



Recognizing Hypertensive Crises

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Arterial hypertension affects more than 20% of the adult population. Despite this significant figure, however, in Canada, only 56% of hypertensive patients are recognized as having arterial hypertension and only 16% receive appropriate treatment.¹ While hypertension is a chronic disease that evolves slowly, a hypertensive crisis — whether a hypertensive emergency or a hypertensive “urgency” — is a rare occurrence encountered primarily by first-line physicians in either emergency room (ER) or clinical settings.

Definitions

The World Health Organization (WHO) and the Joint National Committee VI have broken down arterial hypertension into three grades, according to the degree of blood pressure (BP) elevation (Table 1).^{2,3} The grade scale was established according to the risk of macro- and microvascular complications associated with BP values. It, therefore, constitutes a prognostic assessment tool that helps determine how aggressive treatment should be.

A hypertensive crisis is defined as a significant increase in BP, whether associated with acute injury to target organs or not. Although a hyper-

tensive crisis is not strictly defined by a BP value, it is often associated with a diastolic BP > 120 mmHg.

A hypertensive crisis can be classified as a hypertensive emergency or a hypertensive urgency. A hypertensive crisis is considered an emergency if the hypertension results in acute injury to target organs (retina, heart, kidney or brain), or an urgency if there is no major damage to target organs. This distinction is absolutely essential to appropriate case management, as it is the degree of damage to the target organs and not the absolute BP level that will determine how aggressive the initial approach should be. In certain patients who present with a hypertensive urgency without underlying chronic hypertension (pre-eclampsia), severe target-organ damage may occur at much lower BP levels than in patients with a long history of hypertension — even with BP levels as low as 160/100 mmHg.⁴

Malignant hypertension (defined by the presence of papilledema) and accelerated hypertension (defined by the presence of hemorrhaging or fundic exudates) are true hypertensive emergencies. The distinction between the two is essentially historical, as their prognosis is identical. The

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term accelerated malignant hypertension is sometimes used.

Causes of Hypertensive Crises

While a hypertensive crisis may be the initial sign of essential or secondary hypertension, it most often results from the exacerbation of essential hypertension that has gone undiagnosed or that has not been treated appropriately.

A secondary cause must always be sought, as 40% of cases of hypertensive crises have an identifiable cause, as compared with approximately 5% of cases of chronic hypertension. The prevalence of secondary causes is apparently higher in Caucasians than in people of African origin. In Caucasians, 70% of hypertensive crises have an identifiable cause, whereas in people of African origin, the exacerbation of essential hypertension accounts for nearly 80% of hypertensive crises. Among the most common secondary causes are renal parenchymal disorders, renovascular disease (which accounts for approximately one-third of the secondary causes of hypertensive crises), endocrinal factors, and pharmaceutical factors (including drug use) (Table 2).



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Differential Diagnosis

A differential diagnosis for hypertensive crises must include short-term asymptomatic elevations in BP (usually inconsequential), which may coincide with anxiety or panic attacks or occur post-operatively. Lastly, especially in elderly patients, pseudo-hypertension may be caused by vascular calcification, in which case the sphygmomanometer overestimates systemic BP. In such cases, the Osler's maneuver can be used. When the cuff is inflated beyond systolic BP, if the radial artery can be palpated despite sensitivity, pseudo-hypertension resulting from arterial calcification is likely present.

The Clinical Aspect of Hypertensive Crises

Less than 1% of chronic hypertensive patients will experience a hypertensive crisis, which generally occurs at an average age of 50.⁵ Twice as many men as women present with the condition. The risk factors for hypertensive crisis are noncompliance with antihypertensive treatment, alcohol and/or drug abuse, the use of oral contraceptives (though the correlation is uncertain), underprivileged socioeconomic conditions, and tobacco use.

Clinical signs. There are several clinical signs of hypertensive emergency.⁶



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Table 1

Classification of Arterial Hypertension for Adults Aged 18 and Over

Stage	Blood Pressure		
	Systolic		Diastolic
Optimal BP	< 120 mmHg	and	< 80 mmHg
Normal BP	< 130 mmHg	and	< 85 mmHg
High-normal BP	130 to 139 mmHg	or	85 to 89 mmHg
Grade 1 hypertension	140 to 159 mmHg	or	90 to 99 mmHg
Grade 2 hypertension	160 to 179 mmHg	or	100 to 109 mmHg
Grade 3 hypertension	≥ 180 mmHg	or	≥ 110 mmHg

BP = blood pressure

Adapted from Guidelines Subcommittee: 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension." *J Hypertens* 1999; 17:151; and from Joint National Committee: *The Sixth Report of the Joint National Committee on Prevention, Evaluation, and Treatment of High Blood Pressure*. *Arch Intern Med* 1997; 157-2413.

