

Evaluation and treatment of hypertensive emergencies in adults

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INTRODUCTION AND TERMINOLOGY

— Most patients with significantly elevated blood pressure (systolic pressure ≥ 180 mmHg and/or diastolic pressure ≥ 120 mmHg) have no acute, end-organ injury (so called severe asymptomatic hypertension). Although some propose relatively rapid initiation of antihypertensive therapy in this setting, there may be more risk than benefit from such an aggressive regimen. (See "[Management of severe asymptomatic hypertension \(hypertensive urgencies\) in adults](#)".)

By contrast, some patients with significantly elevated blood pressure have signs or symptoms of acute, ongoing target-organ damage. Such patients have a hypertensive emergency [1]. Hypertensive emergencies can develop in patients with or without known preexisting hypertension [2,3]. In younger (<60 years of age) people, the diastolic pressure is typically ≥ 120 mmHg, but there is no specific threshold since individuals who develop an acute rise in blood pressure can develop symptoms if the previous pressure was normal (such as in a pregnant person who develops eclampsia or a young adult who develops acute glomerulonephritis).

The term "malignant hypertension" entered the medical lexicon in 1928 because, at that time, patients with this condition had a prognosis that was similar to patients with many cancers. However, antihypertensive therapies that can quickly and safely lower blood pressure have improved outcomes [4].

EVALUATION AND DIAGNOSIS

— The history and physical examination in patients presenting with a severely elevated blood pressure (or an acute rise in blood pressure over a previously normal baseline, even if the presenting pressure is <180/120 mmHg) should determine whether any of the following are present [1,5]:

- Acute head injury or trauma

- Generalized neurologic symptoms, such as agitation, delirium, stupor, seizures, or visual disturbances
- Focal neurologic symptoms that could be due to an ischemic or hemorrhagic stroke
- Fresh flame hemorrhages, exudates (cotton-wool spots), or papilledema when direct funduscopy is performed, as these are consistent with grade III or IV hypertensive retinopathy and can rarely be associated with hypertensive encephalopathy
- Nausea and vomiting, which may be a sign of increased intracranial pressure
- Chest discomfort or pain, which may be due to myocardial ischemia or aortic dissection
- Acute, severe back pain, which might be due to aortic dissection
- Dyspnea, which may be due to pulmonary edema
- Pregnancy, as such patients with severe hypertension could have preeclampsia or develop eclampsia ([table 1](#))
- Use of drugs that can produce a hyperadrenergic state, such as cocaine, amphetamine(s), phencyclidine, or monoamine oxidase inhibitors, or recent discontinuation of [clonidine](#) or, less commonly, other antihypertensive agents

In addition, the following tests should be performed to evaluate the presence of target-organ damage in association with targeted clinical symptoms or signs:

- Electrocardiography
- Conventional chest radiography
- Urinalysis, urine pregnancy test (if appropriate)
- Serum electrolytes and serum creatinine
- Cardiac biomarkers (if an acute coronary syndrome is suspected)
- Computed tomography (CT) or magnetic resonance imaging (MRI) of the brain (if head injury, neurologic symptoms, hypertensive retinopathy, nausea, or vomiting are present)
- Contrast-enhanced CT or MRI of the chest or transesophageal echocardiography (if aortic dissection is suspected, although rapid blood pressure lowering need not be delayed in such patients while awaiting the results of imaging)

It is often easiest to categorize hypertensive emergencies by the target organ that is being damaged (eg, brain, heart, kidney). The evaluation above can usually identify the at-risk target organ and can dictate both the target blood pressure and the rapidity with which the target is achieved. (See ['Treatment'](#) below.)

Hypertensive emergencies are uncommon, with an estimated population incidence (based upon large claims databases) of one to two cases per million per year. Various retrospective reviews from adult emergency departments found that hypertensive emergencies account for less than 1 percent of all visits [6-9]. In studies that classified hypertensive emergencies [6,7], eclampsia and aortic dissection were the least common (2 percent), whereas cerebral infarction (39 to 40 percent) and acute pulmonary edema (25 to 32 percent) were the most common, similar to the conclusions of a separate report [10].

TREATMENT

Overall approach to therapy

— Optimal therapy, including the choice of agent and the blood pressure goal, varies according to the specific hypertensive emergency [1,11,12].

