Hypertensive emergency is defined as severe hypertension associated with acute end-organ damage, such as hypertensive encephalopathy, subarachnoid or intracerebral hemorrhage, acute pulmonary edema, or aortic dissection. Severe hypertension is defined as systolic blood pressure 180 mmHg and/or diastolic blood pressure 120 mmHg. Immediate but careful reduction in blood pressure is indicated for hypertensive emergency. Excessive hypotension caused by drug treatment is dangerous and could result in stroke, myocardial infarction or blindness.

# Antihypertensive drugs

**A. Nitroprusside**, an arteriolar and venous dilator, is given as an intravenous infusion. Initial dose: 0.25 to 0.5 µg/kg per min; maximum dose: 8 to 10 µg/kg per min.

**B. Nitroglycerin**, a venous and arteriolar dilator, is given as an intravenous infusion. Initial dose: 5 µg/min; maximum dose: 100µg/min.

**C. Labetalol (Trandate)**, an alpha- and ß-adrenergic blocker, is given as an intravenous bolus or infusion. Bolus: 20 mg initially, followed by 20 to 80 mg every 10 minutes to a total dose of 300 mg. Infusion: 0.5 to 2 mg/min.

**D. Nicardipine (Cardene)**, a calcium channel blocker, given as an intravenous infusion. Initial dose: 5 mg/h; maximum dose: 15 mg/h.

**E. Fenoldopam (Corlopam)**, a peripheral dopamine-1 receptor agonist, is given as an intravenous infusion. Initial dose: 0.1

µg/kg per min; the dose is titrated at 15 min intervals, depending upon the blood pressure response.

**F. Hydralazine**, an arteriolar dilator, is given as an intravenous bolus. Initial dose: 10 mg given every 20 to 30 minutes; maxi- mum dose: 20 mg.

**G. Propranolol (Inderol)** a ß-adrenergic blocker is given as an intravenous infusion and then followed by oral therapy. Dose: 1 to 10 mg load, followed by 3 mg/h.

**H. Phentolamine**, an a-adrenergic blocker, is given as an intravenous bolus. Dose: 5 to 10 mg every 5 to 15 minutes.

**I. Enalaprilat (Vasotec IV)** an angiotensin converting enzyme inhibitor, is given as an intravenous bolus. Dose: 1.25 mg every six hours.

# Choice of agent for hypertensive emergencies

**A.** Nitroprusside is the most rapid-acting and potent parenteral antihypertensive agent. It acts within seconds and has duration of action of only 2 to 5 minutes. Hypotension can be easily reversed by temporarily discontinuing the infusion. The major limitation is the development of cyanide toxicity with high doses, for a prolonged period (>24 to 48 hours), or with underlying renal insufficiency.

**B.** Alternatives to nitroprusside include intravenous labetalol, nicardipine, and fenoldopam. These agents are better than nitroprusside because hypotension is uncommon with these drugs and cyanide toxicity does not occur.

**C.** Intravenous fenoldopam, a dopamine agonist that acts at the vasodilator DA1 receptors, is as effective and almost as

short-acting as nitroprusside. It has the advantages of increasing renal blood flow and sodium excretion, of not causing accu- mulation of cyanide, and of not requiring shielding from light.

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| **Parenteral drugs for treatment of hypertensive emergencies** | | | | | |
| **Drug** | **Dose** | **Onset** | **Duration** | **Adverse Effect** | **Indications Adverse Effect** |
| **Vasodilators** | | | | | |
| Nitroprus side | 0.25-  10  µg/kg/ min as IV in- fusion | Immediate | 1-2 min | Nausea, vomiting, muscle twitching, sweating, thiocynate and cyanide intoxication | Most hyper- tensive emer- gencies; cau- tion with high intracranial pressure or azotemia |
| Nicardipi ne (Cardene) | 5-15 mg/h IV | 5-10 min | 15-  30 min, may ex- ceed  4 h | Tachycar- dia, head- ache, flushing, local phlebitis | Most hyper- tensive emer- gencies ex- cept acute heart failure; caution with coronary ischemia |
| Fenoldop am (Corlopa m) | 0.1-  0.3  µg/kg per min IV infu- sion | <5 min | 30 min | Tachycar- dia, head- ache, nau- sea, flush- ing | Most hyper- tensive emer- gencies; cau- tion with glau- coma |
| Nitroglyc- erin | 5-100  µg/min | 2-5 min | 5-10 min | Headache, vomiting, | Coronary ischemia |

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| **Drug** | **Dose** | **On- set** | **Duration** | **Adverse effects** | **Indications** |
|  | as IV infu- sion |  |  | methemoglo binemia, tolerance with prolonged use |  |
| Enalapril at (Vasotec IV) | 1.25-5 mg every  6 h IV | 15-  30 min | 6-12 h | Precipitous fall in pres- sure in high- renin states; variable response | Left ventricu- lar failure; avoid in acute myocardial infarction |
| Hydralazi ne | 10-20 mg IV | 10-  20 min IV | 1-4 h  IV | Tachycar- dia, flush- ing, head- ache, vomit- ing, angina | Eclampsia |
| 10-40 mg IM | 20-  30 min IM | 4-6 h  IM |
| **Andrenergic inhibitors** | | | | | |
| Labetalol (Trandat e) | 20-80 mg IV bolus every  10 min | 5-10 min | 3-6 h | Vomiting, scalp tin- gling, broncho- constriction, dizziness, nausea, heart block, orthostatic hypotension | Most hyper- tensive emer- gencies ex- cept acute heart failure |
| 0.5-  2.0 mg/mi n IV infu- sion |
| Esmolol (Breviblo c) | 250-  500  µg/kg/ min by infu- sion; may repeat bolus after 5 min or in- crease infu- sion to  300  µg/min | 1-2 min | 10-  30 min | Hypotensio n, nausea, asthma,  first-degree heart block, HF | Aortic dissec- tion, perioperative |
| Phentola mine | 5-15 mg IV bolus | 1-2 min | 10-  30 min | Tachycardia, flushing, head- ache | Catecholamine excess |