Hypertension with variable BP readings, ranging from values as low as 150/100 mmHg, may occur in patients without any history of arterial hypertension. In patients with a known history of hypertension, however, a systolic BP ranging from 200 mmHg to 250 mmHg and diastolic BP from 130 mmHg to 180 mmHg are frequent.

Stage III retinopathy on the Keith-Wagener-Barker scale (hemorrhaging and exudate) or Stage IV retinopathy (papilledema) often occurs. A vision problem may be a symptom of hypertensive emergency in 35% to 60% of patients.

Acute heart failure can also be a symptom of hypertensive emergency in 11% of patients. Although much less common, myocardial ischemia or aortic dissection may also occur.

Lastly, left ventricular hypertrophy (present in 75% of patients suffering from a hypertensive emergency) suggests chronic underlying hypertension.

Nonspecific neurological symptoms (*i.e.*, headache or dizziness) or more specific neurological symptoms (*i.e.*, stroke) may appear in

patients suffering from hypertensive emergency. It is important to recognize hypertensive encephalopathy — a particular neurological symptom that requires prompt antihypertensive treatment. This type of encephalopathy normally manifests itself in the form of headaches, nausea, vomiting, vision problems, focal neurological lesions, convulsions, and impairment of consciousness. It develops within a period ranging from 48 to 72 hours. Hypertensive encephalopathy is induced by impairment of the autoregulation of the brain, leading to vasodilatation and cerebral edema. Magnetic resonance imaging (MRI) sometimes reveals an image that is quite characteristic of posterior leucoencephalopathy affecting the white matter of the parietal and occipital lobes. The condition is potentially reversible with appropriate treatment.

Renal damage may be signaled by anything from slight proteinuria to nephrotic syndrome. In nearly 30% of cases, acute renal failure is apparent at the start of the hypertensive crisis, in which case the prognosis is more serious. The

Table 2

The Causes of Hypertensive Crises

Acute increase in BP: exacerbation of essential chronic hypertension (frequent)

Parenchymal renal disorders

- Glomerulonephritis
- Tubulointerstitial nephritis
- Thrombotic thrombocytopenic purpura
- Hemolytic uremic syndrome
- Systemic diseases involving renal damage (*i.e.*, scleroderma, diabetes, systemic lupus erythematosus [SLE], vasculitis)
- Congenital diseases (*i.e.*, renal aplasia)

Drug ingestion

- Sympathomimetics (*i.e.*, cocaine, amphetamines, PCP, LSD, ephedrine)
- Withdrawal from clonidine or beta blockers
- Interaction with monoamine oxidase inhibitors
- Tricyclic antidepressants
- Cyclosporine
- Erythropoietin

Tumors

- Renal carcinoma
- Wilms' tumor
- Lymphoma

BP = blood pressure; LSD = lysergic acid diethylamide; PCP = phencyclidine
Adapted from Kaplan, NM, Management of hypertensive emergencies. Lancet 1994; 344:1335.

Preeclampsia and eclampsia

Renovascular disorders

- Atherosclerosis
- Fibromuscular dysplasia
- Arteritis

Endocrine disorders

- Pheochromocytoma
- Primary hyperaldosteronism
- Cushing's syndrome
- Renin-secreting tumor

Neurological disorders

- Stroke
- Cranial trauma
- Sympathetic hyperreactivity after a spinal-cord lesion (Guillain-Barré syndrome) or intermittent acute porphyria

Coarctation of the aorta

condition may be reversible, however, even though dialysis may be required. It is recommended that such patients wait a year before considering a kidney transplant. From a metabolic perspective, hypokalemia secondary to activation of the renin-angiotensin-aldosterone (RAA) system is frequent. Since the renin and aldosterone levels are elevated for several

months when a hypertensive crisis occurs, the physician must be cautious about diagnosing hyperaldosteronism. Metabolic alkalosis also may occur.