It is generally unwise to lower the blood pressure too quickly or too much as ischemic damage can occur in vascular beds that have become habituated with the higher level of blood pressure (ie, autoregulation) [13]. For most hypertensive emergencies, mean arterial pressure should be reduced gradually by approximately 10 to 20 percent in the first hour and by a further 5 to 15 percent over the next 23 hours [12]. This often results in a target blood pressure of <180/<120 mmHg for the first hour and <160/<110 mmHg for the next 23 hours (but rarely <130/<80 mmHg during that time frame).

The major exceptions to gradual blood pressure lowering over the first day are:

- **Acute phase of an ischemic stroke** – The blood pressure is usually **not** lowered unless it is $\geq 185/110$ mmHg in patients who are candidates for reperfusion therapy (table 2) or $\geq 220/120$ mmHg in patients who are not candidates for reperfusion (thrombolytic) therapy [14,15]. (See "Initial assessment and management of acute stroke", section on 'Blood pressure management'.)
- **Acute aortic dissection** – The systolic blood pressure should be **rapidly** lowered to a target of 100 to 120 mmHg (to be attained in 20 minutes) to reduce aortic shearing forces (table 3) [16]. (See "Management of acute type B aortic dissection", section on 'Anti-impulse therapy' and "Overview of acute aortic dissection and other acute aortic syndromes", section on 'Acute medical management' and "Management of acute type A aortic dissection", section on 'Acute management'.)
- **Intracerebral hemorrhage** – Goals of antihypertensive therapy in such patients are variable and are discussed elsewhere. (See "Spontaneous intracerebral hemorrhage: Acute treatment and prognosis", section on 'Blood pressure management'.)

After a suitable period (often 8 to 24 hours) of blood pressure control at target in the intensive care unit, oral medications are usually given and the initial intravenous therapy is tapered and discontinued.

Neurologic emergencies

— Severe hypertension with acute neurologic signs or symptoms is usually the most complicated and difficult clinical scenario as the differential diagnosis includes varied conditions that have disparate treatments, only some of which routinely involve lowering the blood pressure:

- Ischemic stroke – Management of blood pressure in patients with acute ischemic stroke, including the preferred antihypertensive medications in this setting, is discussed in detail elsewhere. (See ["Initial assessment and management of acute stroke"](#), section on 'Blood pressure management'.)
- Hemorrhagic stroke – Management of blood pressure in patients with spontaneous intracerebral hemorrhage and subarachnoid hemorrhage is complicated by competing risks (eg, reducing cerebral perfusion) and benefits (eg, reducing further bleeding) [17]. Intravenous [labetalol](#) and [nicardipine](#) are most often used as first-line agents [18], although shorter-acting drugs might be more advantageous. The management of blood pressure in patients with hemorrhagic stroke is discussed in detail elsewhere. (See ["Spontaneous intracerebral hemorrhage: Acute treatment and prognosis"](#), section on 'Blood pressure management' and ["Aneurysmal subarachnoid hemorrhage: Treatment and prognosis"](#), section on 'Blood pressure control'.)
- Head trauma – Head trauma with increased intracranial pressure can produce severe elevations in blood pressure. Hypertension is usually treated in this setting only if the cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) is >120 mmHg and the intracranial pressure is >20 mmHg. Extreme caution is recommended regarding the intensity of treatment as aggressive reductions in blood pressure are associated with worse outcomes. (See ["Evaluation and management of elevated intracranial pressure in adults"](#), section on 'Blood pressure control'.)
- Hypertensive encephalopathy – In contrast to stroke and head trauma, the signs and symptoms of hypertensive encephalopathy (eg, headache, confusion, nausea, vomiting) usually abate after the blood pressure is lowered (see ["Moderate to severe hypertensive retinopathy and hypertensive encephalopathy in adults"](#)). In fact, hypertensive encephalopathy is most often a diagnosis of exclusion, confirmed retrospectively when the mental status improves after the blood pressure is lowered into the autoregulatory range. Thus, patients with suspected hypertensive encephalopathy should have their blood pressure lowered by approximately 10 to 20 percent during the first hour of treatment. However, additional lowering should be gradual such that, compared with the initial blood pressure upon presentation, the pressure is reduced by no more than 25 percent at the end of the first day of treatment. Commonly used medications in this setting include [clevidipine](#), [nicardipine](#), [fenoldopam](#), and [nitroprusside](#).

