Essential Hypertension

(See also *Harrison's Principles of Internal Medicine*, 17th Edition, Chapter 241)

Definition

- Chronic elevation in blood pressure (BP) >140/90 mmHg with no definable cause
  - Represents 90–95% of hypertensive persons
  - The remainder have identifiable causes (secondary hypertension).
- Importance of hypertension relates to increased risks of heart attack, heart failure, stroke, and kidney disease.
  - Each increase of 20 mmHg in systolic BP (SBP) or 10 mmHg in diastolic BP (DBP) doubles the risk of cardiovascular disease across the entire BP range from 115/75 mmHg to 185/115 mmHg.
- Labile hypertension
  - Arterial pressures are sometimes but not always in the hypertensive range.
- Accelerated hypertension
  - Significant recent increase over previous hypertensive levels
  - Frequently associated with evidence of vascular damage on funduscopic examination
- White coat hypertension
  - BP measured in the office by a professional that is persistently higher than when measured at home or under casual circumstances
- Isolated systolic hypertension
  - The predominant form of hypertension after 50 years of age
  - Caused by arterial stiffness
- For discussion of special situations in hypertension, see:
  - Hypertension in African Americans
  - Hypertension in Diabetes Mellitus
  - Hypertension in the Elderly
  - Hypertension Secondary to Chronic Kidney Disease
  - Renovascular Hypertension
  - Hypertensive Emergencies

Epidemiology

- Prevalence
  - 50–65 million persons in the U.S. have high BP.
  - >1 billion persons are affected worldwide
  - Underdiagnosed/undertreated in one third of cases
- Race
  - Prevalence in African Americans: 36%
  - Prevalence in white persons: 23%
- Sex
  - Ratio of women versus men with hypertension increases from 0.6 at 30 years of age to 1.2 at 65 years.

- Age
  - The prevalence of hypertension increases with age.
    - 5% at 20 years of age
    - >50% of persons 60–69 years of age
    - 75% of persons ≥70 years of age

Risk Factors

- Family history of hypertension
- Advanced age
- African-American race
- Obesity
- Inactivity
- Cigarette smoking
- Excessive salt intake
- Excessive alcohol intake

Etiology

- By definition, essential hypertension has no identifiable cause.

Factors that appear to influence development of essential hypertension

Salt sensitivity

- BP is particularly responsive to the level of sodium intake in ~60% of hypertensive persons.

Calcium

- Low calcium intake has been associated with an increase in BP in epidemiologic studies.
- Calcium-channel blockers are effective antihypertensive agents.

Renin

- Low-renin essential hypertension
  - Approximately 20% of patients who have essential hypertension have suppressed plasma renin activity.
  - Clinical features include salt-sensitivity of BP and diuretic responsiveness.
- Nonmodulating essential hypertension
  - Make up 25–30% of the hypertensive population
  - Hypertension is salt-sensitive because of a defect in the kidney's ability to excrete sodium appropriately.
  - Sodium intake does not modulate adrenal or renal vascular responses to angiotensin II.
  - Pathophysiologic characteristics can be corrected by the administration of an angiotensin-converting enzyme (ACE) inhibitor.
- High-renin essential hypertension
  - Approximately 15% of patients with essential hypertension have plasma renin activity levels above the normal range.
  - Elevation of the renin level may have a primary effect on elevating BP or may be secondary to an increase in adrenergic system activity.

- Insulin resistance and/or hyperinsulinemia
  - Hyperinsulinemia can increase arterial pressure by ≥1 of 4 mechanisms.
    - Renal sodium retention (at least acutely) and increased sympathetic activity
    - Vascular smooth-muscle hypertrophy secondary to the mitogenic action of insulin
    - Ion transport changes across the cell membrane, potentially increasing cytosolic calcium levels of insulin-sensitive vascular or renal tissues
    - A marker for another pathologic process, e.g., nonmodulation, which could be the primary mechanism increasing BP
  - The role of insulin as a pathogenic factor in hypertension remains unclear.
- Genes responsible for 3 distinct but rare monogenic hypertensive syndromes have been identified.
  - Glucocorticoid-remediable hypertension
    - A chimeric gene containing the promoter of the 11β-hydroxylase gene and the coding sequence for the aldosterone synthase gene causes ectopic production of aldosterone.
    - Characteristics include early-onset hypertension, with increased frequency of strokes and evidence of hyperaldosteronism.
  - Liddle's syndrome
    - Mutations in the epithelial amiloride-sensitive sodium channel located in the collecting cortical tubule are responsible for sodium retention.
    - Patients have hypertension and hypokalemia, with suppressed plasma renin activity and low plasma aldosterone levels.
  - Syndrome of apparent mineralocorticoid excess
    - Caused by a defect in renal 11β-hydroxysteroid dehydrogenase
    - Protective conversion of cortisol to the inactive cortisone does not occur, and local cortisol binds to the renal mineralocorticoid receptor.

