The Effect of Different Crystalloid Solutions on Acid-Base Balance and Early Kidney Function After Kidney Transplantation

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BACKGROUND: This study aimed to quantify changes in acid-base balance, potassium and lactate levels as a function of administration of different crystalloid solutions during kidney transplantation, and to determine the ideal fluid for such patients.
METHODS: In this double-blind study, patients were randomized to three groups ($n = 30$ each) to receive either normal saline, lactated Ringer's, or Plasmalyte, all at 20–30 mL · kg^{-1} · h^{-1}. Arterial blood analyses were performed before induction of anesthesia, and at 30-min intervals during surgery, and total IV fluids recorded. Urine volume, serum creatinine and BUN, and creatinine clearance were recorded on postoperative days 1, 2, 3, and 7.
RESULTS: There was a statistically significant decrease in pH (7.44 ± 0.50 vs 7.36 ± 0.05), base excess (0.4 ± 3.1 vs −4.3 ± 2.1), and a significant increase in serum chloride (104 ± 2 vs 125 ± 3 mM/L) in patients receiving saline during surgery. Lactate levels increased significantly in patients who received Ringer's lactate (0.48 ± 0.29 vs 1.95 ± 0.48). No significant changes in acid-base measures or lactate levels occurred in patients who received Plasmalyte. Potassium levels were not significantly changed in any group.
CONCLUSIONS: All three crystalloid solutions can be safely used during uncomplicated, short-duration renal transplants; however, the best metabolic profile is maintained in patients who receive Plasmalyte.
(Anesth Analg 2008;107:264–9)

Patients undergoing renal transplantation are subject to a wide variety of intraoperative complications including hemodynamic instability, acid-base and electrolyte disturbances because of impaired renal function, and co-morbid diseases.[1] Maintenance of intravascular volume during kidney transplantation is crucial to ensure optimal graft perfusion and function.[2] Crystalloids alone are usually sufficient to maintain volume during kidney transplantation, carry no infectious risk, and have no specific nephrotoxic effects.[3–5]

Previous studies have shown that metabolic acidosis is a complication of normal saline infusion because of its high chloride content. Ringer's lactate contains potassium and can potentially aggravate hyperkalemia in patients with impaired renal function.[6] Despite widespread use of Plasmalyte for patients with renal compromise, Plasmalyte has not specifically been compared with other crystalloids in patients undergoing kidney transplantation. For this reason, we designed a study comparing metabolic profile and renal function in renal transplant patients managed with saline, Ringer's lactate, or Plasmalyte.

METHODS

Following approval of the Hospital Ethics Committee, written informed consent was obtained from all patients. This prospective, randomized, double-blind study was conducted on 90 patients, aged 18–65 yr, ASA III and IV scheduled for living-related kidney transplantation. Exclusion criteria were severe cardiovascular disease, liver dysfunction, cadaveric kidney transplantation, diabetes, and serum potassium level >5.5 mM/L.

A computer randomization program was used for patient group assignments. Patients received 0.9% normal saline, Ringer's lactate, or Plasmalyte with 30 patients in each group. The study solutions were prepared in unlabeled bags by the hospital pharmacy. Patients and clinicians were blinded to group assignments. Salt make-up the study solutions is shown in Table 1.

Before induction of anesthesia, an 18-G IV catheter and 20-G arterial artery cannula were inserted under local anesthesia. After preoxygenation, general anesthesia was induced using IV thiopental (3–5 mg/kg),

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Accepted date for publication March 3, 2008.

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DOI: 10.1213/ane.0b013e3181732d64

Table 1. Electrolyte Composition of the Solutions and Normal Blood Plasma (mM/L)

	Sodium	Potassium	Chloride	Lactate	Calcium	Magnesium	Bicarbonate
Normal saline	154		154				
Lactated Ringer's	130	4	115	28	3		
Plasmalyte	140	5	98			3	27 (acetate) 23 (gluconate)
Normal blood plasma	134–146	3.4–5	98–108		2.25–2.65	0.7–1.1	22–32

fentanyl (2μg/kg), and cisatracurium (0.15 mg/kg) and was maintained with sevoflurane 0.4%–1.5% in an oxygen-nitrous oxide mixture at a 1:1 ratio; additional doses of fentanyl and cisatracurium were administered as appropriate. Central venous catheter was inserted after induction of anesthesia in the right internal jugular vein. IV fluids were given at a rate of 20–30 $mL \cdot kg^{-1} \cdot h^{-1}$ to maintain CVP at 12–15 mm Hg, and the total volume of fluids was recorded. During surgery, the patient's temperature was kept constant at 36°C and $Paco_2$ was maintained at 30–35 mm Hg.

