Volume 50, No. 4
April 1979

THE JOURNAL OF Anesthesiology
THE AMERICAN SOCIETY OF ANESTHESIOLOGISTS, INC.

Editorial Views

Anesthesiology
50:281-284, 1979

Hypertension and Anesthesia—Fifty Years On

> The situation, therefore, is that the internist believes he can diagnose heart disease in life but can state only in a general way the patient's chances under operation, while the surgeon may deny his ability to discover heart disease while the patient is alive, but often confidently makes such a diagnosis if the patient dies.—*H. B. Sprague* (1929)[2]

ATTITUDES toward the management of anesthesia and operation in the hypertensive patient have tended to swing from one extreme to another. It has been a widely held view among physicians, surgeons and anesthesiologists that patients with cardiac disease in general, and hypertension in particular, have proved to be poor operative risks. While for many this view was intuitively based, there was ample evidence in the earlier half of this century to support this contention. Fifty years ago it was proposed that general anesthesia constituted an unjustifiable risk in patients with hypertensive cardiac disease,[1] a view based on some discouraging statistics from the Massachusetts General Hospital[2] (and other New England centers). Sprague[2] found the highest operative mortality rate in patients with hypertensive cardiac disease. Of the 170 patients whose records he analyzed, 75 had hypertensive cardiac disease, of whom 24 (32 per cent) died during or shortly after operation during general anesthesia, half of them succumbing to cardiac failure. Three of six women who had uncomplicated hypertension died during or shortly after operation during anesthesia with diethyl ether.

With the advent of antihypertensive drug therapy in the early fifties, reports of adverse responses to anesthesia, operation and electroconvulsive therapy in hypertensive patients began to appear. Several investigators reported high incidences of bradycardia and hypotension when anesthesia was administered to patients receiving reserpine, although these features were later attributed to blood loss and surgical manipulation rather than to any interaction between reserpine and anesthetic agents.[3] With the introduction of antihypertensive agents (methyldopa, guanethidine) that were more potent than the rauwolfia alkaloids, fears were expressed that cardiovascular homeostasis would be adversely affected during anesthesia and operation. Thus it was commonly proposed that antihypertensive therapy should be withdrawn prior to elective anesthesia and operation. The reviews of Dingle[4] and Hickler and Vandam[5] drew attention to the empirical nature of such a decision due to the paucity of information about the cardiovascular and other responses to anesthesia and operation of patients with treated or untreated hypertension. During the sixties numerous papers documented preexisting hypertension as a factor in the etiology of myocardial infarction as a sequel to anesthesia and operation,[6-8] but the numbers of patients in these series were small and one could take these findings as an index of suspicion only.

Two general methods of assessing the effects of anesthesia presented themselves in the late sixties. The first was to conduct careful, sophisticated hemodynamic studies in small groups of hypertensive patients, exploring the effects of different anesthetics, techniques and physiologic responses, and comparing the results with those in normal healthy patients. The second was to conduct epidemiologic studies using large groups of patients, but making limited and less sophisticated measurements, thus assessing simple indices of outcome in a relatively short time.

In 1968, my colleagues and I chose the first option because for us access to large numbers of hypertensive patients was not easy, and with an annual availability

Key words: Anesthesia: risk. Blood pressure: hypertension. Complications: hypertension. Polypeptides: renin-angiotensin.

of 25 patients, we anticipated that we might achieve a large number of patients over a ten-year period. We initially made comparisons of treated and untreated hypertensive and normotensive patients, during anesthesia and spontaneous ventilation,[9] artificial ventilation,[10] and various methods of induction and endotracheal intubation.[11] Each study gave further insight into unresolved questions and led to further studies of baroreceptor function,[9,12] the effects of fluid volume replacement,[12] and the influence of preoperative therapy with adrenergic beta-receptor antagonists, practolol or propranolol.[13] Based on these studies, we came to the conclusion that patients with untreated or inadequately treated hypertension were at greater risk of arterial hypotension during anesthesia, and of associated myocardial ischemia. We recommended[14] that patients with untreated hypertension should ideally have their arterial pressures brought under control before being subjected to anesthesia and operation.

In this issue, Goldman and Caldera, who have chosen the second option, describe a prospective survey of the effects of anesthesia and operation in 196 hypertensive patients (79 adequately treated, 40 inadequately treated, and 77 untreated), and compare the results with those obtained in healthy patients. This survey is supplementary to a rather larger general survey of multifactorial risk factors in non-cardiac surgical procedures.[15] Their main conclusion in the present paper is that ". . . elective surgery in the absence of ideal antihypertensive therapy need not subject patients to an added clinical risk provided a) diastolic pressure is stable and not higher than 110 torr, and b) intraoperative and recovery room blood pressure values are closely monitored and treated to prevent hypertensive or hypotensive episodes. . . ."