Associated Conditions

- Metabolic syndrome
- Diabetes

Symptoms & Signs

History

- Most patients are asymptomatic.
- Symptoms related to elevated BP
  - Headache
    - Characteristic of only severe hypertension
    - Most commonly localized to the occipital region and present when the patient awakens in the morning, subsiding spontaneously after several hours
  - Dizziness
  - Palpitations
  - Easy fatigability
  - Epistaxis
- Symptoms referable to vascular disease
  - Hematuria
  - Blurring of vision owing to retinal changes
  - Episodes of weakness
  - Dizziness due to transient cerebral ischemia

    - Angina pectoris
    - Dyspnea due to cardiac failure
    - Pain due to dissection of the aorta or to a leaking aneurysm
    - Impotence
- Symptoms suggesting secondary hypertension
    - Polyuria, polydipsia, and muscle weakness secondary to hypokalemia in patients with primary aldosteronism
    - Weight gain and emotional lability in patients with Cushing's syndrome
    - Episodic headaches, palpitations, diaphoresis, and postural dizziness in patients with a pheochromocytoma

Physical examination

- Measurement of BP
    - Patients should be seated quietly for at least 5 minutes in a chair (rather than on an examination table), with feet on the floor and arm supported at heart level.
    - Measurement of BP in the standing position is indicated periodically, especially in those at risk for postural hypotension.
        - An increase in DBP when the patient goes from the supine to the standing position is most compatible with essential hypertension.
        - A fall, in the absence of antihypertensive medications, suggests secondary forms of hypertension.
    - An appropriate-sized cuff (cuff bladder encircling at least 80% of the arm) should be used to ensure accuracy.
    - At least 2 measurements should be made.
    - SBP is the point at which the first sound is heard.
    - DBP is the point just before the disappearance of sounds.
- General appearance
    - Round face and truncal obesity suggest Cushing's syndrome.
    - Muscular development in the upper extremities out of proportion to that in the lower extremities suggests coarctation of the aorta.
- Funduscopic examination
    - Provides one of the best indications of the duration of hypertension and of prognosis
    - Keith-Wagener-Barker classification of funduscopic changes
        - Normal through grade IV retinopathy is based on the presence of arteriolar light reflex, arteriovenous crossing defects, hemorrhages, exudates, and papilledema.
        - Specific changes in each fundus should be recorded
- Chest/heart examination
    - Is there a left ventricular lift? Its presence suggests left ventricular hypertrophy (LVH).
    - Are third and fourth heart sounds present? Suggest left ventricular dilatation and LVH
    - Are there pulmonary rales?
    - A third heart sound and pulmonary rales are unusual in uncomplicated hypertension; their presence suggests ventricular dysfunction.
    - Chest examination also includes a search for extracardiac murmurs and palpable collateral vessels that may result from coarctation of the aorta.
- Abdomen
    - Bruits due to renal arterial stenosis nearly always have a diastolic component or may be continuous and are best heard just to the right or left of the midline above the umbilicus or in the flanks

    - Palpation for an abdominal aneurysm and for the enlarged kidneys of polycystic renal disease
- Vascular
    - Palpation and auscultation of the carotid arteries, for evidence of stenosis or occlusion
    - Palpation of femoral pulses
        - If decreased and/or delayed in comparison with the radial pulse, the BP in the lower extremities should be measured

Differential Diagnosis

- Secondary hypertension
    - Chronic kidney disease
    - Renovascular hypertension
    - Primary aldosteronism and other mineralocorticoid excess states
    - Cushing's syndrome and other glucocorticoid excess states
    - Drug-induced
        - NSAIDs, cyclooxygenase 2 inhibitors
        - Cocaine, amphetamines, other illicit drugs
        - Sympathomimetics (decongestants, anorectics)
        - Oral contraceptive hormones
        - Adrenal steroid hormones
        - Cyclosporine and tacrolimus
        - Erythropoietin
        - Licorice
        - Selected over-the-counter dietary supplements and medicines (e.g., ephedra, ma huang, bitter orange)
    - Pheochromocytoma
    - Coarctation of the aorta
    - Sleep apnea
    - Hyperparathyroidism
    - Hyperthyroidism

