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Hypokalemia in Trauma Patients

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There is a difference in opinion about whether hyperkalemia or hypokalemia is more common in severe trauma and shock. In traumatic or hemorrhagic shock, intracellular potassium leaks out of cells, resulting in hyperkalemia.¹⁻⁴ Smith reports that hypokalemia is more common in severely traumatized patients.⁵ A prospective study was conducted to resolve these reported differences and to identify possible causes for changes in serum K concentration following trauma.

MATERIALS AND METHODS

Two hundred and twelve consecutive trauma patients who were transported to the Maryland Institute for Emergency Medicine (MIEM) by helicopter were selected and studied. Estimated time between accident and arrival obtained from police records was $55 \pm \text{SD } 17$ min. Age of the patients studied averaged $31 \pm \text{SD } 13$ yr (ranging from 15 to 83 yr). There were 149 males and 63 females. One hundred ninety-nine patients were automobile accident victims, nine suffered from gunshot wounds, and four suffered from industrial accidents. Each injury to the head, chest, abdomen, or extremities and spine was counted as one body-section injury. On average, injuries were found in $1.8 \pm \text{SD } 0.81$ body sections. Twenty-seven patients were dead on arrival or were moribund and died

immediately after admission. Fifty-six of the 212 patients arrived with a systolic arterial blood pressure less than 90 mmHg.

In the 185 patients who were not dead or moribund on arrival, venous blood samples were taken following insertion of a venous catheter and before any treatment or iv fluid infusion therapy was initiated. Sometimes lactated Ringer's solution was infused during transport to the hospital. The venous blood was analyzed for serum electrolytes and blood glucose. Arterial blood was obtained by percutaneous femoral artery puncture at the time of the venous blood sampling and was analyzed for blood gases and lactic acid concentration when enough blood was available. Urine was analyzed for electrolytes if an admission urine sample was obtained. Following the sample taking, the patients were resuscitated with both plasma protein fraction and lactated Ringer's solution. In patients who required blood transfusion, packed red blood cells were administered with an equal volume of plasma protein fraction. For the maintenance of fluid balance, lactated Ringer's solution was used. In patients with serum K concentration less than 3.0 mEq/l, 10 mEq K as a chloride was added to the 1,000 ml iv solution administered. Serum electrolytes were monitored at least every 2 h during resuscitation and surgery. In the 27 patients who were dead on arrival or died shortly after arrival, only serum electrolytes were measured.

Admission serum K concentrations were correlated with admission arterial blood pressure, blood glucose, PaO_2 , PaCO_2 , pH, arterial lactic acid level, and urine K concentration with a Pearson product-moment correlation coefficient. The 185 surviving patients were divided into two groups: Group 1 included 56 patients who had an admission systolic arterial blood pressure less than 90 mmHg and Group 2 included 129 patients with an admission systolic pressure greater than 90 mmHg. Serum K concentrations, pH, blood, and iv fluid volumes during

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TABLE 1. Clinical Information during Resuscitation and Anesthesia

	Group 1	Group 2	Significance
Admission serum K (mEq/l)	3.5 ± 0.08 (56)	3.6 ± 0.05 (129)	NS
Resuscitation*			
Duration (h)	2.3 ± 0.16	2.8 ± 0.07	<0.01
Packed red blood cells (units)	5.5 ± 0.55	1.4 ± 0.13	<0.001
Plasma protein fraction (ml)	1734 ± 117	1447 ± 50	<0.01
Lactated Ringers solution (ml)	1472 ± 130	1229 ± 55	<0.05
KCl administered† (mEq)	29.9 ± 5.29	11.5 ± 1.65	<0.01
Serum K before anesthesia (mEq/l)	3.3 ± 0.07 (52)	3.5 ± 0.05 (112)	<0.01
Anesthesia			
Duration (h)	5.2 ± 0.49	3.9 ± 0.24	<0.01
Packed red blood cells (units)	9.4 ± 1.73	3.5 ± 0.33	<0.001
Plasma protein fraction (ml)	1669 ± 406	1131 ± 79	NS
Fresh frozen plasma (units)	5.7 ± 1.15	1.6 ± 0.25	<0.001
Lactated Ringers solution (ml)	2990 ± 504	2059 ± 204	<0.01
Other solutions not containing K (ml)	941 ± 267	238 ± 61	<0.001
KCl administered† (mEq)	35.4 ± 5.771	24.6 ± 3.39	NS
Serum K at the end of anesthesia (mEq/l)	3.7 ± 0.13 (45)	3.8 ± 0.05 (112)	NS
Serum K 18-24 h following anesthesia (mEq/l)	4.3 ± 0.1 (38)	4.2 ± 0.04 (107)	NS

Number of patients in parentheses.
NS = $P > 0.05$ on t test.
Values are mean ± SEM.

* Admission to induction of anesthesia.
† Not including K in lactated Ringer's solution.

resuscitation and surgery in the two groups were compared with the use of unpaired t test.

