Most patients will require multiple medications. Risk stratification affects the treatment: Sequential monotherapy attempts might be tried. Hemoglobin and hematocrit or CBC. Defined as SBP or DBP ≥ 150 mm Hg and/or diastolic BP (DBP) ≥ 90 mm Hg at ≥ 2 visits.

– Age 40 or older: systolic BP (SBP) ≥ 150 mm Hg and/or diastolic BP (DBP) ≥ 90 mm Hg at ≥ 2 visits.
– Operationally, any BP at which drug treatment results in a net benefit to a population (14A).
– HTN is a strong risk factor for cardiovascular disease.

Geriatric Considerations

– Isolated systolic HTN is common.
– Therapy has been shown to be effective and beneficial at preventing stroke, although target SBP is higher than in younger patients (~ 150 mm Hg systolic), and adverse reactions to medications are more frequent. The benefit of therapy has been conclusively demonstrated in older patients for systolic blood pressure ≥ 160 mm Hg (2).

Pediatric Considerations

– Measure BP during routine exams for ≥ 3 years of age.
– Defined as SBP or DBP ≥ 95th percentile on repeated measurements.
– Pre-HTN, SBP or DBP between 90th and 95th percentile (1).

Pregnancy Considerations

– Elevated BP during pregnancy may be either chronic HTN or pregnancy-induced hypertension (PIH). ACE inhibitors and angiotensin II receptor blockers (ARBs) are contraindicated.
– Maternal and fetal mortality benefit from treatment of severe HTN. Evidence not clear for mild HTN (see topic “Preeclampsia”).

DIAGNOSIS

HISTORY

– HTN is asymptomatic except in extreme cases or after related cardiovascular complications develop.
– Headache can be seen with higher BP, often present on awakening and nocturnal in nature.

PHYSICAL EXAM

– Arteriopathy: Narrowed arteries, arteriosclerosis (AV) nicking, copper or silver wiring of retinal arterials
– Increased S2 (diastolic component heart sound)
– Synchronous radial and femoral pulse can help to rule out coarctation of the aorta.

DIFFERENTIAL DIAGNOSIS

Secondary HTN: Because of the low incidence of reversible secondary HTN, special tests should be considered only if the history, physical exam, or basic laboratory evaluation indicate the possibility. (See “Hypertension, Secondary and Resistant.”)

DIAGNOSTIC TESTS & INTERPRETATION

ECG to evaluate possible presence of left ventricular hypertrophy (LVH) or rhythm abnormalities affecting therapy

Initial Tests (lab, imaging)

– Hemoglobin and hematocrit or CBC
– Complete urinalysis (may reveal proteinuria)
– Potassium, calcium, and creatinine, uric acid
– Lipid Panel (total, HDL, LDL, TG)
– Fasting blood glucose, hemoglobin A1c
– Only if history or physical indicate (low “Hypertension, Secondary and Resistant.”)

Follow-Up Tests & Special Considerations

– Special tests (only if history, physical, or labs indicate). (See “Hypertension, Secondary and Resistant.”)

Ambulatory (24-hour) BP monitoring if “white coat” HTN is suspected.
– Home BP monitoring is effective, especially if “white coat” HTN is a consideration (6). Elevated home BP correlates with adverse outcomes, possibly more so than office BP, and normal readings are reassuring.

Diagnostic Procedures/Other

– Age 60 or older: systolic BP (SBP) ≥ 150 mm Hg and/or diastolic BP (DBP) ≥ 90 mm Hg at ≥ 2 visits.
– Operationally, any BP at which drug treatment results in a net benefit to a population.

