

## Hypertensive Crisis Therapy

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**Annotation:** Prevention of hypertensive crisis in patients with hypertension may be associated with improving hypertension management. When patients come to the emergency room or doctor's office with a sharp increase in blood pressure, proper management is necessary to avoid fatal damage to the central nervous system, heart and kidneys as a result of the delay in starting effective therapy, or too heavy therapy-a rapid drop in blood pressure can cause hypotonic crisis.

**Keywords:** hypertensive crisis, sodium nitroprusside, B-adrenoblocators, Alpha adrenoblocators.

Hypertensive crisis is a serious clinical condition characterized by a sharp rise in blood pressure. Hypertensive crisis risk factor target limbs are at high risk of damage to the heart, eyes, kidneys and brain dysfunction. The crisis situation is not a separate disease. However, in most cases it is the result of poorly controlled arterial hypertension. Hypertensive crisis is a syndrome accompanied by an increase in blood pressure to 160/100 mm HG. etc. However, this indicator can vary significantly in each person. For example, an "experienced" hypertensive patient who does not take medications to correct blood pressure may not notice that it rises to 200-220 mm HG. but a person who first faced a sharp increase in pressure complains of obvious discomfort, even if it is 140/90-150/100 mmHg. it all depends on the individual characteristics of the patient's cardiovascular system and its compensatory capabilities. It should be understood that the state of "crisis" is called not because of the high values of blood pressure, but because of the high risk of damage to the target organs. Against the background of a sharp increase in pressure, the heart (acute coronary syndrome with the transition to myocardial infarction), brain (hemorrhagic stroke), eyeball (detachment of the retina) or kidney (acute kidney failure) can be affected. The possibility of developing such complications leads to the need to quickly correct blood pressure. An interesting fact: during intensive exercise in athletes, blood pressure can rise to 300/160 mm HG, but not cause any discomfort. Despite the high pressure, this condition is not a hypertensive crisis. This is due to the readiness of the trained cardiovascular system for such working conditions. Damage to the target organs is the main criterion for the classification of hypertensive crises. Depending on this, these conditions are divided into 2 categories: hypertensive crises are classified as hypertensive emergencies in the presence of acute or ongoing organ damage, or emergency hypertensive crises in the absence of damage to the last organs. Causes of a hypertensive crisis motivation for a sharp jump in pressure can be the following conditions: excessive physical activity; stress factor; the use of drugs that increase blood pressure; infectious diseases (sepsis) that are accompanied by the penetration of pathogens into the bloodstream; injuries; poisoning.

The clinical picture of a hypertensive crisis is always characterized by pronounced discomfort that appears suddenly, which becomes the main reason for seeking specialized help. Common signs characteristic of all forms of this condition are: headache; redness of the face; "spots" in front of the eyes; general weakness. In addition to the general symptoms indicated, patients often complain about other symptoms, on the basis of which the doctor prescribes a specific form of hypertensive crisis. Depending on this, the treatment may be different. The clinical picture of a hypertensive crisis is always characterized by pronounced discomfort that appears suddenly, which becomes the main reason for seeking specialized help. Common signs characteristic of all forms of this condition are: headache; redness of the face; "spots" in front of the eyes; general weakness. In addition to the general symptoms indicated, patients often complain about other symptoms, on the basis of which the doctor prescribes a specific form of hypertensive crisis. Depending on this, the treatment may be different. The neurovegetative form of an uncomplicated c The neurovegetative form of an uncomplicated crisis is

