THERAPEUTIC GUIDELINES

Treatment of Hypertensive Crisis*

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In a previous issue of this journal,1 the treatment of hypertension was described. Attention was paid only to the chronic phase, for this is the aspect most commonly encountered in practice. This paper is concerned with the acute phase, the management of which is entirely different from that of the prolonged elevation of blood pressure.

In recent years there has been a decrease in the incidence of hypertensive crises. The reason for this has not been established beyond doubt, but it is quite logical to predicate that the general availability and use of various antihypertensive agents in the treatment of hypertension during the past 15 years is very likely the responsible factor.

The hypertensive crisis may occur in hypertensive encephalopathy and acute cardiac insufficiency. Convulsions, headache, stupor, transitory hemiparesis, dysarthria, aphasia and impaired vision are common symptoms. The blood pressure is greatly elevated, and when it exceeds 250/140 mm Hg, prompt antihypertensive therapy is mandatory. Hypertensive encephalopathy usually occurs with severe hypertension of recent onset as in acute nephritis, toxemia of pregnancy and the malignant phase of essential hypertension.

Prolonged severe elevation of blood pressure may produce irreversible brain damage. Subarachnoid hemorrhage and dissecting aneurysm of the aorta are aggravated by an elevated blood pressure.

Acute pulmonary edema occurs with cardiomegaly and high blood pressure. This is characterized by weight gain, nocturnal dyspnea and is precipitated by an inadequate cardiac output causing an accumulation of fluid in dependent parts of the body. This is noted during the day followed by a transfer of fluid to the pulmonary vascular compartment a few hours after the patient lies down.

Determination of the blood urea nitrogen and creatinine should be made as soon as possible and followed by prompt therapy. If the blood urea nitrogen is greater than 50 mg per 100 ml, determinations should be repeated daily and antihypertensive therapy administered carefully to avoid total renal failure. Other studies routinely done in all hypertensive patients must be performed as soon as practical without causing a delay in treatment.

A basic requirement of adequate therapy is constant monitoring of the blood pressure by an automatic device or a sphygmomanometer at minute intervals until the blood pressure is stabilized. Subsequently the blood pressure may be determined every 15 minutes.

Another important precaution is to prepare a liter of 5 percent dextrose in distilled water containing one ampule of levarterenol (Levophed). This must be available in the patient’s room and not located in a remote treatment room or central supply. Therapy consists of the use of potent antihypertensive agents and a pressor agent may be needed, unpredictably, without advance warning, to correct too great a decrease in blood pressure.

A very effective and safe agent is diazoxide (Hyperstat). Its action is that of direct vasodilation without depressing the cardiac output or renal blood flow. One vial containing 300 mg should be given intravenously, quickly, in one dose. The hypotensive effect occurs within five minutes and lasts for two hours. The side effects of this agent are sodium retention, oliguria, hyperglycemia and glycosuria; these are transitory and pose no problem in management. The absence of drowsiness or clouding of the mentality enables the physician to be apprised of the patient’s degree of awareness. Diazoxide, currently available only for investigative use, hopefully will be approved for general medical use in the near future.

The next agent of choice is reserpine (Serpasil)
given as 2.5 mg intramuscularly every two hours, not to exceed 25 mg per 24 hours. Its onset occurs within two hours and its maximum effect in three to four hours with a duration of action of eight hours. It acts by depleting the catecholamines and may have a central effect. The resulting drowsiness may prove to be a handicap in evaluating the patient's mental state.

The third agent is methyldopa (Aldomet) whose dose is 500 mg intravenously every two hours for four doses. The onset of action occurs in two hours with the duration of effect ranging from four to eight hours. This drug probably decreases the sympathetic activity by undergoing conversion to alphanmethyl forms of dopamine and norepinephrine which are then taken into the storage granules in the same manner as norepinephrine. Drowsiness, but less than with reserpine, accompanies the use of this drug.

If an adequate blood pressure decrease is not obtained with the preceding agents, the intravenous infusion of trimethaphan camsylate (Arfonad), a ganglion blocking agent, is indicated. It is effective within three minutes, and its action continues only during the administration of the drug. Two ampules (1,000 mg) are mixed with 500 ml 5 percent dextrose in distilled water and given at a rate of .25 to .5 ml (.5 to 1 mg) per minute. With constant monitoring of the blood pressure, the rate of flow of the infusion is increased or decreased to maintain the desired blood pressure. Because of the potency and transitory effect of this drug, it should be discontinued and other agents substituted after a few hours.

The decrease in blood pressure with the use of a ganglion blocking agent is largely dependent on posture. This is caused by a failure of reflex venous constriction in the legs and splanchnic area and a subsequent pooling of blood. The hypotensive effect of the drug may be increased by elevating the head of the bed on six-inch blocks. Conversely, an excessive hypotensive effect can be modified by placing the blocks under the foot of the bed.

Sodium nitroprusside, a vasodilator, is a potent hypotensive agent. It has the disadvantage of not being commercially available and is chemically stable for only one month. It is made by dissolving 60 mg of sodium nitroprusside in 25 ml of 0.9 percent sterile sodium chloride solution. This solution is stored in a refrigerator in sealed, dated, brown bottles. Two bottles are added to 500 ml of 5 percent dextrose in distilled water immediately prior to its use. Continuous intravenous infusion of the drug is required to maintain its hypotensive effect which is achieved within two minutes after administration has begun. Side effects are rare with this very potent agent.

The drugs described have been named in order of their potency, except trimethaphan camsylate and sodium nitroprusside which are comparable in effectiveness. The next two agents are less potent than some of the previously named drugs. However, due to special characteristics, they are useful in specific conditions present in a hypertensive crisis.

Hydralazine (Apresoline) acts by direct vasodilation and causes reflex cardiac stimulation. Therefore, it may cause angina or myocardial infarction in the patient with impaired coronary circulation. It causes an increase in cardiac output, heart rate and blood flow to the kidneys, brain and coronary circulation. Because of these effects, this drug must be used with caution in patients with cardiac failure and coronary artery disease. It is quite effective and without contraindication during pregnancy and of value in the patient with impaired renal function. It may be given intravenously or intramuscularly in a dose of 10 mg. The onset of its action is 15 minutes, with a duration of several hours.

An additional agent is available for intravenous use in the hypertensive crisis. Ethacrynic acid (Edecrin), a diuretic, causes direct vasodilatation of arterioles and blocks renal tubular reabsorption of sodium. Pulmonary edema and cardiac failure are indications for its use. Its pharmacologic effect begins 30 minutes after administration and is given in a dose of 50 mg. Renal failure is not a contraindication to its use.

More than one of these agents may be given if an adequate decrease in blood pressure is not obtained promptly. Other supportive measures, such as digitalization, must be instituted.

At the earliest possible moment, parenteral therapy is discontinued and oral therapy, as outlined in previous publications, is begun. All the basic canons of treatment must be continuously and carefully followed.

As with the management of the chronic phase of hypertension, a knowledge of the clinical pharmacology of drugs as to time of onset, duration of action, mechanism of action, and side effects is mandatory. This complex problem demands "a combination product" of the careful use of one or more of these agents and sound clinical judgement.

Reference


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