Epidemiology, risk factors, and etiology of hypertension in children and adolescents

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Disclosures
All topics are updated as new evidence becomes available and our peer review process is complete.


INTRODUCTION — It has become clear that hypertension (HTN) begins in childhood and adolescence and that it contributes to the early development of cardiovascular disease (CVD). The supporting data include clinical studies that demonstrate cardiovascular structural and functional changes in children with HTN and autopsy studies that have shown an association of blood pressure (BP) with atherosclerotic changes in the aorta and heart in children and young adults. (See "Definition and diagnosis of hypertension in children and adolescents" and "Risk factors and development of atherosclerosis in childhood", section on 'Hypertension'.)

In hypertensive adults, multiple randomized trials have shown that reduction of BP by antihypertensive therapy reduces cardiovascular morbidity and mortality. The magnitude of the benefit increases with the severity of the HTN. (See "Hypertension: Who should be treated?").

Based upon these observations, identifying children with HTN and successfully treating their HTN should have an important impact on long-term outcomes of CVD.

The epidemiology, risk factors, and etiology of childhood HTN will be reviewed here. The diagnosis, evaluation, and treatment of HTN in children and adolescents are reviewed separately. (See "Definition and diagnosis of hypertension in children and adolescents," and "Evaluation of hypertension in children and adolescents" and "Treatment of hypertension in children and adolescents".)

Neonatal HTN, including etiology, is also discussed separately. (See "Etiology, clinical features, and diagnosis of neonatal hypertension".)

DEFINITION — In children, the following definitions based upon the 2004 National High Blood Pressure Education Program Working Group (NHBPEP) are used to classify blood pressure (BP) measurements in the United States [1]. BP percentiles are based upon gender, age, and height (table 1 and table 2). The systolic and diastolic BPs are of equal importance; if there is a disparity between the two, the higher value determines the BP category. The age- and height-specific blood pressure percentiles may be determined using calculators for boys (calculator 1) or for girls (calculator 2). (See "Definition and diagnosis of hypertension in children and adolescents").
• Normal BP – Both systolic and diastolic BP <90th percentile.
• Prehypertension – Systolic and/or diastolic BP ≥90th percentile but <95th percentile or if BP exceeds 120/80 mmHg (even if <90th percentile for age, gender, and height).
• Hypertension – Hypertension (HTN) is defined as either systolic and/or diastolic BP ≥95th percentile measured on three or more separate occasions. The degree of HTN is further delineated by the following two stages.
  • Stage 1 HTN – Systolic and/or diastolic BP between the 95th percentile and 5 mmHg above the 99th percentile.
  • Stage 2 HTN – Systolic and/or diastolic BP ≥99th percentile plus 5 mmHg.

EPIDEMIOLOGY

Hypertension

United States — In the United States, the prevalence of elevated blood pressure (BP) in children appears to be increasing. This was illustrated in a study that analyzed population-based data from the National Health and Nutrition Examination Survey (NHANES) III from 1988 to 1994 and from the continuous NHANES 1999 to 2008 [2]. The prevalence of elevated BP (defined as either a systolic or diastolic BP ≥90th percentile or ≥120/80) increased from NHANES III to NHANES 1999 to 2008 in both boys (15.8 to 19.2 percent) and girls (8.2 to 12.6 percent). After adjusting for confounding variables, the increase in elevated BP was independently associated with increases in body mass index, waist circumference, and sodium intake.

In contrast, an earlier study using data from the Bogalusa Heart Study showed that, from 1974 to 1993, mean systolic blood pressure (SBP) levels remained the same and mean diastolic blood pressure (DBP) decreased by 2 mmHg, while the rates of obesity increased from 6 to 17 percent [3]. (See ‘Obesity’ below.)

In a study of a large cohort of children of diverse race and ethnicity cared for in community-based practices from 2007 to 2009, the reported prevalence of hypertension based upon confirmed persistent BP ≥95th percentile was 0.3 percent [4]. In this group of children, risk factors for hypertension included increasing body mass index (BMI) and age. Children who were black or Asian also were more likely to be at risk for hypertension compared with other ethnic groups.

Other countries — It is unclear whether childhood BP is increasing worldwide. In the United Kingdom, mean SBP has increased in children between 9 and 11 years of age from 1980 to 2008 [5]. This increase was not explained by increased BMI. In contrast, an earlier report from Northern Ireland demonstrated a decrease in mean systolic and diastolic BPs over the 10 years from 1989 to 1999 [6].