Diagnostic Approach

- Initial history, physical examination, and laboratory tests should be directed at:
    - Uncovering correctable forms of secondary hypertension
    - Establishing a pretreatment baseline
    - Determining presence of target organ damage
        - LVH
        - Coronary heart disease
        - Heart failure
        - Stroke or transient ischemic attack
        - Chronic kidney disease
        - Peripheral arterial disease
        - Retinopathy
    - Assessing factors that may influence the type of therapy
    - Determining presence of other risk factors for arteriosclerotic cardiovascular disease
        - Cigarette smoking
        - Obesity (body mass index $\geq$30 kg/m^2)
        - Dyslipidemia

        - Diabetes mellitus
        - Microalbuminuria and/or estimated glomerular filtration rate (GFR) <60 mL/min
        - Family history of premature cardiovascular disease (men <55 years of age or women <65 years)
        - Physical inactivity

Laboratory Tests

- The following laboratory tests should be done all patients with documented hypertension.
    - Urinalysis
    - Hematocrit
    - Serum potassium
    - Serum glucose
    - Serum calcium
    - Serum creatinine and/or blood urea nitrogen
    - Fasting high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and triglycerides
- Other tests that may be helpful in selected patients.
    - Urinary albumin excretion or albumin/creatinine ratio >30 mg/g, to rule out albuminuria
        - Albuminuria, including microalbuminuria, even in the setting of normal GFR, is associated with an increase in cardiovascular risk.
    - High-sensitivity C-reactive protein
        - Marker of inflammation
        - Persons with elevated levels have higher cardiovascular event rates.
    - Homocysteine
        - Elevated levels have also been linked to higher cardiovascular risk.
        - Not as robust a marker as high-sensitivity C-reactive protein

Imaging

- Echocardiography is indicated in stage II or longstanding, untreated stage I hypertension.
    - LVH suggests chronicity of hypertension.
    - Persons with LVH are more than twice as likely to have premature cardiovascular events or death.

Diagnostic Procedures

- Electrocardiography
    - Changes of LVH may be present.
        - Deep S waves in right precordial leads and tall R waves in left precordial leads [e.g., SV_1 + (RV_5 or RV_6) ≥35 mm; or (RV_5 or RV_6) ≥25 mm]
        - Electrocardiography substantially underestimates the frequency of cardiac hypertrophy compared with that observed with echocardiography.
- Ambulatory BP monitoring
    - Can be useful in:
        - Diagnosing white coat hypertension in the absence of target organ injury
        - Evaluating refractory hypertension and circadian patterns of BP
        - Determining relationship between BP and such symptoms as dizziness and visual changes

Classification

Joint National Committee classification

- Normal BP
  - SBP <120 mmHg
  - DBP <80 mmHg
- Pre-hypertension
  - SBP 120–139 mmHg **or**
  - DBP 80–89 mmHg
- Hypertension
  - Stage I
    - SBP 140–159 mmHg **or**
    - DBP 90–99 mmHg
  - Stage II
    - SBP >160 mmHg **or**
    - DBP >100 mmHg

Treatment Approach

Indications

- SBP >140 mmHg repeatedly
- DBP >90 mmHg repeatedly
- DBP 85–90 mmHg in patients with atherosclerotic vascular disease or diabetes mellitus
- White coat hypertension
  - Management is still debated.
  - Most recommend lifestyle modification.
  - Currently, data are insufficient to warrant treatment with antihypertensive therapy unless other coronary risk factors, such as hyperlipidemia, diabetes or cigarette smoking, are present.

Goal

- Reduce cardiovascular and renal morbidity and mortality.
  - BP <130/80 mmHg for patients with diabetes or kidney disease
  - BP <140/90 mmHg for all others

Therapies

- Lifestyle modifications
  - Dietary management
  - Aerobic exercise
  - Weight reduction
  - Control of other risk factors contributing to arteriosclerosis
- Drug therapy
  - Classes
    - Diuretics
    - β blockers
    - ACE inhibitors
    - Angiotensin receptor blockers

