in blood pressure than do their peers. Conversely, those who have a decrease in body size rank also have a decrease in blood pressure rank.²⁴ These data, and the correlation of blood pressure in childhood and adulthood, suggest that any discussion of blood pressure control in children should emphasize avoiding obesity.

Cation intake

Excessive sodium intake may contribute to the development of hypertension.^{25,26} In populations that consume little sodium, blood pressure does not rise with age, and hypertension is essentially absent.²⁷ Of note, people in these populations consume extremely little sodium from birth on-

TABLE 4
HINTS TO CAUSE OF HYPERTENSION*

Finding	Possible cause
Habitus	
Thinness	Pheochromocytoma, hyperthyroidism (with proptosis), renal disease (growth failure)
Obesity	Cushing's disease
Virilization	Congenital adrenal hyperplasia
Rickets	Chronic renal disease
Skin	
Café au lait spots	Neurofibromatosis
Tubers	Tuberous sclerosis (also "ash-leaf" spots)
Neurofibromas	Neurofibromatosis
Bruises	Cushing's disease, trauma
Rashes	Butterfly, systemic lupus erythematosus; vasculitis, collagen vascular disease; impetigo, acute nephritis; striae, Cushing's syndrome
Head and face	
Bruit	Arteriovenous malformation
Unusual shape	Arteriovenous malformation, mass lesion
Round (moon) facies	Cushing's syndrome
Elfin facies	William's syndrome
Eyes	
Extraocular muscle palsy	Nonspecific
Fundal changes	Nonspecific
Proptosis	Hyperthyroidism
Neck	
Goiter	Possible hyperthyroid goiter
Lungs	
Rales, rhonchi	Nonspecific, related to acute cardiac decompensation
Heart	
Enlarged	Possible longstanding hypertension, possibly related to acute overload
Failure	Same as for enlarged heart
Rub	Possible chronic renal disease with hypertension
Abdomen	
Masses	Wilms' tumor, neuroblastoma, hydronephrosis, polycystic disease
Hepatomegaly	Heart failure
Hepatosplenomegaly	Infantile polycystic disease
Scars	Genitourinary surgery, possible obstruction
Bruit	Renovascular disease
Back and flank	
Bruit	Renovascular disease
Flank tenderness	Pyelonephritis, obstruction, acute nephritis
Scoliosis	Possible hypertension related to procedures
Pelvis	
Mass	Obstructive uropathy
Genitalia	
Ambiguous, virilized	Congenital adrenal hyperplasia
Extremities	
Blood pressure disparity, pulse disparity, delayed capillary filling in legs	Coarctation
Neurologic	
Seizures, Bell's palsy, irritable	Nonspecific

*Adapted from Ingelfinger J. Evaluation of secondary hypertension. In: Holliday MA, Baratt TM, Avner ED, editors. Pediatric nephrology. 3rd ed. Baltimore: Williams and Wilkins, 1993:1148-1164.

TABLE 5
TREATMENT OF CHILDHOOD HYPERTENSION***Nonpharmacologic treatment**

Weight reduction (if appropriate)
Salt restriction (2 g sodium)
Aerobic exercise (30 minutes, 2 to 3 times per week)
Stress reduction via relaxation techniques or biofeedback

Pharmacologic treatment

Drug	Adult dose range (total mg/day)	Frequency	Pediatric dose (mg/kg/day) [†]
Thiazide diuretics			
Chlorthalidone	12.5–50	Once	0.5–2
Chlorothiazide	125–500	Twice	2.0–5.0
Hydrochlorothiazide	12.5–50	Once	0.5–2
Metolazone	0.5–5	Once	0.1–3
Loop diuretics			
Bumetanide	0.5–5	Twice	0.02–0.3
Furosemide	20.0–320	Twice	1–3
Potassium-sparing diuretics			
Spironolactone	25–100	Once or twice	2–4
Triamterene	50–150	Twice	2–4
Beta blockers			
Atenolol	25–100	Once	1–2
Metoprolol	50–200	Once or twice	1.5–5.0
Propranolol	40–240	Twice	0.5–3
Alpha-receptor blockers			
Prazosin	1.0–20	Twice or thrice	0.065–0.5
Angiotensin-converting enzyme inhibitors			
Captopril	12.5–150	Twice	0.05–0.1
Central alpha₂-agonists			
Guanabenz	4–64	Twice	0.08–0.2
Methyldopa	250–2000	Twice	10–20
Clonidine	0.1–1.2	Twice	0.1–0.8
Direct vasodilators			
Hydralazine	50–300	Twice to four times	0.1–3.0
Minoxidil	2.5–80	Once or twice	0.2