In Switzerland, the prevalence of hypertension (HTN) in sixth-grade children after three repeated BP measurements was 2.2 percent [7]. HTN was more likely in children with excess body weight, increased heart rate, and a parental history of HTN. In this cohort, 16 percent of the boys and 12 percent of the girls were overweight or obese.
**Prehypertension** — Globally, the reported prevalence of prehypertension ranges from 9.5 to 24 percent [8-12]. In a study of 1010 adolescents who underwent BP measurements during three separate clinical encounters, about 20 percent of individuals had BP in the range of prehypertension at the first visit, 4 percent had persistent prehypertension defined as BP >90\(^{\text{th}}\) percentile or 120/88 mmHg at all three visits, and 30 percent had a BP in the prehypertensive range during one of the three encounters [12]. In addition, 2.5 percent were hypertensive (BP >95\(^{\text{th}}\) percentile) at all three visits. In this cohort, obesity was a risk factor for both prehypertension and HTN.

**RISK FACTORS** — The incidence of hypertension (HTN) in children may vary according to the patient's body mass index (BMI), gender, ethnicity, and family history.

**Obesity** — In the United States and Canada, the rising risk of HTN is in part due to the increasing prevalence of obesity, as demonstrated by the following observations in school-aged children:

- In a report based upon the United States National Health Survey data, the risk of high blood pressure (BP) (defined as greater than the 95\(^{\text{th}}\) percentile) doubled for every one unit of increase in the BMI z-score unit [13].
- In a longitudinal adolescent Canadian cohort study, biannual blood pressure and anthropometric measurements were performed in 1267 patients who were in grades 7, 9, and 11 from 1999 to 2005 [14]. Multivariate analysis demonstrated that the risk of high systolic blood pressure (SBP) (defined as greater than the 90\(^{\text{th}}\) percentile) was more than a twofold increase in overweight children (defined as a BMI >85\(^{\text{th}}\) percentile) compared with nonoverweight children (OR 2.63, 95% CI 1.75-3.92).
- A similar result was noted in a study of 1111 adolescents of a marked increase in the prevalence of high blood pressure in individuals whose BMI exceeded the 85\(^{\text{th}}\) percentile [15].
- In a study of predominantly minority 8\(^{\text{th}}\) grade adolescents, the risk of HTN increased with rising BMI (6.9 versus 24.6 percent among those with BMI <85\(^{\text{th}}\) and ≥95\(^{\text{th}}\) percentile, respectively) [16]. In addition, the risk of dyslipidemia increased with rising blood pressure and BMI. As an example, the prevalence of high triglycerides was 14.7 and 27.1 in normotensive versus hypertensive students, respectively.
- In the previously mentioned study comparing the National Health and Nutrition Examination Survey (NHANES) III with NHANES 1999 to 2008, increasing body mass index and waist circumference were associated with an increased prevalence of elevated BP between the two periods of time [2].

The relationship between elevated BP and weight begins in early childhood, as illustrated by the following studies:

- In a retrospective review of primary care visits of 18,618 children between 2 and 19 years of age, systolic and diastolic BP increased with increasing BMI in all age groups including children between two and five years of age [17].
In a second study, SBP and weight-for-length were measured in 530 children from birth to three years of age [18]. An increase in the weight-for-length in the first six months of life was associated with higher systolic blood pressure at three years of life, particularly among infants who were thin at birth.

In contrast, a report from the Bogalusa Heart study showed that mean systolic and diastolic BP levels did not increase despite a rise in the prevalence of obesity from 6 to 17 percent over the study period from 1974 to 1993 [3]. Nevertheless, within this cohort of 11,478 children, there was a positive correlation of BMI and BP levels. However, these results suggest that other factors may have ameliorated the expected increase in BP due to the increasing prevalence of obesity. (See "Definition; epidemiology; and etiology of obesity in children and adolescents" and "Comorbidities and complications of obesity in children and adolescents").

Gender — In the United States and Canada, the prevalence of HTN and prehypertension is greater in boys than girls [13,14]. As an example, in the previously mentioned adolescent Canadian cohort study, boys were more likely to have high systolic BP (>90th percentile) compared with girls in 7th (OR 1.29, 95% CI 0.77-2.16), 9th (OR 1.98, 95% CI 1.35-2.93), and 11th grades (OR 2.74, 95% CI 1.52-4.94).

