

## A250

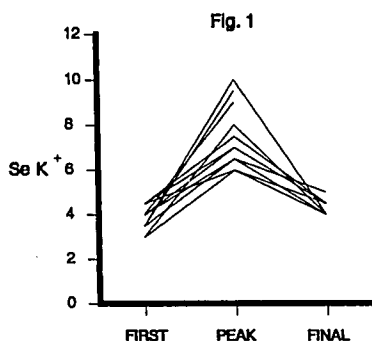
**TITLE:** HYPERKALEMIA WITH BLOOD TRANSFUSION IN EMERGENT AND TRAUMA SURGERY**AUTHORS:** M Wall, MD, EG Pavlin, MD**AFFILIATION:** Department of Anesthesiology, University of Washington, Seattle, WA 98104

Hyperkalemia has been reported with rapid and massive transfusions of blood in a variety of patients<sup>1,2</sup>. On at least one occasion, death from hyperkalemia was attributed to a rapid blood infusion rate<sup>3</sup>. The purpose of this study was to examine the occurrence of hyperkalemia in patients presenting for emergent surgery, for either trauma or ruptured abdominal aortic aneurysm (AAA) who required homologous blood transfusion.

**METHOD:** The study met the requirements of the institutional human subjects review board. In a 6 month period, 11 patients who received blood became hyperkalemic in the operating room. These patients all presented to the ER with a normal serum potassium (SeK<sup>+</sup>), required urgent surgery for hemorrhage. A retrospective review was done and electrolytes and arterial blood gases, core temperatures, volume of blood and fluid transfused, electrocardiograms and other data were obtained from the ER, OR and ICU. We hypothesized that a period of preceding shock, hypothermia and acidosis might contribute to hyperkalemia.

**RESULTS:** Hyperkalemia occurred in 11 patients age 15-84. Operations performed were 5 aortic aneurism resections, 5 laparotomy/thoracotomy, and 1 craniotomy. 10/11 patients had the aorta crossclamped and their hyperkalemia occurred before the clamp was removed. 8/11 patients were in shock preoperatively. They received a mean of 14.7 units of PRBC and WB (Range 5-28 units). Table 1 shows mean peak SeK<sup>+</sup> value, and the SeCa<sup>++</sup>, pHa and temp at the time of the peak K<sup>+</sup>. Fig. 1 shows the individual SeK<sup>+</sup> prior to surgery, at peak SeK<sup>+</sup> and post surgery. All patients had normal or low SeK<sup>+</sup> initially. Peak SeK<sup>+</sup> occurred in the OR with 3 patients developing SeK<sup>+</sup> > 9.0 meq/l. All three had cardiac arrest with arrhythmias typical of acute hyperkalemia, and all 3 died in the O.R. 2 others had peaked T<sub>w</sub> waves consistent with hyperkalemia.

Table I. Peak Measure	Mean Value	Range
SeK <sup>+</sup>	7.4meq/l	(5.8-10.0)
SeCa <sup>++</sup>	0.92meq/l	(0.5-1.4)
pHa	7.19	(7.03-7.31)
core temp	33.4°C	(31.8-35.4)



**DISCUSSION:** Ordinarily K<sup>+</sup> in stored blood causes little increase in SeK<sup>+</sup>. In this group hyperkalemia occurred resulting in EKG changes in 5/11 patients. Hypocalcemia, acidosis and hypothermia are all treatable abnormalities that can exacerbate hyperkalemic arrhythmias. In this group blood transfusion was variable (3 pts < 8 units, only 2 > 20 units) and did not correlate with the degree of hyperkalemia. We propose that hypothermia, shock, and aortic cross clamping may decrease the volume of distribution of the potassium transfused, and thus cause hyperkalemia in this patient group.

**REFERENCES:** 1. Acta Anaes Scand 28:220-221, 1984 2. Can J Anaes 47:747-54, 1990 3. Anesthesiology 73:1050-1052, 1990 [Supported in part by NIH grant GM37619]

## A251

**Title:** EFFECTS OF THE PNEUMATIC TOURNIQUET ON CIRCULATORY VARIABLES, PLASMA CATECHOLAMINES AND RENIN ACTIVITY.**Authors:** N. R. Fahmy, M.D.**Affiliation:** Department of Anesthesia, Harvard Medical School at the Massachusetts General Hospital, Boston, MA 02114.

Pneumatic tourniquets are frequently used during operations on the extremities to provide a bloodless surgical field. The depth of anesthesia is usually increased to prevent the elevation of blood pressure with tourniquet use. The incidence of hypertension ranges from 11% to 49%. Its cause has not yet been identified. We report the changes in plasma concentrations of catecholamines and plasma renin activity (PRA) as well as circulatory variables during tourniquet application in man.

Forty patients undergoing operations on the legs and requiring use of a tourniquet gave consent to an institutionally approved protocol. They were ASA I or II. Thirty patients had general anesthesia (GA) with N<sub>2</sub>O-O<sub>2</sub>-enflurane and increments of fentanyl given as needed. Ten patients had spinal anesthesia (SA) with tetracaine (0.5%); dermatomal level of sensory blockade to pin-prick was T<sub>6</sub> to T<sub>10</sub>. Arterial blood pressure (BP) (automatic blood pressure monitor, Dinamap) and heart rate (ECG) were recorded every 5 min during operation. Blood samples were obtained before and every 15 min during tourniquet application and every 5 min for 20 min after tourniquet release. The plasma was separated and analyzed for its catecholamine content (high pressure liquid chromatography) and PRA (radioimmunoassay). Analysis of variance and regression analysis were used to determine significance (P < 0.05) of the data.

Pre-operative BP was similar in both groups. Three patients in the SA group required neosynephrine for 30 min. In the GA group, BP increased significantly; the most significant change occurred in patients with pre-operative hypertension (n = 9). A small but significant rise in BP occurred in the SA group. There was a significant increase in plasma norepinephrine (NE) from 312 ± 44 to 635 ± 62 pg.ml<sup>-1</sup>; P < 0.01, epinephrine (E) (from 62 ± 12 to 93 ± 17 pg.ml<sup>-1</sup>; P < 0.05) and PRA (from 2.38 ± 0.5 to 6.4 ± 1.9 ng.ml<sup>-1</sup>.h<sup>-1</sup>; P < 0.01) with tourniquet application in the GA group. The increase in BP correlated with the rise in plasma NE and PRA. With SA, these variables did not change.

BP increases significantly in patients under GA; a small increase occurs with SA. Blood pressure starts to increase after 45 min and continues to rise during tourniquet application. This is in keeping with reports of Kaufman<sup>1</sup> and Valli and Rosenberg.<sup>2</sup> The magnitude of the rise in BP is significantly more in patients with pre-operative hypertension than in normotensive patients. Tourniquet-induced hypertension correlates with an increase in plasma NE and PRA.

The increase in plasma catecholamines and PRA may be partly responsible for the rise in BP. Rocco et al<sup>3</sup> found no changes in plasma catecholamines. Further studies are needed to clarify these differences.

**References:**

1. Br J Anaesth 54:333-336, 1982.
2. Acta Anaesthesiol Scand 29:142-147, 1985.
3. Reg Anesth 14:174-180, 1987.