– Assuming proper resting conditions, cuff size, and application are maintained.
– The Joint National Committee (JNC) V recommends emphasis on:
– Family or personal history of HTN, cardiovascular, cerebrovascular, renal disease, and diabetes
– Previous elevated BPs

– Previous treatments

TREATMENT

– History of weight gain, exercise activities, sodium and fat intake, and alcohol use
– Symptoms suggesting secondary HTN
– Psychosocial and environmental factors affecting the BP risk for cardiovascular disease
– Other cardiovascular risk factors, such as obesity, smoking, hypertension, diabetes
– Funduscopic exam for arterial narrowing, AV compression, hemorrhages, exudates, and papilledema
– Body mass index (BMI)
– Waist circumference
– BP in both arms
– Complete cardiac and peripheral pulse exam: Compare radial and femoral pulse for differences in volume and timing, auscultation for carotid and femoral bruits.
– Abdominal exam for masses and bruits: Listen high in the flanks over the kidneys.
– Neurologic assessment

GENERAL MEASURES

– Treating patients to lower-than-standard BP targets, ≤ 140/90 mm Hg, does not further reduce mortality or morbidity (2,6A). Individual goal pressures based on risk factors; best target may be SBP at or just below 150 mm Hg in patients > 75–80 years of age (2) [A].
– Primary focus is SBP goal; treatment should accommodate patient preferences (4).
– Aerobic exercise, (30 minutes of aerobic activity 4–5 days/week), weight reduction for obese patients
– Smoking cessation
– Risk stratification affects the treatment:
– Pre-HTN (SBP 130–139/90–99 mm Hg) Drug therapy for chronic renal disease or diabetes. ACCORD trial did not support < 140/90 for pts ≥ 4 ≥ 2 diabetes mellitus (7).
– Evidence for benefit in treatment of Stage 1 hypertension (140–159/90–99 mm Hg) is sparse: Assess overall risk and individual desire to treat. Begin thiazide diuretics for many patients
– Stage 2 HTN (≥ 160 ≥ 100 mm Hg): Consider starting 2 drugs initially.

MEDICATION

– The amount of BP reduction is probably more important than the choice of antihypertensive.
– Multiple drugs at subclinical dose may achieve target BP with fewer side effects.
– Thiazide diuretics have the most proven benefits (cost, compliance, and effectiveness).
– Chlorothiazide and hydrochlorothiazide (HCTZ) equally effective, some data states rate of adverse events (hyperkalemia, hypotension) are more common with chlorothiazide (8,9A).
– Sequential monotherapy attempts might be tried with different classes because individual responses vary.
– Most patients will require multiple medications.

– First line agents for uncomplicated essential HTN include thiazide diuretics. Angiotensin Converting Enzyme Inhibitors (ACE) and long-acting dihydropyridine calcium channel blocker (Captopril, Lisinopril, Nifedipine). If concomitant conditions, choose first line agent based upon comorbidity (1).
HYPERTENSION, ESSENTIAL

- Combination first-line agents: Benazepril combined with an diuretic may be superior to combination with HCTZ in high-risk patients. Some suggest that ACEHMR + dihydropyridine calcium channel blocker is first choice after monotherapy.
- β-blockers had been strongly recommended until recent meta-analyses. Amiodarone may be particularly ineffective in reducing adverse outcomes of hypertension (except in patients with left ventricular hypertrophy underlying diastolic).
- ACE inhibitors should be used in patients with diabetes, proteinuria, atrial fibrillation, or congestive heart failure (CHF), but not in pregnancy.
- β-adrenergic blockers are not the 1st choice for monotherapy (β-blockers might benefit patients with ischemic heart disease, CHF, or migraine post STEMI).
- Calcium channel blockers could be considered in patients with isolated systolic HTN, atherosclerosis, angina, migraines, or asthma; well documented to reduce risk of stroke (5).

First Line

- Thiazide diuretics:
  - Chlorthalidone: 12.5–50 mg/d
  - Hydrochlorothiazide: 6.25–50 mg/d
  - Indapamide: 1.25–9 mg/d
- ACE inhibitors:
  - Lisinopril: 5–40 mg/d
  - Enalapril: 2.5–40 mg/d
  - Ramipril: 2.5–20 mg/d
  - Benazepril: 10–40 mg/d
- Calcium channel blockers:
  - Amlodipine: 5–30 mg/d
  - Nifedipine (sustained release): 30–90 mg/d
  - Verapamil (sustained release): 120–480 mg/d
  - Amlopidine: 2.5–10 mg/d
- Combinations:
  - Diuretics: May worsen gout.
  - β-blockers (relatively) in reactive airway disease, heart block, diabetes, and peripheral vascular disease; probably should be avoided in patients with metabolic syndrome or insulins requiring diabetes.
  - Olanzapine or varenicline: Do not use with systemic dysrhythmia heart failure or heart block.
- ACE inhibitors can worsen bilateral renovascular disease and are pregnancy Category D.

