Intensive Care Med (1983) 9:275–277

Intensive Care Medicine

Serum Potassium Levels During Prolonged Hypothermia

A. Koht, R. Cane and L. J. Cerullo

Departments of Anesthesia and Neurosurgery, Northwestern University Medical School, Chicago, Illinois, USA

Accepted: 18 January 1983

Abstract. Hypokalemia (mean serum potassium 2.3 ± 0.4 mEq/l) was observed in six hypothermic patients (30° – 32 °C) with head injuries or brain hypoxia. In the first three patients, potassium was administered to maintain serum levels above 3.5 mEq/l and on rewarming after 48 h of hypothermia hyperkalemia (peak serum potassium = 7.1 ± 0.5 mEq/l) associated with cardiac arrhythmias developed. The remaining three patients received sufficient potassium to approximately replace measured losses during the hypothermic period. These patients did not become hyperkalemic on rewarming. Clinically insignificant sinus bradycardia, premature atrial contractions and junctional rhythms were seen during hypothermia with hypokalemia. We conclude that hypothermia produces hypokalemia by a shift of potassium from the extracellular to intracellular or extra vascular spaces. Potassium therapy during controlled hypothermia in the range 30° – 32 °C should only replace measured losses.

Key words: Hypothermia – Hypokalemia – Potassium therapy

Hypothermia may be deliberately induced to reduce swelling of a brain damaged by hypoxia or trauma, to lower metabolic rate during cardiac surgery or may occur accidentally in nonsurgical patients, especially in colder climates. While many of the physiologic changes with hypothermia have been established, there is disagreement as to the effect of hypothermia on serum potassium levels [1 – 5]. Hypokalemia has been observed in hypothermic cardiac surgery patients and in dogs [6 – 9]. We wish to report the serum potassium levels observed in six patients deliberately cooled to about 30 °C as part of their neurosurgical management.

Methods

Six patients who had suffered head trauma or brain hypoxia had their body temperatures lowered to 30° – 32 °C (rectal and pulmonary artery) by surface cooling and maintained at this level for forty-eight hours. Additional therapy included Dexamethasone, 100 mg IV initially followed by 8 mg every 2 h and five patients received thiopental sodium 15 mg/kg followed by 4 mg/kg/h by continuous infusion. Arterial PcO_2 was maintained between 35 and 40 torr by controlled ventilation. The electrocardiogram was monitored. Arterial blood gases and electrolyte concentrations in serum and urine were measured. In the first three patients, potassium was infused to maintain serum potassium levels above 3.5 mmol/l. Because hyperkalemia had been observed on rewarming the first three patients, the next three patients were given potassium in quantities determined in part by measured urinary losses and in part by physician judgement. At the end of 48 h, the patients were rewarmed over a 6 – 8 h period to 37 °C (rectal). Elevated serum potassium levels in the first three patients were treated with sodium polystyrene sulfonate (Kayexalate), insulin and hypertonic glucose.

Results

The mean serum potassium concentrations of the two groups are summarized in Figure 1. In all six patients, when body temperature was lowered to 30° – 32 °C, serum potassium dropped from 4.7 ± 0.9 (SEM) mmol/l to 2.3 ± 0.4 mmol/l. In the first three patients serum levels were 3.6 ± 0.3 mmol/l for the 48 h of hypothermia. At the end of the rewarming period, serum potassium peaked at 7.1 ± 0.5 mmol/l. These levels were associated with cardiac arrhythmias (T-

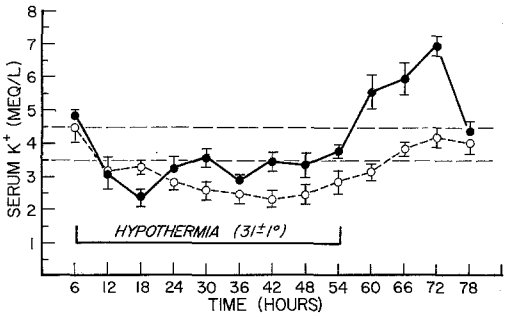

Fig. 1. Serum potassium levels in patients during and after induced hypothermia. Mean values I SEM (vertical bars) solid circles, three patients in whom serum potassium was maintained near normal values by potassium infusion. Open circles, three patients to whom potassium given only to replace measured losses

wave elevation, premature ventricular contractions). The arrhythmias disappeared when potassium concentrations were brought within normal limits.

In the second three patients, serum potassium levels dropped to 2.6 ± 0.4 mmol/l and remained at low levels for 48 h. Upon rewarming, the serum potassium levels rose to normal without overshoot, and the ECG remained normal.

In both groups during hypothermia, sinus bradycardia was seen and occasional premature atrial contractions or junctional rhythms occurred. No other arrhythmias were seen.

Table 1 summarizes the potassium concentration in serum and urine, the serum pH, the urine output and the volume of intravenous fluids and potassium therapy the patients received. pH remained in the range of 7.30 to 7.49. Intravenous fluids included 0.2 normal saline with 5% dextrose, 0.45 normal saline with 5% dextrose and 5% albumen.

Discussion

We cannot attribute the low potassium to alkalosis or loss in urine since the replacement of lost potassium alone during the hypothermic stage did not reverse the hypokalemia. Furthermore, in order to restore serum potassium to normal in the first group, it was necessary to infuse more potassium than had been lost during the hypothermic period.

