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Chronic Hypokalemia and Intraoperative Dysrhythmias

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To investigate whether chronic hypokalemia increases the occurrence of dysrhythmias during anesthesia, the authors recorded the intraoperative electrocardiograms of normokalemic (K+ = 5.0 – 3.5 mEq/l; N = 88) and chronically hypokalemic patients (K+ = 3.4 – 2.6 mEq/l; N = 62). In each patient, serum potassium was measured and a 12-lead ECG was analyzed prior to surgery. No patient received potassium perioperatively. Lead II was monitored continuously during anesthesia, either by a Holter monitor (N = 81) or by a trained observer (N = 69). A variety of general anesthetic techniques were utilized, without consideration for the potassium level. The hypokalemic population had a higher incidence of hypertensive and ASA Class III patients (P = 0.03). Analysis of variance revealed no significant difference in the incidence of other characteristics between the hypokalemic and normokalemic groups: age, hypoxemia, cardiac disease, preoperative dysrhythmias, digitalis therapy, surgical site, anesthetic agent, and intubation. The method of ECG monitoring did not affect the incidence of dysrhythmias recorded. Multivariate analysis revealed that the occurrence of intraoperative dysrhythmias correlated with the presence of preoperative dysrhythmias only. The authors conclude that chronic hypokalemia *per se* is not associated with a higher incidence of intraoperative dysrhythmias. (Key words: Complications: arrhythmia. Heart: arrhythmia. Ions: chronic hypokalemia; potassium.

HYPOKALEMIC PATIENTS are thought to have an increased potential for dysrhythmias during anesthesia. Hazardous levels of hypokalemia have been suggested but not documented. Having observed stable intraoperative cardiac rhythms in chronically hypokalemic patients, two of us (LES and TSV) postulated that the danger was largely theoretic. We performed a clinical study to test the hypothesis that there is a correlation between serum potassium and the incidence of intraoperative dysrhythmias.

Materials and Methods

This prospective study was approved by the Institutional Review Committees of Humana Hospital Sunrise, Valley Hospital, Southern Nevada Memorial Hospital, and Desert Springs Hospital, in Las Vegas, Nevada. Our sample population was drawn from the private practice of ten anesthesiologists. We studied people who had the following: 1) a serum potassium determination within 24 h before anesthesia; 2) an electrocardiogram (ECG) within 24 h before anesthesia; 3) no potassium administration before anesthesia; 4) no indication of acute potassium loss (*e.g.*, gastric suction, rapid change in serum potassium); 5) general anesthesia.

Whenever we discovered such a patient on preoperative rounds, we studied that person the next day. Anesthesiologist–patient pairing occurred by chance. Anesthetics were chosen without regard to the potassium level, and potassium was not administered intraoperatively. Throughout each anesthetic, lead II of the ECG was monitored continuously. An initial group of 69 patients was monitored by a trained observer (PS) who recorded any abnormal rhythm on a strip recorder. To eliminate observer bias, intraoperative ECGs of an additional 81 patients were recorded on a Holter monitor. The Holter recordings were processed by an Avionics® Model 662A Arrhythmia Computer, which automatically printed any abnormal rhythm.

Without knowledge of the potassium value, two of us (LES and TSV) reviewed the ECG tracings and anesthetic records. We noted the types of rhythm and necessity for treatment. Then we listed the characteristics of each patient: age, gender, serum potassium, preoperative ECG analysis, history of hypertension (systole > 160, or diastole > 95, or receiving antihypertensives), preoperative hypoxemia (Pa_{O_2} < 65 mmHg, FI_{O_2} = 0.21), history of heart disease, digitalis therapy (digitalis was continued until surgery), ASA physical status, anesthesiologist, location of surgery, primary anesthetic agent, FI_{O_2}, intubation, and duration of anesthetic. Finally, the patients were divided according to the potassium level and the type of monitoring: Group 1—normokalemia; monitored by observer (n = 34); Group 2—normokalemia; Holter monitor (n = 54); Group 3—hypokalemia; monitored by observer (n = 35); Group 4—hypokalemia; Holter monitor (n = 27). Normokalemia was defined by a serum potassium between 3.5–5.5 mEq/l. Potassium measurements were made by either flame photometry or ion-specific electrode. These techniques yield results that differ by less than 0.1 mEq/l. The constant of reproducibility is 2%. Any potassium value less than 3.0 mEq/l was verified automatically by a repeat determination.