Second Line

- ARBs:
  - Losartan: 25–100 mg 1 or 2 doses; has unique but modest unique effect
  - Valsartan: 80–320 mg daily
  - Irbesartan: 75–300 mg daily
  - Candesartan: 4–32 mg daily
  - Kerin inhibitor: Alosa 150–300 mg daily
- Many may be combined. Choose additional medications with complementary effects (i.e., ACEHMR with diuretic or a vasodilator with a diuretic or β-blocker).

- β-adrenergic antagonists: Prazosin: 1–10 mg b.i.d., terazosin: 1–20 mg/d, or doxazosin: 1–16 mg/d
- Peripherally acting adrenergic-inhibitors: Rarely used: Reolinopril: 0.1–0.25 mg/d
- Valsartan:
  - Hypokalaemia: 25–150 mg b.i.d., risk of tachycardia, so genera combined with β-blocker; also drug-induced systemic lupus erythematosus (SLE)
  - Minoxidil: Rarely used due to adverse effects. May be more effective than other medications in renal failure and refractory hypertension.
- Loop diuretics (for volume overload): Furosemide:
  - Rarely used due to adverse effects. May be more effective than other medications in renal failure and refractory hypertension.

COMPLEMENTARY AND ALTERNATIVE MEDICINE

Bedrest and relaxation exercise

ONGOING CARE

FOLLOW-UP RECOMMENDATIONS

Patient Monitoring

- Re-evaluate patients q4–6mo until stable, then q6–12mo. Consider use of home self BP monitoring quality of life issues, including sexual function should be considered.
- Poor medication adherence is a leading cause of apparent medication failure.
- Annual urinalysis, creatinine, and potassium

DIET

- ~20% of patients will respond to reduced-salt diet (<100 mmol/d, or 6.5 NaCl or <2.4 g Na)
- Consider Dietary Approaches to Stop Hypertension (DASH) diet: www.medsch.ul.hcTUREH/patients/diet/dash/new_dash.pdf
- Limit alcohol consumption to ~1 unit.

PATIENT EDUCATION

- Emphasize the asymptomatic nature of HTN and importance of lifetime treatment.
- Printed aids for high BP education available: www.medsch.hc.ULH/Trud/r/patients/diet/dash/index.html

COMPLICATIONS

Heart failure, renal failure, UV, myocardial infarction, retinal hemorrhage, stroke, hypertensive heart disease, d.d. side effects

REFERENCES


SEE ALSO

Hypertension, Secondary and Resistant; Hypertensive Emergencies; Polycystic Kidney Disease

CODES

ICD10:
I10 Essential (primary) hypertension

CLINICAL PEARLS

- Treatment of HTN reduces risk of many serious medical conditions with numbers needed to treat to prevent 1 serious event (e.g., stroke or myocardial infarction) ranging from ~20 patients/year for severe HTN to more than several hundred per year for mild HTN.
- Multiple submaximal doses are likely to have fewer side effects and more effectiveness than fewer maximum-dosed drugs.
- In older patients, measure BP standing to avoid overestimation and systolic.
HYPERTENSION, SECONDARY AND RESISTANT

George H. Maxted, MD

DESCRIPTION
Uncontrolled hypertension (HTN) is composed of the following entities:
- Resistant hypertension was not addressed by JNC-VIII, but it can be assumed the goal would be <150/90 for those 60 and older who are uncontrolled on 3 antihypertensives, including a diuretic and 140/90 for those with chronic kidney disease and diabetes (1,2).
- Secondary HTN: Elevated BP that results from an identifiable underlying mechanism (1,2).

Geriatric Considerations
- The prevalence of resistant HTN (<20% of resistant HTN) is unknown. NHANES 2001-2002 indicated only 53% of adults are controlled to target end-organ damage, stage 2 HTN (systolic BP ≥160 mm Hg or diastolic BP ≥100 mm Hg), renal disease, and alcohol or drug use.
- In the ACCOMPLISH, INVEST, CONVINCE, ALLHAT, and LIFE studies, percent of patients reaching JNC-7 BP goals ranged from 40-82% (1).