Considering the rise in serum potassium with rewarming, it appears that potassium is not lost during hypothermia. Therefore, the decrease in serum potassium must involve a shift from the extracellular space to the interstitial or intracellular space during hypothermia with a reverse shift upon rewarming. The location of the sequestered potassium is uncertain,

Table 1. Potassium concentrations in serum and urine, serum pH, urine output, potassium therapy and intravenous fluids administered in six patients during 2 days of hypothermia and the 2 days immediately following hypothermia

Patient/ outcome	Day	pH LO – HI	Serum K mmol/l LO – HI	Urine K mmol/l	Urine output ml/24 h	IV fluids ml/24 h	K Therapy mmol/24 h
1. Survived	1	7.45 – 7.49	2.6 – 3.4	84	3280	3010	260
	2.	7.39 – 7.45	3.4 – 4.2	–	2010	2820	250
	3	7.38 – 7.42	4.5 – 6.6	87	2270	3140	80
	4	7.41 – 7.44	3.6 – 5.9	202	2940	2590	30
2, Expired	1	7.31 – 7.41	4.4 – 5.1	–	1380	2590	180
	2	7.31 – 7.39	2.0 – 3.3	8	1700	2380	360
	3	7.30 – 7.38	3.4 – 6.0	10	1565	2116	180
	4	7.38 – 7.45	5.5 – 7.1	–	1504	2390	–
3. Survived	1	7.35 – 7.42	1.9 – 3.8	5	6250	7500	540
	2	7.35 – 7.44	2.3 – 3.1	5	2108	4260	240
	3	7.32 – 7.39	4.9 – 7.1	–	4580	2420	–
	4	7.41 – 7.48	3.8 – 4.1	67	3750	1880	40
4. Expired	1	7.40 – 7.44	2.9 – 3.2	14	4545	3240	180
	2	7.38 – 7.42	2.7 – 5.0	–	2110	3550	120
	3	7.41 – 7.45	3.2 – 4.0	25	3000	2270	30
	4	7.45 – 7.49	3.5 – 3.6	–	1500	2650	90
5. Expired	1	7.38 – 7.42	3.0 – 3.3	–	920	2300	–
	2	7.37 – 7.41	2.1 – 3.1	–	1080	3930	–
	3	7.36 – 7.40	2.1 – 2.4	–	725	2630	90
	4	7.38 – 7.42	2.6 – 3.1	–	1700	2900	60
6. Survived	1	7.34 – 7.41	2.5 – 3.6	12	995	1800	20
	2	7.28 – 7.40	2.4 – 3.0	14	3090	3690	100
	3	7.41 – 7.44	2.9 – 4.0	10	4530	3550	20
	4	7.36 – 7.44	3.5 – 3.6	22	1220	2000	–

but it appears not to be within the red blood cell since blood cooled in vitro does not exhibit a shift in potassium [7, 9]. An increase in liver potassium in dogs during hypothermia has been observed [9]. Todd et al. demonstrated that propranolol pretreatment will prevent hypokalemia developing in dogs when rendered hypothermic [5]. They postulated that increases in circulating catecholamines produce the drop in potassium concentrations via a β-adrenergic mechanism.

We conclude that serum potassium concentration can decrease below the normal range during prolonged hypothermia and that these low levels of serum potassium do not significantly affect cardiac rhythm. We believe that during prolonged hypothermia, potassium should be administered only to replace measured losses.

Acknowledgements. The authors wish to thank Drs. H. Linde and P. Land for their assistance in the preparation of this manuscript.

References

1. Asami S (1975) Studies on electrolyte metabolism in serum and tissues during extracorporcal circulation and the surface cooling. Jpn J Thorac Surg 23:191
2. Black PR, Van Devanter S, Cohn LH (1976) Effects of hypothermia on systemic and organ system metabolism and function. J Surg Res 20:49
3. Chinard FP (1978) Accidental hypothermia – a brief review. J Med Soc NJ 75:610
4. Coniam SW (1979) Accidental hypothermia. Anaesthesia 34:250
5. Machida S, Ohta S, Itoh N (1977) Changes in tissue distribution of potassium during simple hypothermia. Jpn J Thorac Surg 30:413
6. O'Keefee KM (1977) Accidental hypothermia: A review of 62 cases. JACEP 6:491
7. Shida H, Morimoto M, Seki T, Inokawa K (1975) Metabolic changes in surface – induced deep hypothermia combined with cardiopulmonary bypass for cardiac surgery. Jpn J Surg 5:73
8. Shida H, Morimoto M, Sugenoya H, Seki T, Inokawa K (1973) Changes of serum electrolytes at open heart surgery with simple profound hypothermia: relation between transient hypopotassemia and metabolic acidosis. Jpn J Thorac Surg 26:723
9. Todd EP, Campbell HC, Utley JR, Miller RE, McAllister RG Jr, Wachtel CC, Collins JC, Erickson JL (1977) Effect of beta-adrenergic blockade on hypothermia – induced hypokalemia. Surg Forum 28:275

Dr. A. Koht
Department of Anesthesia
Northwestern University Medical School
303 East Chicago Avenue
Chicago, Illinois 60611
USA