        - Calcium-channel blockers
        - Vasodilators
    - For patients without other coronary risk factors, start with a low dose of a single agent and, if BP is not controlled, increase dose; if BP still does not reach goal, move to combination therapy.
    - For medium- to high-risk patients, strongly consider low-dose combination therapy as initial therapy.
    - Coexisting conditions should guide initial drug therapy.
        - Heart failure: ACE inhibitor, angiotensin receptor antagonist, diuretic
        - After myocardial infarction: β blocker, ACE inhibitor
        - Coronary artery disease or high risk: β blocker, ACE inhibitor, calcium-channel blocker, diuretic
        - Diabetes: ACE inhibitor or angiotensin receptor antagonist
        - Chronic kidney disease: ACE inhibitor or angiotensin receptor blocker
        - Recurrent stroke prevention: diuretic or ACE inhibitor
    - If BP is not controlled with 2 agents, a detailed search for a secondary cause of hypertension is indicated.
        - Lower levels of suspicion in diabetics and elderly persons
        - If a secondary cause is not found, dietary assessment will often reveal a high sodium intake.
    - Once an appropriate drug combination has been found, use of a formulation that combines the drugs may simplify the regimen, increasing compliance.
    - Reducing the number of times each day that a patient must interrupt his or her schedule for medication improves compliance.

Specific Treatments

Lifestyle modifications

- Nondrug therapeutic intervention is indicated in all patients with sustained hypertension.
- Sodium restriction
    - Recommendation: <6 g sodium chloride per day
    - Can usually be achieved by eliminating table salt, reducing intake of processed foods, and eliminating all additions of salt during food preparation
    - Significantly potentiates the efficacy of nearly all antihypertensive agents
    - Direct benefit for salt-sensitive hypertensive patients
- Diet
    - Increase in potassium and/or calcium intake may be helpful.
    - DASH (Dietary Approaches to Stop Hypertension) diet
        - Natural foods that are high in potassium and low in saturated and total fat, emphasizing fruits, vegetables, and low-fat dairy products
        - Significantly decreased BP in borderline and stage 1 hypertensive persons
        - The sequel DASH-Sodium trial found that coupling the DASH diet with moderate sodium restriction led to greater decreases in BP than did dietary manipulation alone.
- Weight loss
    - Weight goal: body mass index <25 kg/m^2
    - 10 kg of weight loss has been shown to reduce SBP by 5–20 mmHg.
- Alcohol intake should be limited to 15 mL (1 drink) daily.
- Regular aerobic exercise
    - Everyone who is able should engage in regular aerobic physical activity, such as a brisk walk, for 30 minutes a day on most days.

    - Exercise is helpful in controlling weight, and physical conditioning itself may lower arterial pressure.
- Control of other risk factors contributing to arteriosclerosis
    - Restriction in cholesterol and saturated fat is recommended.
    - Smoking cessation should be strongly encouraged.
- Relaxation techniques may also lower arterial pressure.

Drug therapy

Diuretics

- Should be a component of most antihypertensive regimes
- Have been shown to reduce mortality and morbidity in long-term trials
- Particularly effective in elderly and African-American patients
- Mechanism of action
    - Early effect is related to sodium diuresis and volume depletion.
    - A reduction in peripheral vascular resistance in the long term has been reported.
- Major side effects can be minimized by using lower doses.
    - Hypokalemia
    - Hyperglycemia
    - Hyperuricemia
    - Hyperlipidemia
- Thiazide diuretics
    - Preferred over loop diuretics because of longer duration of action
    - Usual dose range
        - Hydrochlorothiazide: 12.5–50 mg/d
        - Chlorthalidone: 12.5–25 mg/d
        - Indapamide: 1.25–2.5 mg/d
        - Metolazone: 2.5–5 mg/d
- Loop diuretics
    - More potent than thiazides when GFR <25 mL/min
    - Usual dose range
        - Furosemide: 20–80 mg bid
        - Bumetanide: 0.5–2 mg bid
        - Torsemide: 2.5–10 mg/d
- Potassium-sparing diuretics
    - Can also be given along with thiazide diuretics to minimize renal potassium loss
    - A major disadvantage is that they can produce hyperkalemia, particularly in patients with impaired renal function.
    - Usual dose range
        - Amiloride: 5–10 mg once or twice daily
        - Triamterene: 50–100 mg once or twice daily

ACE inhibitors

- ACE inhibitors are well tolerated, with few side effects.
- Especially useful in renal or renovascular hypertension, in diabetic patients, and in accelerated hypertension
- Mechanism of action
    - Inhibit the enzyme converting angiotensin I into angiotensin II, a potent vasoconstrictor
    - Retard the degradation of a potent vasodilator (bradykinin)