Ethnicity — NHANES III showed that the incidence of HTN in adults was affected by race and ethnicity, being most common in non-Hispanic African Americans (figure 1A-B) [19]. The age-adjusted prevalence of HTN was 32 percent in the non-Hispanic African American population compared with 23 percent in the non-Hispanic white and Mexican American populations. African Americans also are more likely to develop hypertensive complications, particularly renal disease. In blacks, the relative importance of environmental and genetic factors has not been established [20]. (See "Hypertensive complications in blacks").

In children, data also suggest that the risk of HTN is greater in minority ethnic groups [13,21-24].

In the previously discussed report based upon the United States National Health Survey data, the prevalence of high BP (greater than the 95th percentile) was 4.6, 4.2, and 3.3 in Mexican American, black, and white children, respectively, during the study period of 1999 to 2002 [13].

In a study of 2368 girls enrolled at the age of 9 or 10 years in the National Heart, Lung, and Blood Institute Growth and Health Study from 1986 to 1997, the overall incidence of HTN based on two clinic visits was greater in African American girls compared with Caucasian girls (5 versus 2.1 percent) [24]. The incidence increased to 10.5 and 3.8 percent in African American and Caucasian girls whose BMI was greater than the 95th percentile and was lower in those with normal BMI (<85th percentile, 3.5 versus 1.7 percent). Similar patterns were seen for prehypertension with incidences of 4.1 and 2.4 in African American and Caucasian girls, 9.7 and 4.6 percent in obese African American and Caucasian girls, and 2.2 and 2.0 percent in African American and Caucasian girls with normal BMI levels. In the entire cohort,
multivariate analyses adjusted for age and race demonstrated higher BMI and lower potassium intake were associated with an increase risk of HTN.

**Genetics** — A family history of HTN is present in as many as 70 to 80 percent of all patients with primary HTN (also referred to as primary HTN), which has no identifiable underlying etiology, and in approximately 50 percent of hypertensive children [25].

In patients with primary HTN, elevated BP is thought to result from the interaction of multiple genes and environmental factors. It has been estimated that genetic factors account for approximately 30 percent of the variation in BP in various populations [26,27] and as much as 60 to 70 percent of HTN in families [28]. (See "Genetic factors in the pathogenesis of primary (essential) hypertension".)

The best evidence for genetic factors influencing BP values comes from BP correlations within families:

- The BP correlation is stronger between parents and children than between spouses [29].
- There is no significant BP correlation between parents and adopted children [30,31].
- Most studies in twins have shown BP correlation is stronger between identical (monozygotic) twins than between fraternal (dizygotic) twins or siblings [29,32]. However, the observation that the BP correlation is stronger among dizygotic twins than other first-degree relatives indicates a nongenetic environmental effect [33].

In both animal and human studies, a number of different genes have been evaluated, but their role remains uncertain in the pathogenesis of primary HTN. (See "Genetic factors in the pathogenesis of primary (essential) hypertension".)

**Monogenic disorders** — Rarely, monogenic disorders can cause HTN and include the following:

- Liddle's syndrome (MIM #177220), due to a gain-of-function mutation in the sodium channel gene, is associated with hypertension, low plasma renin and aldosterone levels, and hypokalemia. (See "Genetic disorders of the collecting tubule sodium channel: Liddle's syndrome and pseudohypoaldosteronism type 1", section on 'Liddle's syndrome'.)
- Pseudohypoaldosteronism type 2 or Gordon's syndrome (MIM #145260) is due to mutations in WNK kinases 1 and 4, which result in increased chloride reabsorption with sodium. It is characterized by HTN, hyperkalemia, normal renal function, and low or low-normal plasma renin activity and aldosterone. (See "Etiology, diagnosis, and treatment of hypoaldosteronism (type 4 RTA)", section on 'Pseudohypoaldosteronism type 2 (Gordon's syndrome)'.)
- Glucocorticoid-remediable aldosteronism, also known as familial hyperaldosteronism type I, is a disorder in which there is a chimeric gene formed from portions of the 11ß-
hydroxylase gene and the aldosterone synthase gene, which results in ACTH stimulating aldosterone synthesis. (See "Familial hyperaldosteronism".)

- Congenital adrenal hyperplasia due to 11ß-hydroxylase deficiency is a disorder that has been associated with multiple mutations of the CYP11B1 gene. (See "Congenital adrenal hyperplasia due to 11-beta-hydroxylase deficiency".)