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The relative incidences of preoperative characteristics, intraoperative characteristics, and intraoperative rhythms between groups were compared by analysis of variances. In order to identify any factor associated with intraoperative dysrhythmias, the entire population was arranged into subgroups whose potassiums values varied by 0.5 mEq/l. These subgroups were subjected to multivariate analysis.

Results

Repetitive analysis of variance revealed no significant differences in the incidence of preoperative or intraoperative characteristics between Groups 1 and 2 or between Groups 3 and 4. The incidence of intraoperative dysrhythmias was similar. Therefore, the data were pooled to form a normokalemic group and a hypokalemic group. The incidence of the preoperative and intraoperative characteristics of these groups was also compared by analysis of variance.

Table 1 summarizes the preoperative characteristics. Of the 150 patients, 62 were hypokalemic (serum K+ < 3.5 mEq/L). Hypokalemia was associated with the following clinical settings: diuretic therapy—43 patients; hypertension, no diuretics—12 patients; gastrointestinal disease—three patients. Four patients had no apparent etiology for their hypokalemia. Significant differences arose in the distribution of ASA physical status and in the incidence of hypertension. The hypokalemic group had more ASA physical status III patients, but fewer status II and IV patients than the normokalemic group ($P = 0.03$). A history of hypertension occurred more frequently in the hypokalemic patients ($P = 0.03$). Hypokalemia was not associated with a higher occurrence of dysrhythmias in the preoperative ECGs. Premature ventricular contractions (PVCs) predominated the preoperative rhythm disturbances. PVCs accounted for 53% of all dysrhythmias in the hypokalemic group and 67% in the normokalemic group. Premature atrial contractions (PACs) constituted 40% of dysrhythmias in the hypokalemic group and 25% in the normokalemic group. The groups were equivalent in their intraoperative characteristics (table 2).

The incidence of intraoperative dysrhythmias is detailed in table 3. The percentage of hypokalemic patients incurring an intraoperative dysrhythmia was not significantly different from the percentage of normokalemic patients. In the hypokalemic group, 35% of patients incurred an intraoperative dysrhythmia; in the normokalemic group, 48% of patients had intraoperative dysrhythmias ($P = 0.8$). Individual rhythms occurred at similar frequencies across the subgroups. No patient required antidysrhythmic therapy. Multivariate analysis revealed no association between potassium levels and the occurrence of intraoperative dysrhythmias. The only factor that correlated with intraoperative dysrhythmias was the presence of preoperative dysrhythmias (Pearson chi-square = 0.0062). For the hypokalemic group, a maximum chance of incurring serious intraoperative dysrhythmias was calculated.[1] At a 95% confidence level, this maximum chance was found to be 5%.

TABLE 1. Preoperative Characteristics

	Hypokalemic Group	Normokalemic Group
n	62	88
K+ (mEq/l) range	2.6–3.4	3.5–5.2
K+ (mEq/l) mean	3.1	4.1
Age (mean ± SD)	61 ± 12	63 ± 12
ASA physical status		
I	6% (4)	7% (6)
II	11% (7)	28% (25)
III	73% (45)	40% (35)
IV	10% (6)	25% (22)
Hypertension	82% (51)	24% (21)
Cardiac disease	16% (10)	27% (24)
Preop dysrhythmias	24% (15)	14% (12)
Hypoxia	11% (7)	6% (5)
Digitalis therapy	3% (2)	8% (7)

Discussion

Our results oppose the dogma that hypokalemia increases the chance of incurring dysrhythmias during anesthesia. Why should our findings deviate from expectations? One possible explanation is that our population sample is too small to detect a true difference among groups in the incidence of dysrhythmias, *i.e.*, we are drawing a falsely negative conclusion. However, beta error analysis[2] indicates only a 10% chance of a false negative. A second possible explanation is an error in potassium measurements. However, serum potassium

TABLE 2. Intraoperative Characteristics

	Hypokalemic Group	Normokalemic Group
n	62	88
Surgical site		
Aortic	3% (2)	3% (3)
Thoracic	2% (1)	15% (13)
Abdominal	19% (12)	15% (13)
Other	76% (47)	67% (59)
Primary anesthetic agent		
Opioid	2% (1)	3% (3)
Nitrous oxide	2% (1)	8% (7)
Halothane	9% (6)	9% (8)
Isoflurane	29% (18)	33% (29)
Enflurane	58% (36)	47% (41)
Intubated	90% (56)	88% (77)
Duration (mean ± SD)	93 ± 56 min.	89 ± 55 min.