    - Increase production of prostaglandin
    - Reduce the activity of the adrenergic nervous system
- Adverse effects
    - Nonproductive cough may develop in the course of therapy in up to 10% of patients.
    - Hyperkalemia, especially in patients with renal insufficiency
        - Potassium supplements and potassium-sparing diuretics should be used cautiously with ACE inhibitors to prevent hyperkalemia.
    - Angioedema, an idiosyncratic reaction
- Renal function may deteriorate as a result of ACE inhibitors in patients with bilateral renal artery stenosis.
    - Serum creatinine should be checked within 2 weeks of starting an ACE inhibitor.
- Usual dose range
    - Benazepril: 10–40 mg/d
    - Captopril: 25–100 mg bid
    - Enalapril: 5–40 mg once or twice daily
    - Fosinopril: 10–40 mg/d
    - Lisinopril: 10–40 mg/d
    - Moexipril: 7.5–30 mg/d
    - Perindopril: 4–8 mg/d
    - Quinapril: 10–80 mg/d
    - Ramipril: 2.5–20 mg/d
    - Trandolapril: 1–4 mg/d

Angiotensin receptor blockers

- Effects similar to those of ACE inhibitors, with fewer side effects (specifically, they do not cause cough or angioedema)
- Mechanism of action
    - Competitively inhibit the binding of angiotensin II to the angiotensin II AT_1 receptor subtype
- Usual dose range
    - Irbesartan: 150–300 mg/d
    - Losartan: 25-100 mg once or twice daily
    - Valsartan: 80–320 mg/d

β Blockers

- Have been shown to reduce morbidity and mortality in long term trials.
- Mechanism of action
    - Block sympathetic effects on the heart
    - Block the adrenergic nerve–mediated release of renin from the renal juxtaglomerular cells
    - Labetalol and carvedilol exert both α- and β-adrenergic blocking actions, so they act by reducing systemic vascular resistance.
    - Useful in conjunction with:
        - Vasodilators, which tend to evoke a reflex increase in heart rate
        - Diuretics, which can result in an elevation of circulating renin activity
    - Particularly effective in young hypertensive patients with "hyperkinetic" circulation

- Relative contraindications
  - Bronchospasm
    - Cardioselective β-blocking agents (so-called β_1 blockers, e.g., metoprolol, atenolol) may be superior to nonselective β blockers in patients with bronchospasm.
  - Decompensated congestive heart failure
  - Atrioventricular block and bradycardia
  - "Brittle" insulin-dependent diabetes
- Usual dose range
  - Atenolol: 25–100 mg/d
  - Metoprolol: 50–100 mg bid
  - Metoprolol, extended release: 50-100 mg/d
  - Nadolol: 40–120 mg/d
  - Propranolol: 40–160 mg bid
  - Propranolol, long acting: 60–180 mg/d
  - Timolol: 20–40 mg bid
  - Labetalol: 200–800 mg bid
  - Carvedilol: 12.5–50 mg bid

Calcium-channel blockers

- Mechanism of action
  - Modify calcium entry into cells by interacting with specific binding sites on the α_1 subunit of the L-type voltage-dependent calcium channel
  - Cause vasodilation
  - Both diltiazem and verapamil can slow atrioventricular conduction, but usually only the dihydropyridines produce reflex tachycardia.
  - Direct arteriolar vasodilators; all have negative inotropic effects and should be used cautiously in patients with congestive heart failure
- Usual dose range
  - Dihydropyridines (use only long-acting agents in this class)
    - The SYST-EUR (Systolic Hypertension in Europe) trial documented that, in patients >60 years with isolated systolic hypertension, a long-acting dihydropyridine calcium-channel blocker reduced cardiovascular morbidity and mortality to an extent equivalent to that previously reported for diuretics and β blockers.
    - Amlodipine: 2.5–10 mg/d
    - Felodipine: 2.5–10 mg/d
    - Nifedipine, long acting: 30–90 mg/d
      - Short-acting nifedipine has been reported to increase the incidence of acute coronary events and is not appropriate therapy for managing essential hypertension.
  - Nondihydropyridines
    - Diltiazem, extended release: 180–300 mg/d
    - Verapamil, long acting: 120–480 mg once or twice daily
    - Verapamil: 30–120 mg 4 times daily

Other medications

- These agents are not first-line therapy; they are most often used in severe hypertension refractory to other therapies.
- Centrally acting α agonists