Other possible clinical signs include microangiopathic hemolytic anemia, increased sedimentation rate, asthenia, weight loss or nonspecific symptoms.

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Case Management

Initial assessment. The initial assessment of a patient in hypertensive crisis must focus on acute dysfunction of the target organs (kidneys, heart, brain, retina). The distinction between a hypertensive emergency and a hypertensive urgency is absolutely essential. The clinician must, therefore, examine the patient's history for diagnosis and treatment of chronic hypertension and determine the duration, severity and control of the condition, compliance with treatment and any chronic damage to the target organs. He/she must also determine whether the patient is taking any substances that could raise BP, such as cocaine, phencyclidine (PCP), lysergic acid diethylamide (LSD) or amphetamines, or any medications such as sympathomimetics (decongestants), nonsteroidal anti-inflammatories (NSAIDs) or contraceptives. Moreover, the clinician must look for acute and chronic target-organ damage.

Physical examination. The physical examination must include a BP reading taken according to standard procedures, after a rest period, with a cuff suited to the patient's weight. The reading must be taken at heart level in both arms.¹ The BP must be taken with the patient lying down and standing in order to rule out orthostatic hypotension that could exist because of dehydration or pheochromocytoma. A detailed eye examination (carried out by an ophthalmologist if necessary) and cardiopulmonary, neurological and vascular examinations are essential.

Laboratory tests. Laboratory tests must be ordered in emergency situations. They should include a complete blood count and blood smear; electrolyte, creatinine, glucose and lactic dehydrogenase (LDH) levels; urinalysis; electrocardiogram (ECG); chest X-ray; and measurement of catecholamines in the urine. Additional examinations may be required, according to the clinical situation (*i.e.*, brain CT scan if there is evidence of neurological damage).⁷

Hypertensive Emergency

In the case of a hypertensive emergency (*i.e.*, with acute damage to target organs), the approach must be aggressive, and immediate efforts must be made to lower BP (even before the laboratory results are received, with the exception of ECG results). Medication would be administered preferably intravenously for rapid action (*i.e.*, less than one hour), and should be chosen in light of organ damage and comorbidity factors (Table 3). The goal should be to reduce average BP by 25% or achieve a diastolic BP of 100 mmHg to 110 mmHg within a few hours (according to the nature of the target-organ damage). The patient should also be admitted to intensive care and BP monitored *via* an arterial cannula.

Hypertensive Urgency

In the case of hypertensive urgency (*i.e.*, without acute damage to target organs), the approach should be less aggressive. The patient should be placed initially in a dark, quiet room for complete bed rest. BP should be taken every 30 minutes for a few hours. Treatment, preferably by mouth, could be commenced if BP readings remain high. BP should also be monitored after medication is administered to ensure a therapeutic response. If there is no response, hospitalization is recommended. In cases of hypertensive urgency, the goal should be to achieve a 20% reduction in average BP or a diastolic BP lower than 120 mmHg in 24 hours rather than a normal BP. If the therapeutic response in the emergency setting is favorable, the patient could be discharged if close out-patient monitoring with 24 or 48 hours is available.

Table 3

Agents of Choice in the Treatment of Hypertensive Crises

Disorder	Agents of Choice	Comments
Hypertensive encephalitis	Nitroprusside, labetalol	Avoid clonidine, methyldopa and diazoxide.
Stroke	Nitroprusside, labetalol, nimodipine	The benefits of these agents in lowering BP are uncertain. Avoid vasodilators* and clonidine.
Aortic dissection	Nitroprusside and beta blockers (propranolol or esmolol), labetalol, verapamil	Lowering of BP to minimum levels is tolerated. Avoid vasodilators.*
Left heart failure	Nitroprusside, nitroglycerin, enalaprilat, furosemide, morphine	Avoid labetalol, esmolol, hydralazine and diazoxide.
Coronary heart disease	Nitroglycerin, nitroprusside, labetalol, esmolol	Gradual lowering of BP. Avoid vasodilators.*
Eclampsia	Hydralazine, labetalol	Avoid diuretics, nitroprusside (unless condition is refractory) and ACE inhibitors.
Catecholaminergic excess**	Nitroprusside, phentolamine, labetalol	Avoid diuretics.
Renal failure	Hydralazine, labetalol	Avoid beta blockers and ACE inhibitors.