- Syndrome of apparent mineralocorticoid excess (MIM #218030) arises from mutations in the gene encoding the enzyme 11ß-hydroxysteroid dehydrogenase. The defective enzyme allows normal circulating concentrations of cortisol (which are much higher than those of aldosterone) to activate the renal mineralocorticoid receptors resulting in increased sodium absorption. (See "Apparent mineralocorticoid excess syndromes (including chronic licorice ingestion)."

Breastfeeding — There is evidence that breastfeeding may be associated with lower BPs in childhood [34-36]. This was illustrated in two prospective cohort studies.

- In the first study, 7276 infants were evaluated at 7.5 years of age [34]. Those who were breastfed as infants had systolic and diastolic BPs that were 1.2 mmHg and 0.9 mmHg lower than in infants who were never breastfed [34]. The reduction in both systolic and diastolic pressures was greater in infants who were exclusively breastfed, and the systolic BP reduction increased with the duration of breastfeeding.

- Similar findings were found in 7223 singleton infants evaluated at five years of age [36]. Those who were breastfed for six months had systolic BP that was 1.2 mmHg lower than in infants who had been breastfed less than six months or not at all [36].

White coat hypertension — White coat or isolated office HTN is defined as office BP readings ≥95th percentile but with normal values outside the clinical setting. Studies suggest a prevalence as high as 35 percent in children being evaluated for persistently elevated casual BP [37] and approximately 44 percent in children with a positive family history of primary HTN [38]. The prevalence of white coat HTN is higher when the office values reveal borderline or mild HTN, and much lower with moderate or severe HTN [39].

It is possible that white coat HTN in children represents two populations: one that is destined to develop primary HTN (prehypertensive) [40] and one that remains normotensive outside the clinical setting. In adults, white coat HTN appears to be a prehypertensive condition with increased left ventricular mass and progression to sustained HTN. Similar findings were noted in a retrospective study of children with white coat HTN from a tertiary center [41]. About two-thirds of children with white coat HTN had either left ventricular hypertrophy (LVH) detected by echocardiography or systolic BP greater than the 95th percentile for age, height, and gender during treadmill exercise. These observations have led to the concern that patients with white coat HTN are at risk for developing HTN and end-organ cardiac damage. (See "Ambulatory blood pressure monitoring and white coat hypertension in adults", section on 'White coat hypertension' and "Ambulatory blood pressure monitoring in children", section on 'White coat hypertension'.)
Prenatal and neonatal factors — There is increasing evidence that prenatal and neonatal factors contribute to higher BP. There are data demonstrating a role for low birth weight in the development of primary HTN (discussed in greater detail separately). (See "Possible role of low birth weight in the pathogenesis of primary (essential) hypertension").

In addition, a systematic review reported in utero exposure to preeclampsia was associated with an increase in systolic (mean 2.4 mmHg) and diastolic (mean 1.4 mmHg) BP, as well as an increase in BMI (mean 0.62 kg/m²) [42].

ETIOLOGY — Causes of childhood hypertension (HTN) are separated into two classes:

- Primary HTN – No underlying cause is identified.
- Secondary HTN – An identifiable cause is determined. In children with secondary HTN, the underlying disorder may be curable with complete resolution of HTN.

Primary hypertension — Primary HTN is the most common cause of HTN in older children and is a diagnosis of exclusion. It is more likely in children who are postpubertal, have a family history of HTN, are overweight or obese, or have only mild hypertension (blood pressure [BP] at or just above the 95th percentile). It is also more common in African Americans. (See ‘Risk factors’ above.)

Secondary hypertension — There are a number of causes of secondary HTN (table 3), and specific symptoms (table 4) and findings (table 5) may point to a particular disorder. In a Polish review, 351 of 636 children (55 percent) with sustained HTN had a known secondary cause [43]. The most common were renal disease (68 percent) and endocrine and renovascular diseases (11 and 10 percent, respectively). Almost all children (98 percent) younger than 15 years of age had a secondary cause, whereas 75 percent of adolescents had primary HTN.

In another observational series, 89 of 132 children (67 percent) with HTN had renal or renovascular disease, 30 (23 percent) had primary HTN, and 13 (10 percent) had nonrenal causes [44]. Glomerulonephritis and reflux nephropathy were the most common renal cause of HTN in these patients.

The following sections review different etiologies of chronic (persistent) secondary HTN.