Cardiac emergencies

— The most common cardiac emergencies associated with severely elevated blood pressure are acute left ventricular dysfunction with pulmonary edema and acute coronary syndrome (including acute myocardial infarction):

- Acute heart failure – Patients with acute left ventricular dysfunction and pulmonary edema should usually receive loop diuretics. A vasodilator that is easy to titrate (eg, sodium [nitroprusside](#), [nitroglycerin](#)) is often added to reduce afterload. Drugs that increase cardiac work (eg, [hydralazine](#)) or acutely decrease cardiac contractility (eg, [labetalol](#) or other beta blocker) should be avoided [19]. The goal of these therapies is amelioration of volume excess and heart failure and improvement in pulmonary edema, which can often be achieved with a 10 to 15 percent reduction in blood pressure. (See "[Treatment of acute decompensated heart failure: General considerations](#)".)
- Acute coronary syndrome – Severe hypertension associated with an acute coronary syndrome (including acute myocardial infarction) is appropriately treated with intravenous [nitroglycerin](#), [clevidipine](#), [nicardipine](#), or intravenous [metoprolol](#) or [esmolol](#) (to reduce myocardial oxygen consumption, reduce the underlying coronary ischemia, and improve prognosis) [20]. (See "[Overview of the acute management of ST-elevation myocardial infarction](#)" and "[Overview of the acute management of non-ST-elevation acute coronary syndromes](#)".)

Vascular emergencies

— Vascular emergencies include acute aortic dissection and severe hypertension in patients who have recently undergone vascular surgery:

- Acute aortic dissection – Patients with acute aortic dissection are treated to rapidly reduce the blood pressure to a goal systolic of 100 to 120 mmHg within approximately 20 minutes of diagnosis ([table 3](#)), although this target is not based upon clinical trial evidence. An intravenous beta blocker is given first (usually [esmolol](#), but [labetalol](#), [propranolol](#), and [metoprolol](#) can also be used) to reduce the heart rate below 60 beats per minute and the shear stress on the aortic wall [21]. In addition, a vasodilator (often [nitroprusside](#) or [clevidipine](#)) is typically required to quickly achieve the goal blood pressure. (See "[Management of acute type B aortic dissection](#)", section on 'Anti-impulse therapy' and "[Overview of acute aortic dissection and other acute aortic syndromes](#)", section on 'Acute medical management' and "[Management of acute type A aortic dissection](#)", section on 'Acute management'.)
- Severe hypertension in patients with recent vascular surgery – Severe elevations of blood pressure can threaten suture lines, and therefore, such patients are often treated with rapidly acting intravenous antihypertensive agents in an intensive care unit setting. Although this is common practice, no controlled studies have proven the benefit of this intervention.

Kidney emergencies

— Severe hypertension may occasionally cause acute injury to the kidneys (acute hypertensive nephrosclerosis, formerly called "malignant nephrosclerosis"). This condition is characterized by hematuria (usually microscopic hematuria, which is found in approximately 75 percent of patients with

hypertensive emergencies) [22] and an elevated serum creatinine. It is important to determine whether these findings are recent since they may predate the severe blood pressure elevation in some patients.

When kidney injury occurs as a result of severe hypertension, the common pathologic findings include fibrinoid necrosis of small arterioles and "onion skinning" of small renal arteries. The histologic changes are indistinguishable from other forms of the hemolytic uremic syndrome (picture 1A-D); however, a kidney biopsy is seldom performed in such patients. The renal vascular disease leads to glomerular ischemia and activation of the renin-angiotensin system, possibly resulting in exacerbation of the hypertension.

Antihypertensive therapy often leads to worsening kidney function; this decline in kidney function may be worse in patients with high-grade ostial stenosis of one or both renal arteries. Although this acute kidney injury sometimes requires dialysis, the reduction in kidney function may be reversed with long-term blood pressure control [23]. By contrast, [fenoldopam](#) is associated with a temporary improvement in kidney function and is therefore a useful antihypertensive agent in patients with kidney hypertensive emergencies [24]. (See "[Drugs used for the treatment of hypertensive emergencies](#)".)