* Vasodilators that induce reflex sympathetic activation: hydralazine, diazoxide, minoxidil, short-duration nifedipine

** Catecholaminergic excess: pheochromocytoma; cocaine, amphetamine, LSD or PCP intoxication; crisis induced by monoamine oxidase inhibitors; cranial trauma; spinal cord lesions; etc.

Seeking Secondary Causes

As soon as the patient is stabilized, secondary causes of hypertension must be sought (Table 2). An assessment of renovascular and endocrine disorders (particularly pheochromocytoma) should be carried out on all patients. Remember that certain drugs may interfere with laboratory analyses. Consequently, tests must be ordered at the time of initial assessment and medication should be selected to avoid drug interaction. When considering drug interactions, the possibility of a false-

positive diagnosis of pheochromocytoma should be examined following the use of labetalol. Similarly, a false-negative diagnosis of renovascular disease should be considered following administration of a drug that affects the RAA system.

Treatment

The choice of medication used to treat a hypertensive crisis depends primarily on the type of acute damage to target organs and the comorbidity factors present. Certain drugs should be avoided in specific situations because of their pharmacodynamic

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effects. Clonidine and alpha-methyldopa should be avoided if hypertensive encephalopathy is present, as they create an increased risk of drowsiness. Similarly, diazoxide, hydralazine and minoxidil are contraindicated in the case of coronary ischemia or aortic dissection because they cause significant activation of the sympathetic nervous system.

In chronic hypertensive patients, autoregulation adjusts upwards, which in turn produces relative cerebral ischemia when BP is reduced too aggressively. Lowering of BP should, therefore, never exceed 25% in the acute phase. In the same perspective, lower doses of antihypertensive drugs should be used with elderly patients or patients with a history of carotid or coronary atherosclerosis.

When a hypertensive urgency is caused by a reversible disorder, such as anxiety or major pain, the treatment of the reversible disorder should take precedence over the treatment of hypertension.

Intravenous agents. Sodium nitroprusside is an extremely effective and powerful arterial and venous vasodilator. It is indicated in most hypertensive emergencies, with the exception preeclampsia, as it crosses the placental barrier. The normal dosage is 0.25 to 3 µg/kg/minute intravenously (50 mg/250 cc at 5 to 60 cc/h per 70 kg), and the maximum dosage is 10 µg/kg/minute for 10 minutes. The drug acts immediately and its duration of action is one to two minutes. It is, therefore, rapidly reversible should BP be lowered too radically. With this drug, monitoring in intensive care and BP monitoring *via* an arterial cannula is required. The side effects include nausea, vomiting and hypotension.

Thiocyanate and cyanide poisoning are also possible, especially if renal or hepatic failure occurs, or with a high dosage or prolonged infusion (more than 72 hours). The symptoms of thiocyanate poisoning are confusion, vision problems, tinnitus, hyperreflexia, and paradoxical hypertension. Thiocyanate poisoning is nevertheless rare. A

dose of thiocyanate can be administered in a case of prolonged infusion (target level: < 1.7 mmol/L). Cyanide poisoning is very rare if the total dosage is below 300 mg. It can be treated with amyl nitrite, sodium thiosulfate or sodium nitrate.

Labetalol is a nonselective alpha- and beta blocker with vasodilating properties. It is indicated in the treatment of coronary heart disease or catecholaminergic excess. It is contraindicated, however, in cases of symptomatic heart failure, second- and third-degree atrioventricular block, severe bradycardia, and bronchospasm. The usual dosage is 0.25 mg/kg to 0.5 mg/kg (or 20 mg to 80 mg) intravenous bolus every 10 to 15 minutes (maximum 300 mg), or 0.5 mg/minute to 2 mg/minute by continuous infusion. Labetalol begins to take effect in 5 to 10 minutes, and its duration of action is three to six hours. The primary side effects are nausea, pruritus, dizziness, and hot flashes.⁸ Paradoxical hypertension has been reported in cases of pheochromocytoma. The use of labetalol does not require BP monitoring by means of an arterial cannula.