Renal parenchymal disease — A variety of intrinsic renal disorders is associated with HTN and includes the following:

- Glomerulonephritis – HTN is a manifestation of both chronic and acute glomerulonephritis (GN). In children, the most common form of acute GN is poststreptococcal GN, which follows after a streptococcal infection. Henoch-Schönlein purpura (IgA vasculitis) can present with renal manifestations including HTN. In children, chronic glomerular disorders associated with elevated BP include IgA nephropathy, membranoproliferative GN, or lupus nephritis. (See "Poststreptococcal glomerulonephritis in children" and "Clinical presentation and diagnosis of IgA nephropathy" and "Diagnosis and classification of renal disease in systemic lupus..."
In children with glomerulonephritis, the most common mechanisms of HTN are volume expansion (as in acute poststreptococcal glomerulonephritis) and activation of the renin-angiotensin system. Other common presenting features of glomerular disorders include hematuria, oliguria, peripheral edema, and an elevated serum creatinine or BUN. (See "Overview of hypertension in acute and chronic kidney disease".)

- Renal parenchymal scarring can be a sequelae of acute pyelonephritis and may be associated with vesicoureteral reflux. It is also seen in children with congenital anomalies of the kidney and urinary tract, and those with irreversible renal injury from hemolytic uremic syndrome. (See "Presentation, diagnosis, and clinical course of vesicoureteral reflux" and "Treatment and prognosis of Shiga toxin-producing Escherichia coli (STEC) hemolytic uremic syndrome (HUS) in children", section on 'Hypertension' and "Acute management, imaging, and prognosis of urinary tract infections in infants and children older than one month", section on 'Prognosis'.)

- Polycystic kidney disease – Polycystic kidney disease (PKD) is due to two genetic disorders, autosomal dominant and autosomal recessive PKD, that involve the formation of renal cysts. HTN is a common presenting sign in children with the recessive form of PKD. (See "Autosomal recessive polycystic kidney disease in children".)

- Chronic renal failure – Chronic renal failure (CRF) of any cause can be associated with HTN because of volume expansion. In addition, children who have undergone renal transplantation are at increased risk for HTN due to several different mechanisms including rejection or the administration of drugs that increase BP. A functional gene MYH9 that is expressed in kidney podocytes has been associated with nondiabetic end-stage kidney disease in African Americans [45]. (See "Overview of the management of chronic kidney disease in children", section on 'Hypertension' and "Complications of renal transplantation in children", section on 'Hypertension' and 'Drugs and toxins' below.)

Renovascular disease — HTN due to renovascular disease is due to a decrease in renal blood flow resulting in increased plasma levels of renin, angiotensin, and aldosterone. Children with renovascular disease generally have stage 2 HTN [46]. (See 'Definition' above.)

Causes of renovascular disease in children include the following:

- Fibromuscular dysplasia – Fibromuscular dysplasia is the most common etiology of renovascular disease [47,48]. It is characterized by arterial stenosis due to a noninflammatory, nonatherosclerotic process. (See "Clinical manifestations and diagnosis of fibromuscular dysplasia".)
- Umbilical arterial catheterization – During the newborn period, catheterization of the umbilical artery may lead to a clot in the renal artery resulting in renal arterial injury and stenosis.

- Other causes of renovascular disease include neurofibromatosis, arteritis, renal artery hypoplasia, and midaortic syndrome (segmental narrowing of the proximal abdominal aorta) [47-49]. (See "Neurofibromatosis type 1 (NF1): Pathogenesis, clinical features, and diagnosis" and "Clinical features and diagnosis of Takayasu arteritis" and "Clinical manifestations and diagnosis of polyarteritis nodosa").

Renal tubular disease — Monogenic diseases in which tubular reabsorption of sodium or chloride is increased are associated with increased vascular volume and HTN, such as Liddle's syndrome, Type 1 pseudohypoaldosteronism, Type 2 pseudohypoaldosteronism, or Gordon's syndrome. (See 'Monogenic disorders' above.)

Endocrinologic disease — Endocrinologic conditions associated with HTN include the following:

- Catecholamine excess – Catecholamine excess that results in HTN occurs in patients with pheochromocytoma and neuroblastoma, and those who use sympathomimetic drugs including phenylpropanolamine (over-the-counter decongestant), cocaine, amphetamines, phencyclidine, epinephrine, phenylephrine, and terbutaline, and the combination of a monoamine oxidase (MAO) inhibitor plus ingestion of tyramine-containing foods. (See "Pheochromocytoma in children" and "Clinical presentation, diagnosis, and staging evaluation of neuroblastoma").