These are valid and important messages which nevertheless require some comment. The first proviso in the report of Goldman and Caldera is of fundamental importance in any discussion of hypertension, since it emphasizes the arbitrary distinction we make between normal and high arterial pressures.[16] There are many internists who would set the arbitrary level for this distinction as high as a diastolic pressure of 110 torr, and many would doubt the benefits of energetic treatment of patients with diastolic pressure/values in the range 90 to 105 torr.[17] Thus by most criteria, the patients in Group V of Goldman's study are largely patients with mild or moderate hypertension. It is indeed gratifying to find that the patients in this group are at no greater risk than their "normotensive" counterparts. It is important to distinguish from this group those patients subjected to anesthesia and operation whose diastolic arterial pressure values are consistently in excess of 110 torr (there were five in Goldman's study group), and whose high arterial pressures have not been properly assessed or treated, especially when they have histories of ischemic heart disease or cerebral vascular disease and their sequelae. Coronary-artery disease is the cause of death or serious morbidity in more than 60 per cent of all patients with established hypertension, and its course is only partially arrested by treatment of the hypertension.[18] In our studies, the main intraoperative problem has been that of the detection and management of myocardial ischemia, which has been associated with a number of other factors. Tachycardia and hypertension in response to the noxious stimuli of endotracheal intubation, surgical incision and traction, and aortic cross-clamping may cause ischemia because of an increase in myocardial work. Arterial hypotension may occur as a result of drug-induced myocardial depression or systemic vasodilatation, or associated with hypovolemia, but such hypotension has to be severe to cause clear electrocardiographic evidence of myocardial ischemia. Persistent supraventricular dysrhythmias in the form of either coupled atrial extrasystoles or, more commonly, junctional bradycardia, are the commonest cause of ischemic changes in the electrocardiogram, particularly those that we interpret as subendocardial ischemia. These dysrhythmias are common during anesthesia in the elderly patient, and are poorly tolerated by those who have preexisting hypertension and ischemic heart disease.

Not all patients who receive antihypertensive therapy are as well controlled as those in Goldman's Group III, and many are scheduled for surgical procedures with persistent high arterial pressures complicated by combinations of drugs used for their treatment, and backgrounds of coronary, cerebral or renal complications of their high arterial pressures. These patients are in a very high-risk group and can be identified by the criteria in Goldman *et al.*'s previous report describing cardiac risk factors.[15] Identification and classification of risk provide a useful means of proper selection of patients suitable for anesthesia, but are of limited value in determining the proper intraoperative and postoperative management of those patients for whom the urgency of the need for surgical treatment overrides the contraindications that may be applied when surgical treatment is elective.

The second proviso of Goldman and Caldera is perhaps more important than the first. When the course of anesthesia is unpredictable, the indication for careful monitoring is greater than at any other time. This is particularly true for patients who have hypertension during the first 24 hours after operation. Recovery from anesthesia is characterized by sudden increases

in arterial pressure from the low levels characteristic of anesthesia to values that may grossly exceed the normal upper range for any individual patient. Our cumulative total of hypertensive patients studied in detail exceeds 200 over a period of nine years. Arterial pressure values in excess of 250 torr systolic, or 140 torr diastolic, during the recovery period after anesthesia have occurred in less than 10 per cent. Our data would confirm those of Goldman and Caldera, that such postoperative hypertension is more common in patients who have previous histories of severe hypertension, regardless of whether the patients' arterial pressures were under control prior to anesthesia. Severe hypertension in the postoperative period may have adverse consequences for the patient, the commonest being subendocardial ischemia related to increased myocardial work, pulmonary edema due to acute left ventricular failure in the face of systolic pressures exceeding 300 torr, and cerebrovascular accidents. When patients are receiving antihypertensive medication prior to anesthesia, it is advisable for that medication to be continued up to and *including* the day of operation, as this will minimize the likelihood of postoperative hypertension.

Several questions remain unanswered, either by the results reported by Goldman and Caldera, or by results of previous studies. Goldman and Caldera suggest that a randomized prospective study of preoperative blood pressure control in patients with mild or moderate hypertension may be useful. It is unlikely that the results of such a study, though tidier in the statistical sense, would differ from the conclusions of their present study. On the other hand, there is certainly a need to define more carefully the criteria for rejecting a patient who comes to an elective operation with severe untreated hypertension, or poorly controlled blood pressure. Antihypertensive drug therapy appears to hold no major terror for the anesthesiologist, although the tight control of blood pressure in the postoperative period in patients who are unable to take oral preparations remains an area of uncertainty for the anesthesiologist and surgeon. There is still considerable antipathy to the parenteral use of adrenergic beta-receptor antagonists in the postoperative period, yet apart from hydralazine, most of the other antihypertensive agents are unsuitable for parenteral use.