Each recipient was given methylprednisolone 500 mg at induction of anesthesia, and was placed on the same immunosuppressive protocol postoperatively.

At the end of surgery, study fluid was discontinued and all the patients received infusion of dextrose 5%/0.45% normal saline at rate of 70 mL/h. The hourly urine output was replaced with 0.45% normal saline 1 mL for each mL of urine.

Arterial blood samples were sent for analysis (before induction of anesthesia, throughout the surgical procedure at 30-min intervals, and at the end of operation) for measurement of pH, arterial carbon dioxide tension ($PAco_2$), serum sodium, potassium, chloride, and lactate (Radiometer ABL 620 GL, Radiometer CO, Copenhagen, Denmark). Additionally serum bicarbonate concentration and base excess were determined with a blood gas analyzer (Cobas b 221, Roche, Germany).

Total urine volume, serum creatinine, BUN and chloride levels were recorded every 24 h until the 3rd postoperative day (POD), then once on the 7th POD. Creatinine clearance was calculated from 24-h urine sample collected on PODs 1, 2, 3, and 7.

Data throughout the study are shown as mean ± SD (SD). Demographic and perioperative data were compared using the Student's *t*-test. All statistical analyses were performed using Statistical Package for Social Sciences version 11.0 for Windows (SPSS, Chicago, IL).

Table 2. Patient Demographic and Perioperative Variables

	Group normal saline (n = 30)	Group lactated Ringer's (n = 30)	Group Plasmalyte (n = 30)
Donor age (yr)	44.4 ± 12.5 (28–60)	48.3 ± 11.5 (30–59)	46.3 ± 12 (25–59)
Donor weight (kg)	72.3 ± 15.6 (51–80)	68.9 ± 19.8 (48–76)	70.2 ± 18.9 (55–83)
Recipient age (yr)	37.7 ± 11.3 (18–52)	35.8 ± 11.2 (18–53)	37.6 ± 9.9 (18–53)
Height (cm)	164.4 ± 8.8 (152–175)	166.8 ± 9.2 (145–175)	169.9 ± 4.5 (150–174)
Body weight (kg)	63.3 ± 10.3 (48–82)	63.0 ± 10.3 (47–80)	62.8 ± 9.5 (47–80)
Baseline serum albumin values (g/dL)	3.8 ± 0.5 (2.8–4.9)	3.6 ± 0.5 (2–3.5)	3.7 ± 0.5 (2.5–3.8)
Preoperative hemo/peritoneal dialysis (n)	24/6	21/9	24/6
Reperfusion time (min)	29.2 ± 7.8 (19–50)	30.6 ± 10.7 (18–56)	31.8 ± 3.3 (20–45)
Duration of arterial anastomosis (min)	13.6 ± 5.5 (9–25)	14.4 ± 6.2 (5–30)	16.3 ± 1.7 (10–30)
Duration of venous anastomosis (min)	15.5 ± 4.3 (10–30)	16.2 ± 4.2 (10–25)	15.5 ± 2.1 (10–21)
Duration of ureteric anastomosis (min)	22.0 ± 7.9 (15–40)	21.9 ± 6.6 (15–40)	20.6 ± 2.2 (15–30)
Operative time (min)	119.5 ± 12.77 (120–300)	118.3 ± 10.46 (110–295)	118.1 ± 17.30 (100–250)
Cold ischemic time (min)	31.5 ± 8.1 (15–35)	32.4 ± 7.5 (17–32)	33.9 ± 7.3 (13–30)
CVP at time of clamping (mm Hg)	12.5 ± 3.3 (10–25)	11.3 ± 4.3 (7–24)	12.4 ± 2.4 (6–20)
CVP at time of declamping (mm Hg)	16.3 ± 2.4 (9–25)	15.6 ± 4.1 (8–24)	16.3 ± 3.2 (9–23)
Intraoperative dose of furosemide (mg)	65.4 ± 34.0† (20–160)	102.0 ± 56.5 (40–200)	103.3 ± 34.1 (40–200)
Intraoperative dose of mannitol (mg)	26.0 ± 6.6† (100–200)	37.7 ± 13.5 (75–300)	31.0 ± 13.8 (50–250)
Volume of study fluid (mL)	2868 ± 780 (1000–3500)	2770 ± 820 (1750–4000)	2756 ± 800 (1750–3300)

Data are expressed as mean ± SD and (range).