- Corticosteroid excess – Corticosteroid excess is more commonly due to exogenous administration of glucocorticoids and rarely due to endogenous production of either glucocorticoids or mineralocorticoids. In both settings, corticosteroid excess results in HTN.

  - Corticosteroid excess may be seen in patients with Cushing's syndrome due to hypersecretion of adrenocorticotropic hormone (ACTH). (See "Causes and pathophysiology of Cushing's syndrome").

  - Mineralocorticoid excess that result in HTN may be seen in patients with congenital adrenal hyperplasia. Other rare causes of HTN due to mineralocorticoid excess include aldosterone-secreting tumors and the monogenic disorder of glucocorticoid-remediable aldosteronism. (See "Causes and clinical manifestations of primary adrenal insufficiency in children", section on 'Congenital adrenal hyperplasia' and "Familial hyperaldosteronism").

- Other endocrinologic disorders – Other endocrinologic abnormalities associated with HTN include thyroid disorders (hypothyroidism and hyperthyroidism), and hypercalcemia (eg, hyperparathyroidism). (See "Clinical manifestations of hypothyroidism", section on 'Cardiovascular system' and "Cardiovascular effects of
Cardiac disease — Coarctation of the aorta is the primary cardiac cause of HTN. The classic findings are HTN in the upper extremities, diminished or delayed femoral pulses, and low or unobtainable arterial blood pressure in the lower extremities. The diagnosis is confirmed by echocardiogram. (See “Clinical manifestations and diagnosis of coarctation of the aorta”.)

Drugs and toxins — A variety of drugs and toxins can cause chronic HTN including the following:

- Glucocorticoids (see "Major side effects of systemic glucocorticoids", section on 'Kidney and systemic hemodynamics')
- Oral contraceptives (see "Effect of oral contraceptives and postmenopausal hormone therapy on blood pressure")
- Arsenic (see "Arsenic exposure and poisoning", section on 'Cardiovascular')
- Cyclosporine and tacrolimus (see "Pharmacology and side effects of cyclosporine and tacrolimus", section on 'Hypertension')

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- Beyond the Basics topics (see "Patient information: High blood pressure in children (Beyond the Basics)" and "Patient information: High blood pressure treatment in children (Beyond the Basics)"

SUMMARY

- Hypertension (HTN) in childhood and adolescence contributes to premature atherosclerosis and the early development of cardiovascular disease (CVD). (See ‘Introduction’ above and "Risk factors and development of atherosclerosis in childhood", section on 'Hypertension'.)
- Since the late 1980s, the prevalence of childhood HTN has increased in the United States. This rise in blood pressure (BP) is associated with a concurrent increase in the prevalence of obesity.
- Children who are overweight (greater than the 85th percentile for weight) are more likely to have higher BP values (adjusted for age, gender, and height) and be hypertensive. (See 'Obesity' above.)
- The prevalence of HTN and prehypertension is greater in boys than girls. (See 'Gender' above.)
- In the United States, Mexican American and non-Hispanic black children compared with non-Hispanic white children are more likely to have elevated BP adjusted for age, gender, and height. (See 'Ethnicity' above.)
- The etiology of chronic HTN is divided into two categories: primary HTN, in which no underlying cause is identified, and secondary HTN, in which an underlying cause is identified. (See 'Etiology' above.)
- A family history of HTN is present in as many as 70 to 80 percent of all patients with primary HTN and in approximately 50 percent of hypertensive children. In patients with primary HTN, elevated BP is thought to result from the interaction of multiple genes and environmental factors. Rarely, monogenic disorders, such as glucocorticoid-remediable aldosteronism or Liddle's syndrome can cause HTN. (See 'Genetics' above.)
- White coat HTN (defined as BP values ≥95th percentile in a clinical setting but with normal values outside the clinical setting) is a frequent finding in children. Although white coat HTN appears to be a prehypertensive condition in adults, it remains uncertain whether white coat HTN is a marker for childhood HTN. (See 'White coat hypertension' above.)
- The most common condition resulting in secondary HTN is renal disease (68 percent), followed by endocrine and renovascular diseases (11 and 10 percent, respectively). (See 'Secondary hypertension' above.)