Sympathetic overactivity resulting in hypertensive emergencies

— Four causes of sympathetic overactivity can lead to severe elevations of blood pressure and acute target-organ damage:

- Withdrawal of short-acting antihypertensive agents (especially [clonidine](#), [propranolol](#), or other beta blockers) can be associated with severe hypertension and may mimic the signs and symptoms of pheochromocytoma. Typically, reinstitution of the recently discontinued drug will lower the blood pressure. Oral clonidine will begin to lower blood pressure within an hour; however, some beta blockers take much longer to lower the blood pressure and, therefore, short-acting intravenous medications are often required while waiting for the reinstituted beta blocker to achieve an effect. (See "[Withdrawal syndromes with antihypertensive drug therapy](#)".)
- Ingestion of sympathomimetic agents (eg, tyramine-containing foods in patients who take chronic monoamine oxidase inhibitors [11], amphetamine-like compounds, cocaine, etc) can precipitate severe hypertension and end-organ damage. Such patients can be treated with intravenous [phentolamine](#) or, if phentolamine is unavailable, [nitroprusside](#) [25]. (See "[Clinical manifestations, diagnosis, and management of the cardiovascular complications of cocaine abuse](#)" and "[Cocaine: Acute intoxication](#)" and "[Methamphetamine: Acute intoxication](#)" and "[Acute amphetamine and synthetic cathinone \("bath salt"\) intoxication](#)".)
- Pheochromocytoma can also produce severe hypertension and acute target-organ damage. The treatment of hypertension in pheochromocytoma is discussed separately. (See "[Treatment of pheochromocytoma in adults](#)".)

- Severe autonomic dysfunction (eg, Guillain-Barré and multiple system atrophy syndromes or acute spinal cord injury) is occasionally associated with hypertensive emergency. Such patients can be treated with [phentolamine](#), [nitroprusside](#), [labetalol](#), or other agents. (See "[Chronic complications of spinal cord injury and disease](#)", section on 'Cardiovascular complications' and "[Guillain-Barré syndrome in adults: Treatment and prognosis](#)", section on 'Cardiovascular and autonomic status'.)

Unless a beta blocker was recently withdrawn, administration of a beta blocker alone is **contraindicated** in these settings since inhibition of beta receptor-induced vasodilation can result in unopposed alpha-adrenergic vasoconstriction and a further rise in blood pressure [26]. (See "[Clinical presentation and diagnosis of pheochromocytoma](#)".)

Hypertensive emergencies during pregnancy

— [Hydralazine](#) and [labetalol](#) have been widely used in pregnant people with severe hypertension, which is usually due to preeclampsia or exacerbation of preexistent hypertension. [Fenoldopam](#) and [nicardipine](#) have also been used.

These issues as well as antihypertensive drugs that are contraindicated in pregnancy are discussed in detail separately. (See "[Treatment of hypertension in pregnant and postpartum patients](#)", section on '[Acute therapy of severe hypertension](#)'.)

ANTIHYPERTENSIVE DRUGS

— An overview of the mechanism of action, doses, and routes of administration of many antihypertensive drugs that are often used for hypertensive emergency is presented elsewhere. (See "[Drugs used for the treatment of hypertensive emergencies](#)".)

FOLLOW-UP

— Secondary causes of hypertension are more common in patients who have a hypertensive emergency compared with other hypertensive populations [27]. Thus, patients with a hypertensive emergency should be evaluated for common forms of secondary hypertension and, if there are suggestive clinical clues, less common causes of secondary hypertension. Patients should be reminded that adherence to long-term antihypertensive drug therapy can reduce the risk of recurrent hospitalization for hypertensive emergencies.

The most important aspect of care for the patient with a hypertensive emergency is assuring that high-quality outpatient follow-up is available as many of the presenting problems (including dialysis for end-stage kidney disease) can improve greatly if the blood pressure is well controlled in the long term. A large proportion of patients without follow-up will return to the hospital with a repeated hypertensive emergency [2].

SOCIETY GUIDELINE LINKS

— Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "[Society guideline links: Hypertension in adults](#)".)

