# Anesthesia for the hypertensive patient

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Depending on the criteria used for its definition, the incidence of arterial hypertension appears to vary between 13% and 41% of the adult population. Thus, a considerable proportion of patients who are to undergo surgery have arterial hypertension and may be receiving antihypertensive therapy. Severe arterial hypertension (> 180/110 mm Hg) has been reported to be present in as many as 11% of surgical patients. The increased operative risk observed in hypertensive patients has been attributed to the cardiovascular complications of arterial hypertension and also to the possibility of adverse interactions between antihypertensive drugs and anesthetic agents. Despite the fact that the diagnosis of arterial hypertension is easy, the diagnosis has been made in only half the hypertensive patients, half of these patients are treated, and only half of the treated patients have adequate blood pressure control. A sustained elevation of arterial pressure aggravates and accelerates the development of the atherosclerotic process. Substantial coronary artery lesions are present in more than 60% of hypertensive patients. Indeed, coronary artery disease is the major cause of death in hypertensive patients, followed by cerebrovascular disease, cardiac failure, and renal failure. The cumulative 5-year morbidity is high (55%) for cardiovascular complications in hy-

pertensive patients. The death rate is eight to ten times higher than that for normotensive subjects. The risk of stroke, heart disease, and heart failure appears to be the same in the case of sustained hypertension and in the case of labile hypertension. Thus, even patients with labile hypertension should be considered at risk from the cardiovascular viewpoint.

The cardiovascular system of hypertensive patients is known to be particularly labile during anesthesia and surgery. Stimulation of the sympathetic nervous system caused by laryngoscopy, endotracheal intubation, bronchoscopy, surgical incision, mesenteric traction, and recovery from anesthesia is responsible for episodes of tachycardia, arterial hypertension, and in some instances elevation of the pulmonary wedge pressure indicating left ventricular failure.[1] Such hypertensive episodes are characterized by substantial increases of all major determinants of myocardial oxygen demand. Impaired oxygen supply is attested by the occurrence of dysrhythmias and of electrocardiographic signs of myocardial ischemia that usually follow hypertensive crises. However, myocardial depression caused by volatile and intravenous anesthetic agents may be enhanced. Sudden and large reductions of arterial pressure may occur and may compromise coronary perfusion. Because of the associated coronary artery disease, autoregulation of coronary blood flow is impaired and reductions of the coronary perfusion pressure will cause large reductions of coronary blood flow. The end result of prolonged imbalance between oxygen demand and oxygen supply is myocardial infarction, a well-documented complication of anesthesia in the hypertensive patient.

Widespread concern has been expressed that antihypertensive drugs may disturb the maintenance of circulatory homeostasis and thus may make the cardiovascular system more labile. Detailed hemodynamic studies, however, have shown that continuation of the hypertensive medication until the day of surgery contributes to improved cardiovascular stability.[2] Attitudes toward the preoperative management of hypertensive patients have gradually changed. Instead of withdrawing antihypertensive therapy, most authors now agree that the treatment of arterial hypertension should be maintained throughout the operative period. However, even recently, the opinion has been expressed that antihypertensive drugs should be stopped unless diastolic arterial pressure was greater than 120 mm Hg.[3,4] Such views are surprising and do not take into consideration the greater stability conferred by the antihypertensive medication and the risks attending its sudden discontinuance. Withdrawal of antihypertensive drugs may cause rebound hypertension sometimes accompanied by symptoms analogous to those of pheochromocytoma.[5] Discontinuing antihypertensive therapy has been compared to stopping insulin therapy in a diabetic patient.[6]

Treatment of arterial hypertension with beta-adrenergic receptor antagonists has caused even more controversy. Beta-adrenergic blockers have been shown to minimize the hypertensive responses to sympathetic stimulation and to reduce the incidence of dysrhythmias and of electrocardiographic signs of myocardial ischemia.[7] The degree of cardiovascular protection conferred by beta-blockers during anesthesia and surgery is strikingly greater than that conferred by other antihypertensive agents. Still some authors recommend withdrawal of beta-blockers before elective surgery under anesthesia.[3,4] They seem

to ignore deliberately both the advantages of beta-adrenergic blockade and the risk of life-threatening complications in the case of their untimely withdrawal. In hypertensive patients, symptoms suggestive of sympathetic overactivity and of hyperthyroidism have been reported after discontinuance of beta-blockers. However, the most commonly reported serious complications are unstable angina, myocardial infarction, and sudden death. Because of the associated coronary artery disease, such complications may occur when hypertensive patients are deprived of the myocardial protection due to beta-adrenergic blockade. Maintaining the antihypertensive medication including beta-adrenergic blockade is the logical approach to preoperative management of the treated hypertensive patient. The responsibility of the anesthetist is to be fully aware of the pharmacology of the antihypertensive drugs received by the patient and then to choose anesthetic agents and techniques that will cause the least drug interactions.

The preoperative management of the *untreated* hypertensive patient is open to discussion. In the case of mild hypertension it does not appear that the incidence of postoperative cardiac complications is higher in untreated hypertensive patients than in adequately controlled hypertensive subjects.[8] The untreated patient may not be subjected to an added clinical risk provided that (1) diastolic pressure is stable and not higher than 110 mm Hg, and (2) intraoperative and postoperative blood pressures are closely monitored, and episodes of hypertension or hypotension are decreased by immediate treatment. There is no doubt that more severe hypertension is an indication for postponement of elective surgery until blood pressure is adequately controlled. It is also obvious that even in the case of mild hypertension careful monitoring is essential to satisfy the requirement of active prevention and treatment of hypertensive and hypotensive episodes. Such problems are minimized by antihypertensive medication and may be suppressed by the perioperative use of adrenergic beta-receptor blockers.