RESULTS

Admission serum K concentration of all 212 patients averaged $3.61 \pm \text{SEM } 0.04$ mEq/l ranging from 1.9 to 5.1 mEq/l. In 108 of the 212 patients, serum K concentration was less than 3.5 mEq/l, which is the lower normal laboratory limit (3.5–5.5 mEq/l) at MIEM. The average serum K concentration of the 27 patients who were dead or moribund on arrival was $3.9 \pm \text{SEM } 0.20$ mEq/l. The admission serum K concentration of the Group 1 and Group 2 was $3.5 \pm \text{SEM } 0.08$ and $3.6 \pm \text{SEM } 0.05$ mEq/l, respectively. There were no statistically significant differences in admission serum K concentration between these groups. There were poor correlations between admission serum K concentration and other admission variables such as systolic arterial blood pressure, $p\text{H}$, PaCO_2 , blood glucose, and number of body sections injured ($-0.3 < r < 0.3$). Arterial lactic acid levels and urinary K concentrations were available in only 55 and 36 patients, respectively. There were no correlations between serum K concentration and lactic acid or urinary K values. ($-0.2 < r < 0.2$).

Serum K concentration remained low in the 185 patients during resuscitation and anesthesia. However, in the Group 1 patients who were in shock on admission, serum K concentration further decreased during resuscitation and the lowest value averaged $2.9 \pm \text{SEM } 0.06$ mEq/l, which was significantly lower ($P < 0.01$) than $3.4 \pm \text{SEM } 0.05$ of the Group 2 patients who did not have hypotension on admission. Group 1 patients received sig-

nificantly more blood and iv fluid volume during their shorter resuscitation (table 1). Despite the greater amount of KCl administered during resuscitation, serum K concentration measured before the induction of anesthesia was significantly lower in Group 1 than in Group 2.

There was no difference in serum K concentration at the end of anesthesia and the amount of KCl administered during anesthesia between the two groups. During anesthesia Group 1 received significantly more volume of blood and iv fluids except plasma protein fraction (table 1). There was no difference in PaCO_2 or $p\text{H}$ between the two groups during resuscitation and anesthesia.

Only two of the Group 1 patients had a serum K concentration greater than 6 mEq/l during anesthesia: both sustained traumatic thoracic aorta rupture and were undergoing surgery using aortic cross-clamping. Both patients died of uncontrolled bleeding shortly after the unclamping of the aorta and hyperkalemia was noted following unclamping. Average serum K concentration 18–24 h following surgery was $4.3 \pm \text{SEM } 0.1$ and $4.2 \pm \text{SEM } 0.04$ in Group 1 and Group 2 patients, respectively. These values are significantly greater than the admission serum K concentrations ($P < 0.01$).

DISCUSSION

The results of this study are contrary to the reports that serum K concentration increases in trauma patients and following shock.¹⁻⁴ In animal experiments an increase in serum K concentration was observed 20 min following hemorrhagic shock.^{2,3} In our study, hypokalemia was seen in more than one-half of the trauma patients arriving, on average, 55 min following trauma. Hypokalemia was ob-

served even in the presence of acidosis, hypercarbia, and severe injury, which often cause hyperkalemia.^{1,6-8} Illner *et al.* report that serum K concentration was lower in patients in shock of longer duration than in those in shock of shorter duration.⁴ Their report and our observations suggest that, in trauma, serum K concentration is influenced by factors other than shock, the duration of shock, *pH*, *PaCO₂*, or muscle damage. Hypokalemia occurs with potassium loss, hyperadrenocorticism, lack of dietary intake, alkalosis, or increase in catecholamine level.⁹⁻¹² Hypokalemia during the first several hours of trauma is not likely from potassium loss, lack of dietary intake, alkalosis, or adrenal gland insufficiency. Epinephrine might have played a role in decreasing serum K concentration in trauma patients. β -adrenergic stimulation from epinephrine causes a decrease in serum K concentration by driving K into the cells.^{11,12} Rosa *et al.* suggested that serum K concentration may change in trauma patients who respond to injury by releasing catecholamines.¹¹ Although epinephrine levels were not measured in our study, it is likely that epinephrine levels were high in all these patients following trauma. Serum K concentration may be erroneously low if the blood sample is obtained through a central venous catheter during massive fluid infusion. This possibility is ruled out because the patients were only receiving K-containing lactated Ringer's solution on admission.

It is not clear why serum K concentration further decreased during resuscitation in the Group 1 patients. The amount of blood and iv fluid administered was greater in Group 1. However, it is not clear if serum K change during resuscitation was due to the differences in blood loss and the iv fluid volume replaced. Serum K concentration at the end of anesthesia was similar, although Group 1 received more blood and iv fluid. It is possible that differences in catecholamine levels were related to the changes in serum K.

The impact of hypokalemia on trauma patients is not known. Smith reported that hypokalemia was associated with severe trauma and a high mortality.⁵ Whether this high mortality was due to hypokalemia or other factors is not clear. Dysrhythmias, muscle weakness, gastrointestinal atony, and polyuria are all associated with hypokalemia.^{8,13} However, acute hypokalemia may not be associated with those symptoms and signs.¹⁴ We empirically

administered KCl to restore serum K concentration to within normal limits during this study. Whether or not this practice is appropriate should be investigated.

In summary, hypokalemia is commonly found in trauma patients whether or not injuries and shock are severe. Further studies are required to understand K metabolism following trauma and the impact of hypokalemia on trauma patients.

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