Hypotensive Anesthesia for Total Hip Arthroplasty:

A Study of Blood Loss and Organ Function (Brain, Heart, Liver, and Kidney)

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The authors attempted to determine whether hypotensive anes-thesia or the method of inducing hypotension has any effect on postoperative brain, liver, or kidney function and myocardial sta-tus following total hip arthroplasty. Thirty patients were anesthe-tized with halothane–nitrous oxide for total hip arthroplasty and randomly assigned to one of three groups. In two groups mean arterial blood pressure was decreased to 50 torr by high inspired concentrations of halothane (n = 9) or sodium nitroprusside (n = 12). In the third group (n = 9) mean blood pressure was main-tained within 20 per cent of control. Intraoperative blood losses decreased from 1,183 ± 172 ml in the normotensive group to 406 ± 102 ml and 326 ± 41 ml in the halothane and nitroprusside hy-potensive groups, respectively. Neither method of inducing hypo-tension nor hypertensive technique affected the results of postopera-tive tests of cerebral, hepatic, or renal function and myocardial sta-tus. These tests were performed before anesthesia and operation and at intervals in the postoperative course. In this small group of patients, deliberate hypotension for total hip arthroplasty added no morbidity and significantly shortened operating time, decreased blood loss, and decreased the number of blood transfusions needed. (Key Words: Anesthetic techniques: hypotension, in-duced, nitroprusside; Anesthetics, volatile: halothane; Blood loss; Transfusion; Surgery, orthopedic.)

CONTROVERSY regarding the use of deliberate hypo-tension for surgical procedures has existed for several years.[1] Although blood loss can be decreased and sur-gical operating conditions possibly facilitated by its use, the possible increased risk of tissue damage from hypotension-induced hypoperfusion is not well quan-tified.[1,2] The lack of data concerning the existence and rate of complications is not surprising, since testing for tissue damage and minor organ dysfunction is dif-ficult. Davis et al.[3] used pentolinium to decrease systolic blood pressure to 60 to 75 torr in 253 patients under-going total hip arthroplasty. They found little clinical evidence of cerebral, myocardial, or renal injury. However, their indices of organ function were too gross to assess subtle damage because they examined for "all-or-none" responses, e.g., stroke, rather than graded responses.

Additional controversy exists concerning the method for producing hypotension. Most techniques attempt to avoid profound myocardial depression and pro-duce hypotension by increasing venous capacitance and arteriolar dilation by administration of ganglionic blockers or sodium nitroprusside.[4] However, recently Bland and Lowenstein[5,6] found that halothane, a po-tent myocardial depressant, decreases the severity of experimentally-induced myocardial ischemia in the non-failing canine heart. This suggests that produc-tion of hypotension by a myocardial depressant such as halothane may be a desirable approach.

The advantages of decreased blood loss, shorter operating time, and decreased need for blood trans-fusions appear to be sufficient to warrant further in-quiry into the potential complications of deliberate hypotension. Also, recent laboratory evidence sug-gests that methylmethacrylate attaches to bone better in a dry surgical field, which would be more likely dur-ing deliberate hypotension (unpublished data), than a bloody one. We, therefore, designed a study that uses sensitive, but practical, testing of cerebral, hepatic, and renal function and myocardial injury during deliber-ate hypotensive anesthesia. We conducted this study in elderly patients undergoing total hip arthroplasty. Tests were performed before anesthesia and opera-tion and at two intervals in the postoperative course.

Methods and Materials

Thirty patients undergoing total hip arthroplasty gave voluntary informed consent. The protocol and consent procedures were approved by the local Com-mittee on Human Research. None of the patients had previously had any surgical procedure on the involved hip. Patients were excluded from the study when pre-operative evaluation revealed the following condi-tions: stroke or cerebral ischemic episodes; myocardial infarction within the preceding three years; renal dis-ease as indicated by an increased serum creatinine level or previous renal transplantation; systolic blood pressure greater than 170 torr or diastolic pressure greater than 110 torr. The underlying hip diseases were osteoarthritis (19 patients), rheumatoid arthritis (5 patients), congenital (5 patients), and Paget's dis-ease (1 patient). Although more than 50 per cent of the patients in each group were taking aspirin or indo-methacin preoperatively, all had normal preoperative

activated coagulation times, prothrombin times, and partial thromboplastin times.