    - Mechanism of action
        - Stimulate α_2 receptors in the vasomotor centers of the brain, reducing sympathetic outflow and arterial pressure
        - Decrease in cardiac output and heart rate usually occurs.
    - Agents include clonidine and methyldopa.
    - Rebound hypertension may occur rarely when clonidine therapy is stopped.
- Vasodilators
    - Cause direct relaxation of vascular smooth muscle
    - Hydralazine
        - Most versatile
        - Effect on peripheral resistance is partly negated by a reflex increase in sympathetic discharge that increases heart rate and cardiac output, limiting usefulness, especially in patients with severe coronary artery disease.
    - Minoxidil
        - More potent than hydralazine
        - Produces significant hypertrichosis and fluid retention
        - Mainly limited to patients with severe hypertension and renal insufficiency
- α-Adrenergic receptor blockers
    - Prazosin, terazosin, and doxazosin selectively block only postsynaptic α receptors.
    - Use has decreased with a report of their association with an increase in cardiovascular events.

Monitoring

- Follow-up based on initial BP
    - Normal: 2 years
    - Pre-hypertension: 1 year
    - Stage I hypertension: 2 months
    - Stage II hypertension: 1 month
    - BP >180/110 mmHg: Evaluate and treat immediately or within 1 week.
- After BP is at goal and stable, follow-up visits can usually be at 3- to 6-month intervals.
    - Comorbid conditions, such as heart failure; associated diseases, such as diabetes; and the need for laboratory tests influence the frequency of visits.
- Serum potassium and creatinine should be monitored at least 1–2 times per year.
- Other cardiovascular risk factors should be treated to their respective goals.
- Tobacco avoidance should be promoted vigorously.
- Low-dose aspirin therapy should be considered only when BP is controlled, because the risk of hemorrhagic stroke is increased in patients with uncontrolled hypertension.

Complications

- Accelerated atherosclerosis is responsible for many of the complications.
- Effects on the heart
    - LVH
    - Cardiomegaly
    - Congestive heart failure
    - Angina pectoris
    - Myocardial infarction
- Neurological effects
    - Cerebral infarction
    - Cerebral hemorrhage
    - Hypertensive encephalopathy

- Effects on the kidney
  - Arteriosclerotic lesions
  - Decreases GFR
  - Tubular dysfunction
  - Hypertension renal changes are a common cause of renal dysfunction and failure.
- Retinal changes
  - Retinal exudates and hemorrhages
  - Papilledema

Prognosis

- Age, race, sex, smoking, alcohol intake, serum cholesterol, glucose intolerance, and weight may alter the prognosis of essential hypertension.
- Hypertension is a progressive and lethal disease if left untreated.
  - Untreated hypertension is associated with shortening of life by 10–20 years.
  - Persons with relatively mild disease—i.e., without evidence of end-organ damage—who are untreated for 7–10 years have a high risk of developing significant complications.
    - Nearly 30% will exhibit atherosclerotic complications.
    - >50% will have end-organ damage related to the hypertension itself (e.g., cardiomegaly, congestive heart failure, retinopathy, a cerebrovascular accident and/or renal insufficiency).
- Probably fewer than one-third of hypertensive patients in the U.S. are being treated effectively.
  - Most related to:
    - Failure to detect hypertension
    - Failure to institute effective treatment
    - Failure of the asymptomatic patient to adhere to therapy

Prevention

- Maintain a healthy body weight; avoid obesity.
- Exercise regularly.
- Limit salt, alcohol, and caffeine intake.

ICD-9-CM

- 401.0 Essential hypertension, Malignant Essential Hypertension
- 401.1 Essential hypertension, Benign Essential Hypertension
- 401.9 Essential hypertension, unspecified Essential Hypertension

See Also

- Health Care Screening and Disease Prevention
- Hypertension in African Americans
- Hypertension in Diabetes Mellitus
- Hypertension in Pregnancy
- Hypertension in the Elderly
- Hypertension Secondary to Chronic Kidney Disease
- Hypertensive Emergencies
- Renovascular Hypertension

Internet Sites

- Professionals
  - 7th Report of the Joint National Committee
    Prevention, Detection, Evaluation, Treatment of High Blood Pressure, NHLBI
  - Homepage
    American Heart Association
- Patients
  - High Blood Pressure
    MedlinePlus
  - Do the Effects of Blood Pressure Drugs Differ by Kidney Function?
    Annals of Internal Medicine, Summaries for Patients

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PEARL

- In persons >50 years of age, SBP >140 mmHg is a more important cardiovascular disease risk factor than is DBP.