characterized by the following symptoms: cold; emotional excitement accompanied by a feeling of fear, irritability; thirst; nausea; vtez heart rate; at the end of the crisis, there may be a large amount of urination. The water-salt form of the crisis is characterized by apathy and lethargy. A pronounced weakness develops in the muscles. The lower limbs and face can swell. According to the type of parasthesia, sensitive sensitivity is impaired. There is a pronounced nausea with vomiting many times. The patient may faint, convulsions develop. In acute coronary syndrome, the patient complains of severe burning pain behind the sternum. With impaired cerebral circulation, there may be loss of consciousness, paralysis, paresis (the subject is determined by the localization of the damaged vein). With aortic dissection against the background of high blood pressure, the patient complains of severe pain in the chest, which moves away and does not respond well to medication. While there is evidence of a rapid or progressive central nervous system, myocardial, hematological or kidney deterioration, a sharp rise in blood pressure is considered an emergency. Hypertensive emergency patients require immediate lowering of blood pressure, usually done by intravenous injection under intensive care conditions. Patients with chronic hypertension tolerate a much higher rise in blood pressure than previously normotensive people. For example, encephalopathy rarely develops in patients with long-term hypertension until diastolic blood pressure exceeds 150 mm Hg. Conversely, a child with acute glomerulonephritis or a young woman with eclampsia may have encephalopathy when diastolic blood pressure is 100 mm Hg or less. Hypertensive crisis often occurs in patients with a history of hypertension. Most often, the patient did not take constant medication for the treatment as prescribed or received adequate treatment. Under such conditions, a hypertensive crisis occurs as a result of a sharp increase in systemic vascular resistance due to an increase in the circulating level of vasoconstrictor substances such as norepinephrine, angiotensin II or antinatriuretic hormone. Arteriolar fibrinoid necrosis is caused by a sharp rise in blood pressure, endothelial damage, deposition of platelets and fibrins, and loss of autoregulatory function, resulting in end organ ischemia. Ischemia, in turn, triggers the subsequent release of vasoactive substances, thereby initiating a vicious circle of subsequent vasoconstriction and myointimal proliferation. The purpose of therapy is to stop this cycle by reducing systemic vascular resistance. In addition to cases of aortic aneurysm or separation of myocardial ischemia, a significant decrease in cardiac output should be avoided. Preliminary history and physical examination should quickly distinguish hypertensive emergencies from urgent hypertensive crises.

A conversation with the patient and a physical examination should determine the provoking causes of an increase in blood pressure. Damage to the acute last organs or both due to a serious increase in blood pressure. The doctor should receive information about the history of any hypertension (duration, severity and degree of control), the degree of damage to pre-existing organs, the use of medications (prescription, over-the-counter and illegal drugs), concomitant acute or chronic diseases and any symptoms associated with a serious illness. increased blood pressure. In addition to determining blood pressure in the supine and upright position, the patient must undergo a guided physical examination, focusing on evidence of Retinopathy, heart failure, aortic dysfunction, or neurological dysfunction. Electrocardiogram, chest X-ray film, urine analysis, blood count and blood urea nitrogen and creatinine levels and serum electrolytes should be measured. Any evidence of acute or rapid progressive end-organ deterioration in Anamnesis (chest pain, neurological changes or shortness of breath), physical examination (new retinopathy, including bleeding, exudates or papilledema, neurological deficits, rales, S3 or pulse deficiency) or laboratory evaluation (ischemic changes). electrocardiogram, pulmonary edema in the chest X-ray film, increased levels of urea nitrogen or creatinine in the blood, hematuria or hemolysis in the blood smear) distinguish a hypertensive emergency from an urgent hypertensive crisis. Causes of a hypertensive crisis. Patients without evidence of progressive endorgan injury should be placed in a dark, quiet place if present, and blood pressure should be measured again in 30 minutes if blood pressure rises sharply, oral therapy should be started. If blood pressure no longer rises sharply, the patient should be sent for rapid monitoring. In a hypertensive emergency, the purpose of therapy is to carry out a rapid but gradual decrease in blood pressure. A reasonable goal for most hypertensive emergencies is to reduce the average arterial pressure by about 25 percent (or reduce diastolic blood pressure from 100 to 110 mm Hg) from a few

minutes to several hours, depending on the clinical case. a sharp drop in blood pressure and a decrease in normotensive or hypotensive levels should be avoided, as they can trigger end organ ischemia or infarction, blood pressure should be maintained at this initial target level for several days and reduced to normotensive levels in the following weeks.

