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Title : PRIMARY ALDOSTERONISM: ANESTHETIC MANAGEMENT

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Introduction: Primary aldosteronism may result from an adrenal adenoma, adrenocortical hyperplasia or rarely adrenal carcinoma. Clinical presentation is characterized by the triad of hypertension, hypokalemia and metabolic alkalosis. The normal renin production is suppressed, the baroreceptor response is lost and a new steady-state is achieved with a 2-3 liter volume expansion due to increased total body sodium. The present study examines factors influencing the anesthetic stability of these patients.

Method: Thirty-eight patients with primary aldosteronism were studied. The mean diastolic pressure was 120 torr (range 100-150), mean serum K^+ was 2.8 mEq/L (range 1.7-3.3) and mean serum bicarbonate was 32 mEq/L. All patients received potassium supplementation preoperatively. Pharmacologic regulation of hypertension prior to surgery is summarized in Table 1. The induction of anesthesia with thiopental and succinylcholine followed a conventional premedication. Anesthesia was maintained with either a narcotic/relaxant regimen or a halogenated inhalation agent. Anesthetic instability was defined as a ±40 torr change in mean arterial pressure during induction or two such episodes during the maintenance of anesthesia.

Results: Preoperative hypertension control was best achieved with spironolactone. Preoperative supplementation raised the K^+ to 3.0 mEq or greater in all but one patient. In addition, patients placed on a salt restricted diet required less K^+ and attained a higher level of serum K. During induction, hypotension occurred in 26 patients (a reduction in mean arterial pressure of 40 torr) and hypertension in one patient (an increase in the mean arterial pressure of 40 torr). Twenty one of the 38 patients had a stable anesthetic course. Of the 7 patients receiving a narcotic/relaxant anesthetic, 6 exhibited pressure instability. A stable anesthetic course was most often seen in those patients prepared with spironolactone, who subsequently received a halogenated anesthetic (Table 2).

Additional factors influencing intraoperative stability were: 1. Patient positioning (hypotension in 14 patients) 2. Tumor manipulation (hypertension in 9 patients and 3. Tumor removal (hypotension in 4 patients). Normotension was observed in only 20% of the patients in the first 24 hours following surgery. Hypertension was observed in 58% with the remainder having hypotension. All normotensive patients had received spironolactone.

Discussion: The major determinants of intraoperative stability are the preoperative preparation with spironolactone and the use of a halogenated anesthetic. Spironolactone is a competitive antagonist of aldosterone. It has been shown that within 10 days of the administration of spironolactone to patients with primary aldosteronism, a significant naturesis occurs and a corresponding weight loss.[1] The hypervolemic state is reduced, thereby lowering the blood pressure and restoring the baroreceptor response. This will allow for postural volume responsiveness. Normal renin levels have been measured in patients medicated with spironolactone[2] and may result in normal aldosterone production in the immediate postoperative period. This is in contrast to the untreated patient with primary aldosteronism with depressed renin levels whose renin-angiotensin system does not function normally for several weeks postoperatively. The use of a halogenated anesthetic, either enflurance or halothane, offers the advantages of precise and immediate control of the depth of anesthesia and its attendant influence on cardiovascular stability.

Table 1 PREOPERATIVE PREPARATION

	Diastolic Pressure >120	120-100	<100
NONE	2	1	2
METHYL DOPA	1	1	1
GUANETHIDINE	3	-	-
SPIRONOLACTONE	1	4	8
9 Meq Na DIET	3	4	4
OTHER	-	2	2

Table 2 INTRAOPERATIVE COURSE

	STABLE	UNSTABLE
NONE	1	3
METHYL DOPA	-	3
GUANETHIDINE	2	1
SPIRONOLACTONE	11	2
9 Meq Na DIET	7	4
OTHER	2	2

References: 1. Horton R: Aldosterone: Review of its physiology and diagnostic aspects of primary aldosteronism. Metab 22:1525-1545, 1973
2. Ganguly AA, Melada GA, Leutscher JA et al: Control of plasma aldosterone in primary aldosteronism: Distinction between adenoma and hyperplasia. J Clin Endocrinol Metab 37:765-775, 1974