TABLE 3. Incidence of Intraoperative Dysrhythmias

	Hypokalemic Group		Normokalemic Group		
K + (mEq/l)	2.6–2.9	3.0–3.4	3.5–3.9	4.0–4.4	4.5–5.2
n	21	41	39	32	17
PACs	14% (3)	10% (4)	13% (5)	16% (5)	6% (1)
PVCs	10% (2)	17% (7)	21% (8)	34% (11)	18% (3)
Bigeminy	0 (0)	7% (3)	3% (1)	0 (0)	0 (0)
Junctional	10% (2)	17% (7)	13% (5)	31% (10)	12% (2)
Sinus tachycardia	0 (0)	2% (1)	10% (4)	19% (6)	18% (3)
Total dysrhythmias	29% (6)	39% (16)	41% (16)	59% (19)	47% (8)

measurements are among the most accurate and reproducible laboratory tests. Although serum potassium levels fluctuate during the day, it is unlikely that a chronically depleted patient would attain normokalemia overnight, without potassium supplementation. A third possible explanation is an abnormal control group. However, the incidence of dysrhythmias in our normokalemic group is similar to that reported in other surveys.[3] Additionally, anesthetic records give no indication that special precautions or techniques were utilized in anesthetizing the hypokalemic patients. Therefore, such explanations cannot account for the results.

Other considerations may be more germane. Current beliefs about the effects of hypokalemia on cardiac cells are based predominantly on *in vitro* experiments.[4] Such experiments usually involved only one type of cardiac cell (fast fibers) obtained from several animal species. There was no opportunity for physiologic adaptation, and the effects of anesthetics were not included. In contrast, experiments conducted in anesthetized hypokalemic animals showed no increase in dysrhythmias.[5] Therefore, *in vitro* investigations may not be applicable to chronically hypokalemic patients receiving anesthetics. Electrocardiographic surveys on nonsurgical patients[6,7] also contribute to the concept that hypokalemic patients are prone to rhythm disturbances. Harrington *et al.* have raised serious doubts about the conclusions of those reports.[8] Our study is the only investigation that examines intraoperative cardiac rhythms in hypokalemic patients. The results challenge the validity of extrapolating from *in vitro* preparations and ECG surveys.

There are important limits to interpretations of our study. Our patients were asymptomatic, chronically hypokalemic people. The results may not pertain to patients with acute hypokalemia, patients receiving digitalis, symptomatic hypokalemic patients, or patients who are hypokalemic as a result of severe disease processes (*e.g.*, cirrhosis). Our data cannot be used to establish acceptable preoperative potassium levels. In order to determine the importance of a potassium value, the entire clinical situation must be evaluated. Finally, emphasis should be placed on the fact that we restricted our investigation to the examination of intraoperative dysrhythmias only. This means the data cannot be used to predict the role of hypokalemia in other complications, *e.g.*, renal tubular dysfunction, cirrhotic encephalopathy, myocardial response to hypoxia, and postoperative dysrhythmias.

This study does indicate that chronic hypokalemia alone is rarely responsible for serious intraoperative rhythm disturbances. Identifying the actual incidence would require several thousand intraoperative recordings. This may be unnecessary. The correlation of preoperative and intraoperative dysrhythmias implies that the preoperative ECG would identify those at risk.

Our results lead us to question two common practices in the management of chronically hypokalemic patients: automatic surgical postponement and aggressive potassium replacement. Postponement, potassium supplementation, and serial blood tests lengthen hospitalization and increase costs. The value of potassium replacement is questionable. There is evidence that vigorous potassium administration neither reliably restores normokalemia, nor consistently decreases dysrhythmias.[9] More importantly, potassium administration may precipitate serious morbidity.[10] It is estimated that one of every 200 patients receiving potassium will suffer fatal or life-threatening hyperkalemia.[11] In our community, perioperative potassium administration was associated with at least four cardiac arrests in 1 year.

Our data not only yield evidence that some beliefs about hypokalemia are unfounded but also suggest that routine surgical postponement and acute potassium replacement may be unnecessary practices in chronic hypokalemia.

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