Prevalence
Prevalence of resistant HTN is unknown. NHANES analysis indicates only 53% of adults are controlled to a BP of <140/90.

Etiology and Pathophysiology
- Obstructive sleep apnea (OSA): A study diagnosed OSA in 69% of treatment-resistant hypertension (2).
- Primary hyperaldosteronism (17-22% of resistant HTN cases) (2).
- Chronic renal disease (2-5% of hypertensives)
- Renovascular disease (0.2-0.7%, up to 39% of elderly, 20% of patients undergoing cardiac catheterization) (2).
- Cushing syndrome (0.1-0.6%)
- Pheochromocytoma (0.04-0.1% of hypertensives)
- Drug-related causes:
- Medications, especially NSAIDs (may also blunt effectiveness of ACE inhibitors), decongestants, stimulants (e.g., amphetamines), attention-deficit hyperactivity disorder (ADHD) medications, anabolic agents (e.g., modafinil), ephedra, pseudoephedrine, ma huang, bitter ephedra, ephedrine, natural licorice (in some chewing tobacco), yohimbine, glucocorticoids
- Oral contraceptive: Uterine association, mainly epidemiologic and with higher estrogen pills
- Cocaine, amphetamines, other illicit drugs, drug and alcohol withdrawal syndromes
- Lifestyle factors: Obesity, dietary salt may negate the beneficial effect of diuretics. Excessive alcohol, physical inactivity also contributors

Genetics
In some patients, there is a possible relationship to Ethnic gene variants (Liddle syndrome) and a CYP3A4 allele (constitutively expressed, especially African American) (2).

Risk Factors
A recent large cohort study revealed that those with resistant HTN (16.2%) were more likely to be male, Caucasian, older, and diabetic. They were also more likely to be taking β-blockers, calcium channel blockers, and α-adrenergic blockers compared with other drug classes (3). Factors predictive of resistant or secondary HTN: Female sex, African-American race, obesity, diabetes, worsening of control in previously stable hypertensive patient, onset in patients age ≥20 or ≤50, lack of Family history of HTN, significant target end organ damage, stage 2 HTN (systolic BP >160 mm Hg or diastolic BP >100 mm Hg), renal disease, and alcohol or drug use.

General Prevention
The prevention of resistant and secondary HTN is thought to be the same as for primary or essential HTN. Adopting a DASH (Dietary Approaches to Stop Hypertension’s diet, a low-sodium diet, weight loss in obese patients, exercise, limitation of alcohol intake, and smoking cessation may all be of benefit. Relaxation techniques may be of help, but data are limited.

Diagnosis
History
- Ask or review at every visit: “The Big Four”: SANS mnemonic: 1. Salt intake, 2. Alcohol intake, 3. NACOS use. 4. Sleep (author’s suggestion, based on reference list).
- Review home BP readings; consider ambulatory BP monitoring.
- History will vary with etiology of HTN:
- – Pheochromocytoma: Episodes of headache, palpitations, sweating
- – Cushing syndrome: Weight gain, fatigue, weakness, easy bruising, amenorrhea
- – OSA: Loud snoring while awake, daytime somnolence

Physical Exam
- Ensure that the BP is measured correctly. The patient should be sitting quietly with back supported for 5 minutes before measurement. Proper cuff size. Bladder ensuring at least 80% of the arm. Support arm at heart level. Minimum of 2 readings at least 1 minute apart. Check BP in both arms. Also check standing BP for orthostasis.
- Attention to findings related to possible etiologies: Renovascular HTN: Systolic-diastolic, abdominal bruit. Pheochromocytoma, Diaphoresis, tachycardia. Cushing syndrome: Hirsutism, moon face, dorsalis humps, purple striae, truncal obesity. Thyroid disease: Enlarged thyroid, bruxism, exophthalmos, tachycardia. Cricopharyngeal or the aorta: Upper lab HHTN with decreased or delayed femoral pulses.

Diagnostic Tests & Interpretation
- ECG performed as part of the initial workup; LVM is an important marker of resistant HTN.
- Sleep study if history and physical indicate. Overnight oximetry is a good screen for nocturnal hypoxemia. If positive, formal polysomnography may be indicated.