Nitroglycerin is a venous vasodilator indicated especially in the treatment of heart failure coronary or heart disease. It is generally contraindicated in cases of stroke, as it can cause intracranial hypertension. The normal dosage is 5 µg/minute to 300 µg/minute intravenously. Nitroglycerin begins to take effect in 2 to 5 minutes, and its duration of action is from 3 to 5 minutes. The primary side effects are headache, nausea, dizziness and methemoglobinemia. Pharmacological tolerance to nitroglycerin may also develop.

Phentolamine is a nonselective alpha-adrenergic receptor blocker. It is indicated in the treatment of catecholaminergic excess (pheochromocytoma, cocaine use) and contraindicated in the treatment of coronary heart disease. The dosage is from 5 mg to 20 mg intravenous bolus at 5- to 15-minute intervals. Phentolamine begins to take effect in

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one to two minutes and its duration of action is 10 to 30 minutes. Side effects are tachycardia, nausea, abdominal pain and hot flashes. Nitroprusside seems as effective as phentolamine in the treatment of catecholaminergic excess.⁹

Enalaprilat is an angiotensin II converting enzyme (ACE) inhibitor. It is especially indicated in the treatment of heart failure or sclerodermic crisis, but is contraindicated in the treatment of renal failure, bilateral stenosis of the renal arteries and during pregnancy. The usual dosage is 1.25 mg to 5 mg intravenously for 5 minutes every six hours. Enalaprilat starts taking effect in 15 minutes and its duration of action is six to 12 hours. Side effects are hypotension (especially if hypovolemia is present), renal failure, hyperkalemia and angioedema.

Hydralazine is a direct arterial vasodilator. It is especially indicated in the treatment of preeclampsia/eclampsia and contraindicated in the treatment of heart failure, coronary ischemia, and aortic dissection. The usual dosage is 5 mg to 10 mg intravenously or by intramuscular injection every 20 minutes (to a maximum of 50 mg). Hydralazine begins to take effect in 10 to 20 minutes and its duration of action is three to six hours. The side effects are reflex tachycardia, water retention, nausea, diaphoresis, intracranial hypertension, headaches, local phlebitis, and even fetal distress.

Other drugs, such as the following, are not of significant use in the treatment of hypertensive crises:

- Intravenous dioxide is an arterial vasodilator, but is of little use.⁹
- Diuretics are of little use in treating acute hypertensive crises, as hypovolemia is already present.
- Intravenous nicardipine, intravenous fenoldopam and intravenous urapidil are not available in Canada.

Oral agents. Clonidine is a central alpha₂-adrenergic antagonist that diminishes sympathetic

activation. It is contraindicated in the presence of hypertensive encephalopathy for the reasons mentioned previously. The usual dosage is 0.1 mg to 0.2 mg hourly, as required (to a maximum of 0.7 mg). Clonidine begins to take effect in 30 to 60 minutes, and its duration of action is six to eight hours. Side effects are drowsiness, confusion, dryness of the mouth, bradycardia, severe hypotension, and rebound hypertension when usage is discontinued suddenly.

Labetalol's mechanism of action and side effects of labetalol are described in the section on intravenous agents. The oral dosage is 200 mg hourly, as required, to a maximum of 1,200 mg per day. Labetalol begins to take effect in one to three hours, and its duration of action is six to eight hours.

Captopril is an ACE inhibitor. Like enalaprilat, it is contraindicated in cases of renal failure, bilateral stenosis of the renal arteries, and during pregnancy. The usual dosage is 6.25 mg to 50 mg every eight hours. Captopril starts to take effect in 15 to 30 minutes, and its duration of action is four to six hours. Side effects are hypotension (especially if hypovolemia is present), renal failure, hyperkalemia and angioedema.

Calcium channel blockers, such as short-duration nifedipine (sublingual or oral), are no longer recommended for the treatment of hypertensive urgency, given the risk of severe hypotension and secondary cerebral ischemia associated with it.¹⁰ Long-acting or time-released nifedipine or second-generation dihydropyridines (amlodipine or nicardipine) may be useful, however, especially in the presence of renal failure. Further study results are needed before firm recommendations can be made. Verapamil may be used in cases of aortic dissection if beta blockers are contraindicated.

Other drugs, such as prazosine, minoxidil, hydralazine, alpha-methyldopa, phenoxybenzamine, losartan and valsartan have been used in the

treatment of hypertensive urgency, but insufficient studies have been carried out to date to allow for a recommendation of these agents.