# Treatment of specific hypertensive emergencies

**A. Ischemic stroke or subarachnoid or intracerebral hemor- rhage.** The benefit of reducing the BP in these disorders must be weighed against possible worsening of cerebral ischemia caused by the thrombotic lesion or by cerebral vasospasm. These cerebrovascular events manifest as an abrupt onset of focal neurologic findings. Hypertensive encephalopathy, however, manifests as gradual insidious onset of headache, nausea, vomiting, and confusion.

**B. Acute pulmonary edema.** Hypertension in patients with acute left ventricular failure due to systolic dysfunction should be treated with vasodilators. Nitroprusside or nitroglycerin with a loop diuretic is the regimen of choice. Drugs that decrease cardiac contractility (labetalol, other beta blockers) should be avoided.

**C. Angina pectoris or acute myocardial infarction.** Acute coro- nary insufficiency frequently increases the systemic blood pres- sure. Intravenous parenteral vasodilators, such as nitroprusside and nitroglycerin, are effective and reduce mortality in patients with acute myocardial infarction with hypertension. Labetalol is also effective in this setting. Drugs that increase cardiac work (hydralazine) are contraindicated.

**D. Aortic dissection.** The initial aim of medical therapy is to de- crease the systolic pressure to 100-120 mmHg and reduce cardiac contractility. These goals are achieved by the combina- tion of nitroprusside and an intravenous beta blocker, such as propranolol or labetalol. Nitroprusside should not be given without a beta blocker.

**E. Withdrawal of antihypertensive therapy.** Abrupt discontinua- tion of a short-acting sympathetic blocker (such as clonidine or propranolol) can lead to severe hypertension and coronary ischemia. Control of the BP can be achieved in this setting by readministration of the discontinued drug and, if necessary, phentolamine, nitroprusside, or labetalol.

**F. Acute increase in sympathetic activity.** Increased adrenergic activity can lead to severe hypertension.

**1.** Pheochromocytoma.

**2.** Autonomic dysfunction, as in the Guillain-Barré syndrome or post-spinal cord injury.

**3.** Sympathomimetic drugs, such as, cocaine, amphetamines, phencyclidine, or the combination of an MAO (monoamineoxidase) inhibitor and tyramine-containing foods (fermented cheeses, smoked or aged meats, Chianti, champagne, and avocados).

**4.** Control of the hypertension in these disorders can be achieved with phentolamine, labetalol, or nitroprusside. Ad- ministration of a ß-blocker alone is contraindicated, since inhibition of ß-receptor-induced vasodilation results in unop- posed alpha-adrenergic vasoconstriction and a further rise in BP.

**G. Malignant hypertension** is marked hypertension with retinal hemorrhages, exudates, or papilledema. Malignant hypertension is usually associated with a diastolic pressure above 120 mmHg.

**1.** Malignant hypertension most often occurs in patients with

long-standing uncontrolled hypertension, many of whom have discontinued antihypertensive therapy.

**H. Hypertensive encephalopathy** refers to cerebral edema caused by severe and sudden rises in blood pressure.

**1.** Hypertensive encephalopathy can be seen at diastolic pres- sures as low as 100 mmHg in previously normotensive pa- tients with acute hypertension due to preeclampsia or acute glomerulonephritis.

**2.** Hypertensive encephalopathy is characterized by the gradual onset of headache, nausea, and vomiting, followed by rest- lessness, confusion, seizures, and coma.

**3.** These neurologic symptoms differ from the abrupt onset of focal neurologic symptoms seen with a stroke or hemorrhage. However, an MRI scan should be obtained to exclude stroke or hemorrhage.

# Goal of therapy

**A.** The initial aim of treatment in hypertensive crises is to rapidly lower the diastolic pressure to about 100 to 105 mmHg within two to six hours, with the maximum initial fall in BP not exceed- ing 25 percent of the presenting value.

**B.** More aggressive hypotensive therapy may reduce the blood pressure below the autoregulatory range, possibly leading to ischemic events (such as stroke or coronary disease).

**C.** Once the BP is controlled; the patient should be switched to oral therapy, with the diastolic pressure being gradually reduced to 85 to 90 mmHg over two to three months.

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| **Hypertensive Emergencies** |
| Accelerated-malignant hypertension with papilloedema |
| **Cerebrovascular**  Hypertensive encephalopathy  Atherothrombotic brain infarction with severe hypertension  Intracerebral hemorrhage  Subarachnoid hemorrhage |
| **Cardiac**  Acute aortic dissection  Acute left ventricular failure  Acute or impending myocardial infarction  After coronary bypass surgery |
| **Renal**  Acute glomerulonephritis  Renal crises from collagen vascular diseases  Severe hypertension after kidney transplantation |
| **Excessive circulating catecholamines**  Pheochromocytoma crisis  Food or drug interactions with monoamine-oxidase inhibitors  Sympathomimetic drug use (cocaine)  Rebound hypertension after sudden cessation of antihypertensive drugs |
| Eclampsia |
| **Surgical**  Severe hypertension in patients requiring immediate surgery  Postoperative hypertension  Postoperative bleeding from vascular suture lines |
| Severe body burns |
| Severe epistaxis |