Initial Tests (lab, imaging)
Initial limited diagnostic testing should include (2,4):
- Urinalysis, CBC, potassium, sodium, glucose, creatinine, cholesterol, thyroid-stimulating hormone (TSH), calcium. 50% of patients with hypokalemic HTN may have normal potassium levels (1,2).
- Imaging tests listed are necessary only if history, physical, or lab data indicate.
- Abdominal US. If renal disease is suspected:
- Adrenal “incidentallyoma” frequently arise in this era of multiple CT studies. If present in the setting of resistant HTN, consider hyperaldosteronism or hyperadrenocortical states.
- Doppler or CTA imaging of aorta along with chest x-ray looking for notched ribs (sign of coarctation)
Follow-up Tests & Special Considerations
Further testing for primary aldosteronism (PA) may be considered:
- Empiric treatment with an aldosterone inhibitor may be preferable, and more clinically relevant.
- Spironolactone or eplerenone. Amlodipine may be more effective in African Americans.
- Plasma aldosterone-to-renin ratio (ARR) is the preferred lab test, but the test is difficult to perform and interpret properly. Consult your reference lab and interpret results with caution.
- Consider increasing the dose of thiazide or adding an aldosterone inhibitor (6).
- Additional benefit has been demonstrated with ACE inhibitors, angiotensin II receptor blocker (ARB) agents, calcium-channel blockers (2EC).
- RAAS blockers are considered 2nd-line only for patients with ischemic heart disease or CHF. Combined α-antagonists (e.g., labetalol) may be more effective for HTN.
- Aldosterone antagonists may offer significant benefit (15C;2,3).
- Central acting agents (e.g., clonidine) are effective at reducing BP but outcome data are lacking.

Alert
- Agents specific for treatment of HTN emergencies should be initiated under a situation in which immediate BP reduction will prevent or limit end-organ damage (see topic “Hypertensive Emergencies”).
- Renovascular HTN: Angioplasty is the choice for thromboendarterectomy or stenosis.
- End-organ damage: Despite treatment, only a few patients may recover full organ function.

In-Patient Considerations
Admission Criteria/Initial Stabilization
Hospitalization may be necessary for hypertensive urgency or emergency general measures.

Ongoing Care
Follow-up Recommendations
Encourage aerobic activity of 30 min/d, depending on patient condition.
- Diuretics, especially thiazide diuretics, are 1st line (6).
- Tolerance to diuretics may occur. Long-term adaptation to thiazide or the “breakthrough” effect. Consider increasing the dose of thiazide or adding an aldosterone inhibitor (6).
- Treatment specific to certain secondary etiologies:
  - Primary aldosteronism: Aldosterone receptor antagonist: Spironolactone or eplerenone
  - Cushings syndrome: Aldosterone receptor antagonist
    ◆ OSA: Continuous positive airway pressure (CPAP)
    ◆ Sleep apnea: Oxygen supplementation
  - Hypothyroidism: Hypothyroidism
  - Hyperparathyroidism; Hypertension, Essential; Cushing Disease and Cushing Syndrome; Aldosteronism, Primary; Coarctation of the Aorta; Fibromuscular Dysplasia of a Renal Artery; Hypertension, Secondary; Diabetes Mellitus Type 1 (IDDM); Diabetes Mellitus Type 2 (NIDDM); Hypertensive Event

Codes
ICD10
- I15.0 Renovascular hypertension
- I15.8 Other secondary hypertension
- I15.9 Secondary hypertension, unspecified

Clinical Pearls
- Avoid use of HTN in adults <60 years of age is a strong indicator of secondary HTN.
- Common causes of resistant HTN: Obstructive sleep apnea, excessive salt intake, medication nonadherence
- Common secondary causes include sleep apnea, retinal diseases, renal artery stenosis, and primary aldosteronism
- Home BP monitoring predicts outcomes better than office monitoring of BP.