By random selection the patients were assigned to one of three groups: 1) a normotensive group (n = 9) in which mean arterial blood pressure was maintained within 20 per cent of control using nitrous oxide, 60 per cent, and halothane, 0.5 to 1.5 per cent inspired, for anesthesia; 2) a hypotensive halothane group (n = 9) in which mean arterial blood pressure was maintained at 50 torr using nitrous oxide, 60 per cent, and halothane, 2 to 4 per cent inspired, for anesthesia; 3) a hypotensive nitroprusside group (n = 12) in which mean arterial blood pressure was maintained at 50 torr using nitrous oxide, 60 per cent and halothane, 0.5 to 1.5 per cent inspired, plus sodium nitroprusside.

Mean ages and preoperative systolic blood pressure values were 61 ± 5, 57 ± 5, and 58 ± 4 years and 132 ± 7, 134 ± 5, and 135 ± 4 torr in the normotensive, hypotensive halothane, and hypotensive nitroprusside groups, respectively. In all groups, halothane concentration was measured every five to ten minutes with an infrared analyzer. All patients received pentobarbital, 100 mg, and atropine, 0.4 mg, intramuscularly one hour before operation. Anesthesia was induced with thiopental, 100–200 mg, intravenously, followed by inhalation of nitrous oxide and halothane. The trachea was intubated following intravenous administration of succinylcholine, 1 mg/kg. In addition to a peripheral intravenous line, we inserted an indwelling catheter into a radial artery in the hypotensive groups for continuous measurement of arterial blood pressure. Blood pressure was measured by oscillometry in the normotensive group. Hypotension was induced immediately before incision of the skin and maintained until closure of the skin was completed. Rectal or esophageal temperatures were between 34.5 and 36.5 C, with no significant difference among the groups.

The electrocardiogram was displayed continuously. Blood was given in the operating room when blood loss exceeded 5 per cent of the estimated blood volume. Intravenous fluids were given as 5 per cent dextrose in lactated Ringer's solution or as 0.45 physiologic saline solution, except for the solution of 5 per cent dextrose in water used to prepare the nitroprusside. The total volume of crystalloid solution given in the operating room was 1,000–1500 ml.

All operations were performed by one surgeon (WRM), with the patients in the lateral position. A Harris modified hip prosthesis was used in each patient. Use of this prosthesis did not necessitate osteotomy of the greater trochanter.

Data were collected one or two days preoperatively, one or two days postoperatively, and seven or eight days postoperatively. At each of these times the following tests of organ function were obtained. Cerebral function was measured by a clinical psychologist who used the Neurological Index of Mental Impairment (NIMI) and the Wechsler Memory Scale (WMS). A practice factor was included in the final scoring.[7] Neurologic examinations were performed in an attempt to detect sensory or motor deficits. Cardiac status was assessed by measurement of creatinine phosphokinase (CPK) with isoenzymes, lactate dehydrogenase (LDH), electrocardiogram, and vital signs. Serum creatinine, blood urea nitrogen (BUN), 12-hour creatinine clearance, serum electrolytes, urinary electrolytes, and urinalysis were used to assess renal function. Bromsulphalein retention (BSP), bilirubin, serum proteins, alkaline phosphatase, serum glutamic oxaloacetic transaminase (SGOT), and serum glutamic pyruvic transaminase (SGPT) were measured to estimate hepatic function. Operative blood loss was assessed by weighing sponges, measuring suction drainage, and estimating the amount surrounding each wound. Postoperative blood loss was determined by measuring suction drainage. Hematocrit and hemoglobin also were measured preoperatively, and one, and seven days postoperatively. Samples of arterial blood were obtained intermittently during the operation for analysis of blood gases and pH in all patients except four in the normotensive group and five in the hypotensive nitroprusside group.

