Hypertension and antihypertensive therapy

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Induction of anesthesia constitutes one of the more serious threats to circulatory stability. In addition to possible direct effects of anesthetic agents on the cardiovascular system, the mechanisms of cardiovascular control are also distributed during that time. Baroceptor sensitivity is altered, sympathetic activity is either depressed or reflexly increased, and autonomic input from various sources can be activated by different surgical and anesthetic manipulation. The results of this complex interplay of factors can be either a severe hypotension or a marked hypertension; the former was often stressed in the past. More recently, the frequency, complexity, and dangers of the latter are being more widely recognized.

Of particular importance in this setting is the question of antihypertensive therapy and the advisability of its discontinuance before surgery. Antihypertensive drugs are of different types; hence, one cannot indulge in generalizations regarding their use. A few years ago sympatholytics were mostly used. The spectrum of antihypertensives is much wider now. Conventional discussions regarding antihypertensive therapy during anesthesia must therefore be viewed in that context. Classic viewpoints were greatly influenced by the sympatholytics in use in the past. The contractility of the

isolated heart is severely depressed by most anesthetic agents, but this is counterbalanced in intact organisms by reflex sympathetic activity. Undue depression of adrenergic activity was therefore judged undesirable in diseased hearts, which are more dependent than normal on this drive for adequate performance. Under these conditions, a case could be made for the preoperative discontinuance of drugs that could interfere with autonomic reflexes.

However, many of the drugs now used are vasodilators and beta-adrenergic blockers, which do not interfere with alpha-mediated vasoconstrictive reflexes. Moreover, hypertension during surgery could be dangerous in cardiac patients, particularly those with coronary artery disease. Because newer antihypertensive agents have a much shorter duration of action than that of older drugs such as reserpine and guanethidine, the return of arterial pressure to hypertensive levels could occur rapidly. Moreover, "rebound" adrenergic hyperactivity has been described after abrupt cessation of clonidine and propranolol; the clinical findings are not restricted to an increase in pressure but include a recognized autonomic crisis. In this situation the possible dangers to a patient with cardiac or coronary disease are obvious. Hence, we must reconsider the advisability of discontinuing antihypertensive therapy routinely before surgery. The tendency today would be to avoid cessation of antihypertensive therapy to prevent the burden of a rising pressure on the heart and atherosclerotic vessels.

Obviously, each case should be handled on its own merits. It has been our impression that maintained blood pressure control was preferable to discontinuing treatment. Cessation of diuretics for a day or two, however, may help avoid the risk of dehydration in patients who may have marginal renal function.

For the treatment of hypertension during and after cardiac surgery, short-acting peripheral vasodilators that do not overstimulate the heart are the drugs of choice. These vasodilators have a rapid onset of action and the dosage can usually be titrated to achieve a desirable level of blood pressure. However, all vasodilators are not equivalent in their spectrum of action; those such as diazoxide or hydralazine, which have little effect on veins, may produce undue stimulation of the heart. More appropriate to the conditions of cardiac surgery are vasodilators such as sodium nitroprusside, which dilate veins as well as resistance vessels.