Sodium nitroprusside is the drug of choice for the treatment of hypertensive emergencies, as it allows you to reduce blood pressure under control. Since it is administered with constant infusion, the use of sodium nitroprusside requires constant monitoring, preferably with intraarterial monitoring. Such intensive surveillance can prevent the use of this agent in many emergency departments. Labetalol to effective alternatives. or intravenous administration of diazoxide, a new agent that promises in small clinical trials is nifedipine, a calcium channel antagonist. Fentolamine, nitroglycerin, hydralazine and trimethaphane are indicated in specific situations, as discussed below. Sublingual nifedipine can cause an immediate drop in blood pressure when intravenous access is delayed or inaccessible. Angiotensin-enzyme inhibitors such as captopril administered orally or under the tongue and enalaprilate administered intravenously can quickly lower blood pressure in patients with renin-dependent hypertension. These remedies are often ineffective in patients with non-renin-dependent hypertension, so their use is not recommended for patients with hypertensive crises. Most patients with hypertensive crises probably have a decrease in volume as a result of pressure-related diuresis. In these cases, a decrease in volume can worsen hypertension. Accordingly, the use of diuretics and fluid restriction should be maintained clinically for patients who are overloaded with fluid and should not be prescribed regularly. A marked orthostasis (a decrease in blood pressure while standing), a decrease in central venous pressure or pulmonary capillary wedge pressure or prerenal azotemia, replacement of sodium and fluid with isotonic saline may be necessary to control blood pressure and improve kidney function. Sodium nitroprusside is the drug of choice for the treatment of hypertensive encephalopathy. Alternatives include labetalol, diazoxide, and nifedipine. A remedy such as clonidine and methyldopa weakens the central nervous system, which can be mixed with further deterioration of the central nervous system; therefore, they should be avoided. A sharp rise in blood pressure is a common cause and general consequence of a brain infarction. In both cases, the regulation of blood pressure after a stroke becomes dangerous, and even minimal intervention can lead to a decrease in blood pressure this risk is exacerbated by the loss of cerebral blood flow regulation in the area of a heart attack. Calcium-channel blockers are also effective and well absorbed benefits. B—adrenergic-receptor antagonists reduce renal plasma flow and glomerular filtration rate and should therefore be avoided in patients with kidney failure. Often the crisis is complicated by drug seizures, strokes, myocardial infarction or encephalopathy. The hypertonic crisis caused by the abuse of sympathomimetic agents usually responds to labetalol, while fentolamine and sodium nitroprusside can be used as alternatives. So, what to do for a hypertensive crisis: measure blood pressure. Take one of the drugs under the tongue: captopril 25 mg, corinfar 10 mg. Situation: sitting with your legs facing down (to facilitate heart function). If there is pain behind the Sternum, take nitroglycerin under the tongue. Measurement of blood pressure after 30-40 minutes. If it is elevated, then repeat taking the medication: Captopril 25 mg.or corinthar 10 mg. if it is not possible to give the patient the usual medication or if he has not taken antihypertensive drugs before, he may be given a 10 mg nifedipine tablet (but if he does not have clear tachycardia and angina pectoris). The drug effectively reduces pressure. If there is information about heart disease or the patient's nifedipine intolerance,the drug can be replaced with captopril12.5-25 mg, which normalizes blood pressure, protects the heart and prevents the development of nephropathy. In addition, unlike nifedipine, captopril does not cause drowsiness, dizziness and tachycardia. For the first type of hypertensive crisis, it is recommended to use 40 mg of anapriline orally or ground under the tongue. In the second type of hypertensive crisis, diuretic drugs are used, for example, furosemide 20-40 mg. It is recommended to give the patient 40 drops of Corvalol to relieve anxiety, panic , fear of death, which usually envelops a person in a state of hypertensive crisis. Instead of Corvalol, valocordin, valerian or motherwort tincture is suitable. For heart pain, give the patient validol or nitroglycerin, but the latter should be used very carefully: it dilates blood vessels and can lead to collapse.A 25% solution of magnesium sulfate can also be administered between the muscle or

intravenously for possible Parenteral administration: helps to overcome the hypertensive crisis with events of cerebral edema, including arterial hypertension.

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