The role of the renin–angiotensin–aldosterone system in the development and sustained progress of hypertension is undisputed in renal parenchymal or renovascular disease, but is still controversial with regard to essential hypertension. One feature of the study of Goldman and Caldera that should give us reason for concern is the incidence of *new* postoperative renal failure; this occurred in only one patient in my personal series. Intraoperative urine production may be severely impaired in hypertensive patients, especially those with pre-existing evidence of impaired renal function, but urinary output recovers in the 24 hours after operation. Goldman and Caldera note that intraoperative systolic arterial pressure values tended to be higher in those patients who had histories of renal complications. My own observations would support this finding. Because of increased plasma renin activity, one would anticipate that angiotensin II would maintain some arteriolar constriction during anesthesia, but there are no published data to support such a speculation.

Finally, a further area for investigation and improved clinical monitoring concerns the electrocardiographic detection of myocardial ischemia. Although precordial ST-segment mapping may be useful as a research method,[19] it has limited application in clinical anesthesia. A single bipolar precordial lead is the best alternative, but the interpretation of ST-segment changes is fraught with uncertainty.[20] Most patients who show depressed ST segments during anesthesia recover with no postoperative sequelae, but a few suffer myocardial infarction. How can the anesthesiologist predict which patient will respond adversely?

Patients who have severe hypertension will continue to demand the highest standards of preoperative assessment, intraoperative and postoperative monitoring, and careful anesthesia if the outcome of surgery is to be as successful as that in their normotensive counterparts. It is in the clarification of the grey area between normal and very high arterial pressures that the study of Goldman and Caldera has made an important contribution.

CEDRIC PRYS-ROBERTS, M.A., D.M., PH.D., F.F.A.R.C.S.
Professor of Anaesthesia
University of Bristol
Bristol, United Kingdom

References

1. O'Hare JP, Hoyt L: Surgery in nephritic and hypertensive patients. N Engl J Med 200:1292–1295, 1929
2. Sprague HB: The heart in surgery. An analysis of the results of surgery on cardiac patients during the past ten years at the Massachusetts General Hosptial. Surg Gynecol Obstet 49: 54–58, 1929
3. Katz RL, Weintraub HD, Papper EM: Anesthesia, surgery and rauwolfia. ANESTHESIOLOGY 25:142–147, 1964
4. Dingle HR: Antihypertensive drugs and anaesthesia. Anaesthesia 21:151–172, 1966
5. Hickler RB, Vandam LD: Hypertension. ANESTHESIOLOGY 33:214–228, 1970
6. Chamberlain DA, Edmonds-Seal J: Effects of surgery under

general anaesthesia on the electrocardiogram in ischaemic heart disease and hypertension. Br Med J 2:784–787, 1964

7. Mauney FM, Ebert PA, Sabiston DC: Postoperative myocardial infarction. Ann Surg 172:497–503, 1970
8. Breslin DJ, Swinton NW: Elective surgery in hypertensive patients—preoperative considerations. Surg Clin North Am 50:585, 1970
9. Prys-Roberts C, Meloche R, Foëx P: Studies of anaesthesia in relation to hypertension. 1. Cardiovascular responses of treated and untreated patients. Br J Anaesth 43:122–137, 1971
10. Prys-Roberts C, Foëx P, Greene LT, et al: Studies of anaesthesia in relation to hypertension: IV The effect of artificial ventilation on the circulation and pulmonary gas exchanges. Br J Anaesth 44:335–349, 1972
11. Prys-Roberts C, Greene LT, Meloche R, et al: Studies of anaesthesia in relation to hypertension. II Haemodynamic consequences of induction and endotracheal intubation. Br J Anaesth 43:531–547, 1971
12. Prys-Roberts C: Studies of anaesthesia in relation to the hypertensive patient. D. M. Thesis, University of Oxford, 1975
13. Prys-Roberts C, Foëx P, Biro GP, et al: Studies of anaesthesia in relation to hypertension. V Adrenergic beta-receptor blockade. Br J Anaesth 45:671–681, 1973
14. Foëx P, Prys-Roberts C: Anaesthesia and the hypertensive patient. Br J Anaesth 46:575–588, 1974
15. Goldman L, Caldera DL, Nussbaum SR, et al: Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 297:845–850, 1977
16. Pickering GW: High Blood Pressure. London, Churchill, 1968, pp 229–235
17. Veterans Administration Cooperative Study Group on Antihypertensive Agents: Effects of treatment on morbidity in hypertension: Results in patients with diastolic blood pressures averaging 90 through 114 mm Hg. JAMA 213: 1143–1152, 1970
18. Berglund G, Wilhelmsen L, Sannerstedt R, et al: Coronary heart-disease after treatment of hypertension. Lancet 1:1–5, 1978
19. Maroko PR, Libby P, Covell JW, et al: Precordial ST segment mapping: An atraumatic method for assessing alterations in the extent of myocardial ischemic injury. The effects of pharmacologic and hemodynamic interventions. Am J Cardiol 29:223–230, 1972
20. Kupersmith J: Electrophysiologic and electrocardiographic aspects of myocardial ischemia, including stress testing, Current Cardiovascular Topics. II: Angina Pectoris. Edited by Donoso E, Gorlin R, Stuttgart G, Thieme, 1977, pp 28–44