Provided that sufficient time is spent during the preoperative visit to reassure the patient, premedication with sedatives is largely unnecessary. However, until the patient sees the anesthetist in the operating suite he or she may experience anxiety while being transported from the more familiar surroundings of the ward to the operating room. Thus, a modest sedative appears justified.

The electrocardiogram should be continuously monitored before, during, and after anesthesia. The bipolar CM5 configuration gives an excellent single-lead display of the ST segments and T waves. Direct arterial pressure monitoring provides beat-to-beat information and early warning signs of cardiovascular responses to drugs and to stimulation of the sympathetic nervous system. It should be regarded as routine in patients with cardiovascular diseases. Insertion of arterial cannulae under local anesthesia does not cause much discomfort and is particularly well accepted if carefully explained to the patient who then understands the value of monitoring. Since induction of anesthesia is one of the phases when cardiovascular lability is at its worst, it does not make sense to delay arterial pressure monitoring until the patient is asleep. Measurement of central venous pressure is useful when major surgery is envisaged and adequacy of filling of the vascular compartment may be difficult to assess. If central venous pressure is recorded, this should be continued during the early postop-

erative phase, thus facilitating volume replacement. Monitoring of the pulmonary wedge pressure (Swan-Ganz balloon-tipped catheter) is of considerable value in the severely hypertensive patient, in case of recent left ventricular failure, and in all patients with a high cardiac risk.

All intravenous induction agents, if used in doses that could be considered appropriate for normal patients, cause marked reductions of arterial pressure in hypertensive patients. Intravenous diazepam and propanidid have been found to be particularly unsatisfactory.[9] The sequence of intravenous administration of fentanyl (3 to 4 μg/kg) in divided doses for 5 minutes followed by a small dose of thiopentone (0.5 to 1.5 mg/kg) causes only modest reductions of arterial pressure.

Protection against the hypertensive responses to laryngoscopy and intubation could be obtained by the timely administration of vasodilating drugs, but this would be unlikely to protect against surgically induced hypertension (incision, mesenteric traction, aortic cross-clamping) or to prevent hypertensive episodes during awakening from anesthesia. Beta-blockers offer this type of protection.[1, 7]

Maintenance of anesthesia with the association of fentanyl and nitrous oxide/oxygen has been found satisfactory in hypertensive patients undergoing major surgery. Minimal concentrations of halothane have also been used. If, despite treatment, hypertensive episodes develop, treatment with an alpha-adrenergic receptor blocker or with a pure vasodilator will reduce the afterload presented to the left ventricle and improve its performance. Though hypotension is one of the complications most feared, its incidence is low if the doses of anesthetic agent are suitably scaled down and if the circulating volume is adequately maintained.

In the case of patients treated with beta-adrenergic blockers several precautions are essential. Experimental data have shown that the cardiovascular depression due to enflurane or methoxyflurane is more pronounced if the beta-adrenergic receptors are blocked.[10, 11] These volatile agents should be used cautiously at the lowest possible concentration. In the case of treatment with large doses of propranolol (> 10 mg/kg/day) or equivalent doses of oxprenolol or labetalol, the heart rate response to atropine is inhibited; despite atropinization, neostigmine consistently causes severe bradycardia.[12] Thus, reversing the effects of nondepolarizing muscle relaxants should be avoided in patients taking large doses of beta-blockers. In the postoperative phase, two types of hazard must be borne in mind. First, the heart rate response to hypovolemia will be blunted and unless all members of the team looking after the patient are aware of this consequence of beta-adrenergic blockade, hypovolemia may be underestimated, too much reliance being put on tachycardia as an index of hypovolemia. Careful observation of other clinical signs of hypovolemia become much more important than the heart rate response. Second, postoperative hypercapnia is more likely to cause cardiovascular depression in the case of treatment with beta-blockers: the direct negative inotropic effect of $CO_2$ is no longer compensated for by beta-adrenergic activation.[11] Knowledge of these possible risks makes the management of patients taking beta-blockers safer. The price to pay for improved cardiovascular stability is small.

Most anesthesiologists have experienced the disillusion of observing delayed postoperative cardiac complica-

tions, whereas the anesthetic management has been apparently impeccable. Unfortunately, even careful monitoring may fail to reveal profound alterations of left ventricular wall dynamics. Experimentally, anesthesia may induce severe regional myocardial dysfunction, (paradoxical systolic expansion, postsystolic shortening) in areas where coronary blood flow is critically reduced while electrocardiogram, heart rate, arterial pressure or pulmonary wedge pressure remain normal.[12] Concealed regional dysfunction with the attending tissue strain could be responsible for delayed cardiac complications. Experimentally, beta-adrenergic receptor blockade has been shown to minimize myocardial dysfunction. It is likely that beta-adrenergic blockade not only improves cardiovascular stability in the anesthetized patient but also prevents the development of concealed but severe regional myocardial dysfunction.

## References


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