Preanesthetic control values were compared with postoperative values within each group using an analysis of variance. Comparisons between groups were accomplished by using a two-tailed Student's t test. P values less than 0.05 are regarded as significant.

Results

There was no significant difference in ages, weights, or preoperative systolic blood pressures among the three groups. Sixteen patients were more than 60 years old, and 11 of these received hypotensive anesthesia. Four of the hypotensive patients were more than 70 years old.

Mean operating time was decreased significantly by hypotension (fig. 1). Mean operating times in the two hypotensive groups were the same. Intraoperative blood loss was less in those patients receiving hypotensive anesthesia than in those receiving normotensive anesthesia (fig. 1). There was no significant difference between operative blood losses of patients made hypotensive with halothane and nitroprusside. Intraoperative blood losses exceeded 500 ml in only two of the 21 hypotensive patients, and in 12 the blood losses were less than 300 ml. The mean total amounts

FIG. 1. Operating times, intraoperative blood losses, and postoperative blood losses in patients receiving normotensive and hypotensive anesthesia. Operating times and intraoperative blood losses were less in both hypotensive groups than in the normotensive group (P < 0.05). There was no difference in postoperative blood losses.
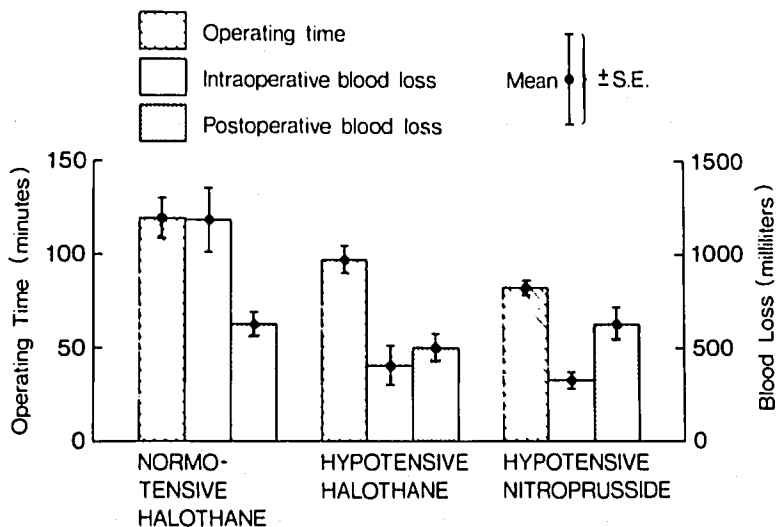
of blood transfused during hospitalization were: normotensive group, 1,330 ± 200 ml (SE); hypotensive halothane, 500 ± 120 ml; and hypotensive nitroprusside, 230 ± 80 ml. Ten of the 21 hypotensive patients (six nitroprusside, four halothane) did not need blood transfusion, and nine of the remaining 11 received only one unit (500 ml) each. Postoperative bleeding was no greater in patients receiving hypotensive anesthesia than in those whose blood pressures were maintained near normal levels (fig. 1). There was no significant difference in mean hematocrits among the three groups at any of the three measuring times.

The mean total dose of sodium nitroprusside was 34 ± 13 mg, or an average of 0.5 mg/kg body weight. Total doses ranged from 12 to 50 mg, given over 65 to 115 minutes. Mean inspired concentrations of halothane, measured every five to ten minutes, were 3.1 per cent in the deep halothane group and 1.0 per cent in the other two groups.

The insertion of methylmethacrylate into the femoral neck was associated with significant decreases in systolic arterial blood pressure in six patients from each group. These decreases amounted to a mean of 17 torr in the control group; 12 torr in the deep halothane group; 8 torr in the nitroprusside group. The pressures all returned to the pre-methylmethacrylate levels within 10 minutes.