REFERENCES
**HYPERTENSION EMERGENCIES**

John A. Guisto, MD, FACEP • Arthur B. Sanders, MD, MHA

**BASICS**

**DESCRIPTION**
- Numerous terms, often overlapping. Some definitions include a specific BP reading; others emphasize acute change in BP or presence of specific clinical syndromes.
- Severe HTN is defined as a diastolic BP of ≥115 mm Hg (15.3 kPa).
- A hypertensive emergency occurs only when an acute elevation of BP causes rapid and progressive end-organ damage, particularly in the cardiovascular, renal, and CNS.
- System(s) affected: Cardiovascular; Nervous; Pulmonary; Renal
- Systemic hypertension crisis; Severe HTN; Multiple HTN; Accelerated HTN; Hypertensive emergency

**EPIDEMIOLOGY**

**Incidence**
Incidence of hypertensive emergency: 1% of patients with hypertension annually in the US

**Prevalence**
Overall prevalence of HTN in the US: 29.3% based on a 2003–2004 survey data

**ETIOLOGY AND PATHOPHYSIOLOGY**
- Increased sympathetic tone leads to increased BP.
- Angiotensin II has multiple effects contributing to HTN and end-organ damage:
  - Stimulates sympathetic tone, aldosterone release, and antidiuretic hormone release
  - Chronic HTN induces vascular thickening and sclerosis.
  - Central effects include reduced growth of salt and water.
- Chronic HTN shifts autoregulation of BP and renal blood flow.

**Renal disease**
- Acute withdrawal from antihypertensives, especially chlorothiazides (Diuretics)
- Withdrawal from CNS depressants
- Medications: NSAIDs, decongestants, appetite suppressants, drugs of abuse (cocaine, amphetamines)
- Edema/pleural effusions
- Thrombotic thrombocytopenic purpura
- Nephrotoxicity
- Severe burns
- Postoperative HTN

**Genetics**
- Genetic: Risk of hypertensive emergency is higher in African Americans.
- Pregnancy or drugs of abuse

**RISK FACTORS**
- History of poorly controlled HTN
- Drug abuse
- Noncompliance with medications; abruptly stopping antihypertensive medication without supervision

**GENERAL PREVENTION**
- Treat HTN and counsel patients on importance of compliance with antihypertensive treatment and dangers of stopping medications abruptly.

**COMMONLY ASSOCIATED CONDITIONS**
- Renovascular HTN
- Retinal hemodynamics
- Renal failure

**Geriatric Considerations**
- Elderly patients may experience isolated systolic HTN due to decreased baroreceptor sensitivity.

**Pediatric Considerations**
- Usually associated with renal disease
- May present with abdominal pain
- May present with abdominal pain

**Pregnancy Considerations**
- Labetalol, nicardipine, or hydralazine are preferred.
- Nitroprusside decreases placental blood flow and cytosol metabolite crosses the placenta; may result in fetal toxicity with prolonged exposure.
- Treat preeclampsia.

**DIAGNOSIS**

Clinical presentation varies depending on organ system affected.

**HISTORY**
- Headache
- Elevated mental state
- Nausea, vomiting
- Neurologic disturbance
- Shortness of breath, dyspnea, orthopnea
- Chest pain
- Abdominal pain
- Epistaxis

**PHYSICAL EXAM**
- HTN
- Focal neurologic deficits, stupor, coma
- Retinopathy
- Funduscopic exam may reveal papilledema, exudates, or hemorrhages.
- Pulmonary edema
- Hemorrhage, thoracostomy, embolus
- Renal or abdominal bruits
- Unequal BP or pulses in the extremities

**DIFFERENTIAL DIAGNOSIS**
- Aortic dissection
- Carotid
- Stroke
- Other CNS pathology (e.g., encephalopathy)
- Acute pulmonary edema
- Renal failure

**DIAGNOSTIC TESTS & INTERPRETATION**
- **Urinalysis and renal function tests** (red cell casts, hematuria, proteinuria are common)
- Drug screen
- Blood count and smear may indicate microangiopathic hemolytic anemia or thrombocytopenia.

**TREATMENT**

**GENERAL MEASURES**
- If ongoing end-organ damage is thought to be secondary to hypertensive state, prompt treatment with IV medication is indicated. Monitor patient closely to avoid rapid fall in BP can be avoided.