*Adapted from the Task Force on Blood Pressure Control in Children, reference 3, and the 1993 report of the Joint National Committee on Detection, Evaluation and Treatment of High Blood Pressure, reference 12

[†]This table lists only the drugs for which pediatric doses have been established; all antihypertensive drugs have been used in children; pediatric dosages should not exceed adult dosage

ward. Dahl and colleagues²⁸ bred a strain of rat in which even brief exposure to a high-sodium diet very early in life led to permanent increases in blood pressure, even if the high-sodium diet was removed. In a study in humans, Hofman et al²⁹ found that infants exposed to a lower sodium intake from birth had lower blood pressures at 5 to 6 months than did infants exposed to a higher sodium intake.

Although studies in animals have suggested that high potassium intake tends to lower blood pressure, studies in humans have been inconclusive, especially in children.¹⁸

Recently, interest has risen in the relationship between dietary calcium and blood pressure. Inter-

vention trials in adults have demonstrated a small lowering of blood pressure with calcium supplementation.³⁰ There have been few studies in children, except for the report of Gillman et al,³¹ which showed a strong inverse association between dietary calcium and systolic blood pressure in a group of 3- to 5-year-old children.

**PHYSICAL FINDINGS
AND LABORATORY DATA**

Once the diagnosis of hypertension has been made, a pertinent physical examination should be undertaken (Table 4). If a secondary cause is suspected, the laboratory evaluation should be directed toward that cause; if not, it may be useful to obtain a urinalysis, a hemoglobin and hematocrit, and serum concentrations of electrolytes, urea nitrogen, and creatinine. A fasting lipid profile should be obtained to help evaluate cardiovascular risk, and an echocardiogram or electrocardiogram should be obtained to evaluate possible end-organ damage.

TREATMENT**Nonpharmacologic treatment**

Nonpharmacologic therapy includes dietary sodium restriction, weight reduction (if indicated), aerobic exercise, and relaxation.

Blood pressure may be particularly sensitive to salt restriction, which has been said to be of benefit in 25% of hypertensive adolescents.³² Sodium intake should be reduced to no more than 2 g per day. Patients can achieve this goal by avoiding salty food

and not adding salt in cooking or at the table. If this is unpalatable, potassium chloride or low-sodium spices can be used as seasoning. If sodium restriction fails to lower the blood pressure, one should prescribe a diuretic (paying appropriate attention to hypokalemia).

Aerobic exercise can also be effective for adolescents, who should participate in aerobic exercise for at least 30 minutes three to four times a week. Isometric or static exercise to improve strength is not recommended to reduce blood pressure and in fact may increase it. As long as their blood pressure is under control, there is no reason children should not participate in athletic activities. Weight reduction is an important component of blood pressure control in obese adolescents. A weight loss of just 5% has been shown to decrease blood pressure into the normal range.³²

In addition, home monitoring of blood pressure is critical. Since blood pressure is the end point of therapeutic intervention, patients must know what their blood pressure is and measure it daily.

Pharmacologic treatment

Table 5 lists antihypertensive agents for which pediatric doses have been established. Many of them have specific indications, such as angiotensin-con-

verting enzyme (ACE) inhibitors in renovascular hypertension. Others may be more useful because they are taken once a day (eg, some new ACE inhibitors) as opposed to three to four times a day (eg, hydralazine). As recommended by the fifth report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure,¹² all antihypertensive drugs lower blood pressure equally well, but diuretics and beta blockers are more well studied. In general, starting with one drug, increasing the dose if necessary, and then adding another drug would seem a wise policy to follow.

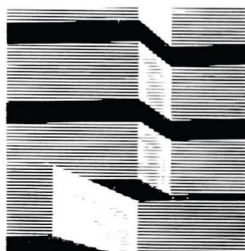
SUMMARY

Childhood hypertension is not uncommon. Children's blood pressure should be monitored routinely at physician visits and compared with normal values. When elevated blood pressures are documented, home blood pressure monitoring is indicated. If therapy is indicated, nonpharmacologic therapies such as weight reduction, aerobic exercise, and salt restriction should be encouraged. If pharmacologic therapy is needed, a single agent with which the physician is familiar should be tried and then other drugs added if necessary, while home blood pressure monitoring continues.

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HIGHLIGHTS FROM MEDICAL GRAND ROUNDS

*Concise, current,
critical information
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