CEREBRAL FUNCTION

There was no significant difference in the results of the two psychologic function tests among the three groups. The patients in each group scored higher a week postoperatively than they did preoperatively. Nine patients failed to complete these tests because of pain, immobility, or sedation. Four of these patients

were in the normotensive group; two were in the halothane hypotension group, and three were in the nitroprusside hypotension group. No patient had sensory or motor deficits on neurologic examination. One patient in the nitroprusside group became disoriented during the first 24 hours after operation and recovered subsequently.

CARDIOVASCULAR STATUS

Creatinine phosphokinase was increased in every patient following operation, but in no instance were myocardial isoenzymes (CPK-MB) present. Lactate dehydrogenase (LDH) levels were all within normal limits. No electrocardiographic evidence of myocardial infarction was present. Nonspecific nonprogressive changes in ST–T-wave segments were seen in one patient in the normotensive group and in two patients in each of the hypotensive groups. One patient receiving nitroprusside had multifocal premature contractions whenever mean arterial pressure decreased below 55 torr. She had a history of paroxysmal atrial tachycardia.

Intraoperative arterial oxygen partial pressures were significantly higher in those patients receiving deep halothane anesthesia than in those receiving nitroprusside or normotensive halothane anesthesia (table 1). The deep halothane group also had higher arterial carbon dioxide partial pressure values when allowed to ventilate spontaneously.

RENAL FUNCTION

None of the groups showed significant differences or significant changes in serum creatinine, blood urea nitrogen, serum electrolytes, or urinary electrolytes. Mean creatinine clearance values were more than 75

TABLE 1. Intraoperative Acid–Base and Blood–gas Value (Mean ± 1 SE)

Group	n	Pa_{O_2} (torr)	Pa_{CO_2} (torr)	pH	Base Excess (mEq/l)
Normotensive, spontaneous ventilation	5	89 ± 12	44 ± 3	7.32 ± 0.02	−3.7 ± 1
Deep halothane, controlled ventilation	4	147 ± 26*	41 ± 5	7.32 ± 0.03	−5.1 ± 2
Deep halothane, spontaneous ventilation	5	162 ± 19*	54 ± 1*	7.24 ± 0.04	−3.6 ± 1
Nitroprusside, spontaneous ventilation	7	81 ± 6	39 ± 1	7.31 ± 0.03	−5.8 ± 1

Base excess determined from Siggaard-Andersen nomogram.　　* Significantly higher ($P < 0.05$) than values for the other groups.

ml/min at each measuring period for each group (table 2). These patients did not have urinary catheters during operation, and none needed catheterization postoperatively.

HEPATIC FUNCTION

The changes in bromsulphalein retention indicated some alteration in hepatic function in all groups (fig. 2). However, none of the groups had significant differences postoperatively from each other or from preoperative values in mean values for serum bilirubin, alkaline phosphatase, SGOT, or SGPT. The mean values for total serum proteins were between 5.75 and 6.75 g/100 ml in all groups at all times.

Discussion

Our study demonstrates that deliberate hypotension to a mean arterial blood pressure of 50 torr during total hip arthroplasty decreases operating time, blood loss, and the number of transfusions needed without apparent injury to the heart, brain, liver, or kidney. The two methods by which hypotension was induced produced similar results despite the fact that different mechanisms are involved. Halothane produces hypotension primarily by myocardial depression[6,8] and nitroprusside by decreasing total peripheral resistance.[4,9] Many variables prevent exact comparisons with other studies, but it is apparent that both hypotensive general anesthesia and regional anesthetic techniques decrease intraoperative blood loss[10-12] (table 3). Our approach appears to have produced the lowest blood loss.

The absence of CPK-MB isoenzymes[13] suggests that

TABLE 2. Creatinine Clearance (ml/min ± SE)

	Control	Halothane Hypotension	Nitroprusside Hypotension
Preoperative	75.4 ± 11*	86.4 ± 10	76.4 ± 9
1 or 2 days postoperative	95.5 ± 15	83.4 ± 12	101.5 ± 19
7 or 8 days postoperative	101.0 ± 18	77.4 ± 9	73 ± 7

* Corrected to 1.73 m² BSA.