**MEDICATION**
- A 2008 Cochrane Review (1) noted no randomized clinical trials show reduction in mortality from recommended treatments and no clear basis for recommendations in drug over another. These treatments do reduce BP in these circumstances, and evidence levels reflect their effectiveness. A single randomized trial and review article compared IV nicardipine with labetolol, suggesting nicardipine more reliably reaches target BP with similar rate of overshoot or need for additional rescue medication (2,3).

**ALERT**
- The general goal is to lower mean arterial pressure (MAP) by ~20% or reduce diastolic pressure to 100–110 mm Hg (13.3–14.6 kPa) over 1 hour
- **Lower BP < 20% in the 1st hour; then, if stable, lower to 160/110 over the next 2–6 hours**
- MAP = 1/3 of the sum of twice the diastolic pressure plus the systolic pressure.
Follow BP closely to avoid a rapid drop.

Ongoing BP control plus monitoring of affected organ system(s) may outweigh the risks in patients with severe HTN but no secondary end-organ damage. No studies prove that aggressive treatment reduces the risk of long-term morbidity or mortality from hypertensive urgency.

BP should return to acceptable levels within 24 hours.

Follow-up recommendations:

- Follow-up closely to avoid a rapid drop.
- Begin oral therapy as soon as possible after BP control has been achieved with IV medications.
- Ongoing BP control plus monitoring of affected organ system(s) (e.g., renal function) is recommended to ensure continued morbidity.

DIET

Low-sodium diet

PATIENT EDUCATION

- Avoid abrupt discontinuation of antihypertensive medicines.
- Stress importance of compliance.

Surgery/other procedures

- An arterial catheter may be used to monitor BP advantage over noninvasive monitoring not clearly proven (T1C)

Complementary and alternative medicine

Hypertensive environment, which may lower the BP

In-patient considerations

Admission criteria/initial stabilization

In general, lower the BP no more than 20% in the first hour; then, if stable, lower to 160/100–110 in the next 2–6 hours.

All patients with true hypertensive emergencies should be hospitalized.

Associated end-organ effects may require specific treatment (e.g., acute myocardial infarction).

IV fluids

Fluid restriction may be appropriate for associated pathophysiology, such as pulmonary edema.

Nursing

Bed rest

Discharge criteria

Patient should be stabilized on PO antihypertensives as appropriate (R1A).

Follow-up recommendations

- Close outpatient follow-up with primary care physician is recommended to ensure ongoing control of HTN.

Patient monitoring

- Follow-up closely to avoid a rapid drop.
- Begin oral therapy as soon as possible after BP control has been achieved with IV medications.
- Ongoing BP control plus monitoring of affected organ system(s) (e.g., renal function) is recommended to ensure continued morbidity.

Diet

Low-sodium diet

Patient education

- Avoid abrupt discontinuation of antihypertensive medicines.
- Stress importance of compliance.

- Emphasize the lack of symptoms with HTN until secondary end-organ damage occurs.

Prognosis

- BP should return to acceptable levels within 24 hours.
- Long-term prognosis depends on extent of secondary end-organ damage in addition to ongoing BP control.

Complications

- Depend on organ system(s) secondarily affected.
- Abrupt or excessive lowering of BP may result in inadequate cerebral or cardiac blood flow, leading to strike or myocardial infarction.
- The benefits of aggressive treatment may outweigh the risks in patients with severe HTN but no end-organ damage. No studies prove that aggressive treatment reduces the risk of long-term morbidity or mortality from hypertensive urgencies.

Surgeon/other procedures

- An arterial catheter may be used to monitor BP advantage over noninvasive monitoring not clearly proven (T1C).

References


See also

Acute Dissection, Hypertension, Essential; Phaeochromocytoma; Preeclampsia and Eclampsia (Essentials of Pregnancy)

ICD10

I10 Essential (primary) hypertension

Clinical pearls

- Treatment of severe HTN (hypertensive urgency) without evidence of acute end-organ damage is controversial. This treatment is in the emergency department by initiation of oral medication, with close follow-up.
- If severe HTN and evidence of end organ damage, slowly initiate IV treatment with close inpatient monitoring is recommended.
- Avoid rapid prehospital lowering of BP.
- Treatment depends on the organ systems affected.
- Esmolol, nitroprusside, and ACE inhibitors are contraindicated in pregnancy.