INFORMATION FOR PATIENTS

— UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics links (see "[Patient education: High blood pressure emergencies \(The Basics\)](#)")

SUMMARY AND RECOMMENDATIONS

- **Definition** – Most patients with significantly elevated blood pressure (systolic pressure ≥ 180 mmHg and/or diastolic pressure ≥ 120 mmHg) have no acute, end-organ injury (so called severe asymptomatic hypertension). However, some patients with significantly elevated blood pressure have signs or symptoms of acute, ongoing target-organ damage. Such patients have a hypertensive emergency. (See '[Introduction and terminology](#)' above.)
- **Evaluation** – The history and physical examination in patients presenting with a severely elevated blood pressure (or an acute rise in blood pressure over a previously normal baseline, even if the presenting pressure is $<180/120$ mmHg) should seek to identify signs and symptoms of acute target-organ damage; in addition, certain laboratory and, in some settings, imaging studies may be needed. (See '[Evaluation and diagnosis](#)' above.)

Common findings that raise concern for acute target organ damage include:

- Acute head injury or trauma
- Generalized neurologic symptoms (eg, agitation, delirium, seizures, visual disturbances)
- Focal neurologic symptoms
- Fresh flame hemorrhages, exudates (cotton-wool spots), or papilledema
- Nausea and vomiting

- Chest discomfort or pain
- Acute, severe back pain
- Dyspnea
- Pregnancy
- Use of drugs that can produce a hyperadrenergic state
- **Acute management** – Optimal therapy, including the choice of agent and the blood pressure goal, varies according to the specific hypertensive emergency. It is generally unwise to lower the blood pressure too quickly or too much, as ischemic damage can occur in vascular beds that have reset their autoregulatory threshold to the higher level of blood pressure. For most hypertensive emergencies, mean arterial pressure should be reduced by approximately 10 to 20 percent in the first hour and then gradually during the next 23 hours so that the final pressure is reduced by approximately 25 percent compared with baseline. (See ["Treatment"](#) above.)

The major exceptions to modest and gradual blood pressure lowering over the first 24 hours are:

- Acute phase of an ischemic stroke – The blood pressure is usually **not** lowered unless it is $\geq 185/110$ mmHg in patients who are candidates for reperfusion therapy ([table 2](#)) or $\geq 220/120$ mmHg in patients who are not candidates for reperfusion therapy. (See ["Initial assessment and management of acute stroke"](#), section on ["Blood pressure management"](#).)
- Acute aortic dissection – The systolic blood pressure is **rapidly** lowered to a target of 100 to 120 mmHg (to be attained in 20 minutes). (See ["Management of acute type B aortic dissection"](#), section on ["Anti-impulse therapy"](#) and ["Overview of acute aortic dissection and other acute aortic syndromes"](#), section on ["Acute medical management"](#) and ["Management of acute type A aortic dissection"](#), section on ["Acute management"](#).)
- Spontaneous hemorrhagic stroke – The systolic blood pressure can be rapidly reduced if no contraindications exist. Therapy in such patients is discussed elsewhere. (See ["Spontaneous intracerebral hemorrhage: Acute treatment and prognosis"](#), section on ["Blood pressure management"](#).)

A variety of intravenous (IV) antihypertensive drugs can be used to treat hypertensive emergencies; an overview of the mechanism of action, doses, and routes of administration of these agents is presented elsewhere. (See ["Drugs used for the treatment of hypertensive emergencies"](#).)

- **Postacute management**

- Conversion from IV to oral therapy – After a suitable period (often 8 to 24 hours) of blood pressure control at target in an intensive care unit, oral medications are usually given and the initial intravenous therapy is tapered and discontinued. (See ["Overall approach to therapy"](#) above.)

- Secondary causes of hypertension are more common in patients who have a hypertensive emergency compared with other hypertensive populations. Thus, patients with a hypertensive emergency should be evaluated for common forms of secondary hypertension and, if there are suggestive clinical clues, less common causes of secondary hypertension. (See 'Follow-up' above.)
- The most important aspect of care for the patient with a hypertensive emergency is assuring that high-quality outpatient follow-up is available. A large proportion of patients without follow-up will return to the hospital with a recurrent hypertensive emergency. (See 'Follow-up' above.)

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