myocardial injury did not occur in any patient. No electrocardiographic evidence of infarction appeared, although a few patients had transient nonspecific ST- and T-wave changes that were associated with pain. The partial pressures of oxygen in arterial blood determined intraoperatively at an inspired concentration of 40 per cent (table 1) indicated that adequate arterial oxygenation was present, which is consistent with results of other studies in which oxygen pressures were measured during hypotension induced by halothane or nitroprusside.[14,15]

Blood and urinary chemistry values reflected no impairment of renal function after two hours of hypotension. Whether a longer hypotensive period would produce injury remains unanswered by this study. Hugosson and Hogstrom[16] also found no change in creatinine clearance when systolic blood pressure was maintained between 50 and 60 torr by deep halothane anesthesia and trimethaphan for 5 to 45 minutes in 96 patients.

Stevens *et al.*[17] reported altered hepatic function tests in normal volunteers following prolonged anesthesia with halothane. We observed BSP increases similar to those they found following halothane anesthesia without operation. Deep levels of halothane anesthesia as used in our study did not cause any greater impairment of function than lighter levels with or without hypotension (fig. 2).

The tests of memory, reasoning ability, and neurologic status used to assess cerebral function are more sensitive than those previously used to study complications of deliberate hypotensive anesthesia. The NIMI test[18] was originally designed to evaluate subtle cortical impairment in patients suffering strokes. The WMS[7] is an abbreviated form of the traditional memory test. Its nine subscores indicate the ability to recall and reason with information of both recent and distant input. Davison et al[19] used these tests three, six, and 30 days after anesthesia with halothane or isoflurane. They demonstrated slight impairment of intellectual function with both drugs three and six days after anesthesia. Our test results indicate no difference in cerebral function between normotensive and hypotensive groups. We believe, but cannot prove, that the

disorientation experienced by one patient during the first 24 hours after operation would not have been prevented by using normotensive rather than hypotensive anesthesia, since postoperative disorientation can occur following normotensive anesthesia.[20]

The finding of no brain damage following deliberate hypotension is consistent with results of previous studies using physiologic rather than psychologic tests. Griffiths et al.[21] induced hypotension (mean arterial blood pressure 67 torr) with sodium nitroprusside and found no decrease in jugular venous blood oxygen tension, a measure of total cerebral oxygenation.[22] Prys-Roberts et al.[14] found a mixed jugular venous blood oxygen tension of less than 20 torr in two of 15 patients in whom hypotension was induced with halothane. Mean arterial blood pressure was 34 torr in six of their patients. In spite of the low mixed venous oxygen tensions, all patients recovered consciousness and showed no sign of cerebral damage. Eckenhoff et al.[23] compared two groups of young patients, one of which was subjected to a mean systolic blood pressure of 63 torr for 90 minutes. Use of six psychometric tests chosen to elicit minimal brain damage showed no significant difference between the groups. Rollason et al.[24] also used six psychometric tests to compare two groups of 27 patients whose mean ages were 63 and 68 years during spinal anesthesia. One group had a mean arterial systolic blood pressure of 66 torr and the other by the use of vasopressor drugs had a mean pressure of 119 torr. These tests showed no difference between the groups. These studies and our results indicate that the brain can tolerate a mean arterial pressure of 50 torr quite well. Until there is evidence indicating factors that govern perfusion in the presence of these diseases or defining the limits of allowable hypotension, we believe hypotension is contraindicated in patients with cerebral, renal, hepatic, or cardiovascular
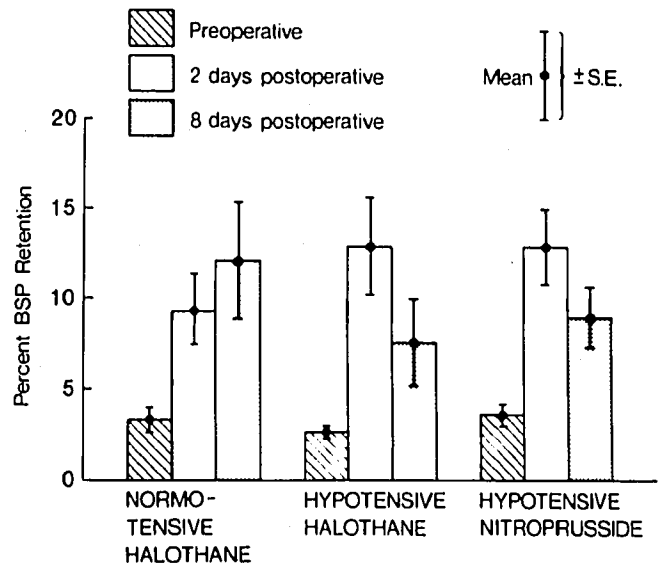