Treating Associated Disorders

Hypertensive encephalopathy. The drug of choice for this complication is nitroprusside. The treatment goal should be to reduce average BP by 20% in one to three hours. Rapid neurological recovery should be noted. Otherwise, the diagnosis should be questioned.

Stroke. Optimal BP in stroke cases has yet to be determined.¹¹ It is established that arterial hypertension in stroke victims is likely a reflex to maintain adequate cerebral perfusion. Too rapid a drop in BP may be associated with increased morbidity. Thus, in the case of ischemic stroke, it is suggested that BP be lowered if systolic BP is > 220 mmHg or if average BP is > 130 mmHg, with a target of diastolic BP of around 100 mmHg.¹² In the case of ischemic stroke, BP should be corrected before any thrombolytic agents are administered.¹³

If intracerebral hemorrhaging occurs, arterial hypertension should be treated if systolic BP is \geq 180 mmHg or if diastolic BP is \geq 105 mmHg after two consecutive readings.¹⁴

In the case of a subarachnoid hemorrhage, nimodipine is the agent of choice. The target BP should be approximately 170/100 mmHg to 180/100 mmHg. All of these suggestions are based on the opinions of experts; a number of studies are under way to clarify how to approach the treatment of hypertensive emergencies when neurological damage is a factor.¹¹⁻¹⁴

Aortic dissection is a rather special complication, as it requires a rapid and substantial lowering of blood pressure (within 15 minutes). Systolic BP must be brought down to between 100 mmHg

and 110 mmHg. The first few hours are critical, as the mortality rate is as high as 1% to 2% per hour.¹⁵ Drugs that induce reflex sympathetic activation must be avoided in cases of aortic dissection. A consultation with a vascular surgeon is essential, and an immediate assessment should be performed by means of transesophageal echocardiography, CT-scan, MRI or arteriography.

Adrenergic crisis. With complications such as pheochromocytoma, cocaine, amphetamine or LSD intoxication, clonidine withdrawal, interactions with monoamine oxidase inhibitors, or in cases of spinal cord injury, arterial hypertension is caused by adrenergic hyperreactivity. The agent of choice is phentolamine. Nitroprusside seems to be equally effective, however, in lowering BP. Labetalol has also been used, but there are reported cases of paradoxical elevation of hypertension in cases of pheochromocytoma.⁹ The use of beta blockers without prior use of an alpha-receptor blocker is contraindicated, as it could result in excessive alpha-activation (causing vasoconstriction), leading to paradoxical BP elevation. In the case of clonidine withdrawal, treatment should consist of resuming administration of the drug to allow for slow weaning.

Acute renal failure. Beta blockers and ACE inhibitors are contraindicated in cases of acute renal failure. Minoxidil may be useful. Since it creates significant water and sodium retention and sympathetic activation, however, it should be combined with a beta blocker. In cases of terminal renal failure with substantial nephrogenic edema, dialysis or loop diuretics could become the treatment of choice for arterial hypertension.

Preeclampsia, or gestational hypertension with proteinuria, is another rather special disorder. Cases of hypertensive urgency in this context are considered situations that require immediate treatment, preferably with hydralazine, labetalol or nifedipine, with a target BP of 90 mmHg to

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100 mmHg. Childbirth is the definitive means of resolving preeclampsia, and a multidisciplinary approach is needed. A consensus was recently published in order to standardize the treatment of this condition.¹⁶

Prognosis

The prognosis of patients who present with a hypertensive crisis has greatly improved over the years thanks to modern pharmacological treatments. In 1939, the survival rates at one and five years for patients with malignant hypertension were only 21% and less than 1%, respectively.¹⁷ In 1995, the survival rates at one and five years were 95% and 74%, respectively. The most common causes of mortality for such patients are still chronic, terminal renal failure (40%), stroke (24%), myocardial infarction (11%) and heart failure (10%).¹⁸

Conclusion

Hypertensive crisis is a rare sign of a common disease. Distinguishing between a hypertensive emergency and urgency is essential to appropriate case management. A number of effective drugs now exist, helping to greatly reduce the morbidity associated with arterial hypertension. The practitioner must know the specific indications and precautions for each agent. And while an acute hypertensive crisis must be treated effectively, appropriate follow-up is as important in order to ensure the patient's BP is brought under control as effectively as possible. [CME](#)

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