FIG. 2. Hepatic function pre- and postoperatively as determined by bromsulphalein (BSP) dye retention. Each retention was determined after 45 minutes. Although BSP values were significantly higher postoperatively ($P < 0.05$), there was no difference among the three groups.

disease. Evidence suggesting that our list of conditions contraindicating the use of hypotensive anesthesia may be too restrictive is beginning to appear. Torvik and Skullerud[25] found that the risk of precipitating cerebral infarcts by lower blood pressure in hypertensive patients is not greater in atherosclerotic than in non-atherosclerotic patients. Recent studies suggest that benefits to the heart may result from decreases in contractility, heart rate, and afterload produced by an anesthetic drug such as halothane.[5,6] These preliminary studies suggest that with more information the use of hypotensive anesthesia may be liberalized.

Since wasted ventilation (physiologic deadspace) is

TABLE 3. Published Reports of Average Blood Loss for Total Hip Arthroplasty

Series	Blood Pressure	Anesthetic Technique	Mean Intraoperative Blood Loss (ml)
Thompson et al. (present study)	Normotensive	Halothane–N_2O	1,183
Davis et al.[3]	Normotensive	Halothane	2,250
Amaranath et al.[10]	Normotensive	N_2O-morphine-d-tubocurarine	1,764 ± 261
Amaranath et al.[10]	Normotensive	Halothane–N_2O	1,514 ± 273
Sculco and Ranawat[11]	Normotensive	N_2O-fentanyl or halothane	1,088
Davis et al.[3]	Hypotensive	Halothane–N_2O–pentolinium	1,150
Amaranath et al.[10]	Hypotensive	N_2O-morphine–trimethaphan	884 ± 89
Amaranath et al.[10]	Hypotensive	N_2O-morphine–nitroprusside	820 ± 96
Thompson et al. (present study)	Hypotensive	Halothane–N_2O	407 ± 102
Thompson et al. (present study)	Hypotensive	Halothane–N_2O–nitroprusside	326 ± 42
Stanton-Hicks[12]	Normotensive	Epidural—bupivacaine	490
Sculco and Ranawat[11]	Normotensive	Spinal—tetracaine	594

increased during deliberate hypotension,[26] the use of spontaneous ventilation in all but five patients (table 1) may be questioned. Except for the increased Pa_{CO_2} in the hypotensive halothane group (table 1), arterial blood-gas levels were satisfactory. In fact, when ventilation is controlled, extreme hyperventilation (Pa_{CO_2} < 25 torr) should be avoided, since it may increase susceptibility to cerebral hypoxia when cerebral perfusion pressure is less than 50 torr.[27] We cannot explain the higher Pa_{O_2} levels in the hypotensive halothane group.

One might suggest that our study population was too small; that is, complications would have become evident if our sample size had been larger. We reasoned that a study dependent on a relatively infrequent response, such as presence of cerebral vascular accident,[3] requires a large number of patients to be studied. We elected to use sensitive tests with graded responses, which we hoped would detect subtle changes without frank organ damage. The results of our study would apply to the general population if any injury in response to hypotension were a graded phenomenon occurring to a greater or lesser extent in all patients. However, our study population was small and may have failed to include the unusual patient who, for various reasons, might respond to hypotension in a qualitatively different manner. For example, we deliberately excluded patients with cerebrovascular accidents or hypertension, and those who had had myocardial infarction within the previous three years. We cannot say that such patients would respond as well as did our study group.

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