* $P < 0.05$ (intragroup difference).

† $P < 0.05$ (intergroup difference).

Table 3. Electrolyte and Acid-Base Patterns

	pH	Base excess (mEq)	Lactate (mM/L)
Saline group			
0 min	7.44 ± 0.50 (7.30–7.58)	0.42 ± 3.11 (−10 to 6.4)	0.56 ± 0.64 (0.2–2.4)
30 min	7.42 ± 0.06 (7.33–7.56)	−0.95 ± 3.07 (−10.4 to 3.6)	0.68 ± 0.71 (0.1–2.6)
60 min	7.40 ± 0.06*† (7.33–7.52)	−1.49 ± 3.69*† (−12 to 3.6)	0.66 ± 0.79 (0.1–2)
90 min	7.38 ± 0.73*† (7.31–7.53)	−2.02 ± 3.53*† (−10.4 to 3.3)	0.61 ± 0.55 (0.1–2.1)
At the end	7.36 ± 0.05*† (7.35–7.50)	−4.29 ± 2.12*† (−7 to 2)	0.58 ± 0.53 (0.2–1.8)
Ringer's lactate group			
0 min	7.44 ± 0.05 (7.34–7.56)	0.72 ± 1.44 (−7.1 to 5.3)	0.48 ± 0.29 (0.1–1.1)
30 min	7.43 ± 0.06 (7.32–7.54)	−0.84 ± 1.80 (−10.0 to 6.3)	0.86 ± 0.33* (0.2–1.6)
60 min	7.43 ± 0.05 (7.33–7.60)	−0.77 ± 1.44 (−7.0 to 4.3)	1.42 ± 0.45*† (0.2–1.9)
90 min	7.42 ± 0.06 (7.35–7.56)	−0.85 ± 1.47 (−8.9 to 4.1)	1.68 ± 0.44*† (0.6–2.6)
At the end	7.42 ± 0.06 (7.36–7.52)	−0.98 ± 1.06 (−7.6 to 4.0)	1.95 ± 0.48*† (0.9–2.4)
Plasmalyte group			
0 min	7.42 ± 0.06 (7.25–7.54)	0.44 ± 1.71 (−14 to 3.9)	0.61 ± 0.58 (0.1–2.8)
30 min	7.44 ± 0.07 (7.20–7.54)	−0.45 ± 1.53 (−10 to 4)	0.68 ± 0.61 (0.1–2.5)
60 min	7.44 ± 0.08 (7.20–7.54)	−0.56 ± 1.87 (−11.2 to 4.8)	0.73 ± 0.60 (0.1–2.4)
90 min	7.44 ± 0.07 (7.20–7.53)	−0.61 ± 1.87 (−10.7 to 5)	0.65 ± 0.62 (0.1–3.8)
At the end	7.44 ± 0.06 (7.20–7.48)	−0.56 ± 1.95 (−10.7 to 2.5)	0.62 ± 0.78 (0.1–2.9)

Data are expressed as mean ± SD and (range).

* $P < 0.05$ (intragroup difference).

† $P < 0.05$ (intergroup difference).

Comparison of data between groups was determined by using one-way analysis of variance and Tukey Honestly Significant Difference Intragroup analyses were performed with paired *t*-tests. For all tests, values were considered to be statistically significant at $P < 0.05$.

RESULTS

The demographic characteristics of the recipients and their corresponding donors were similar in the three groups (Table 2). All groups were hemodynamically stable, received similar volumes of fluid during surgery, and no patient received colloid or blood products. There were no significant differences between groups with respect to donor cold ischemia time, or duration of anastomoses ($P > 0.05$). The amount of intraoperative diuretics administered was significantly lower in the saline group compared with the other groups (Table 2).

pH decreased significantly from 7.44 to 7.36 in patients receiving saline, but no patients developed acidosis. pH did not change significantly in the other two groups. Changes is base excess corresponded to those in pH with a significant fall in base excess only in the saline group, from 0.4 to −4.9. Saline infusion also resulted in a significant decrease in bicarbonate levels, unique to this group (Table 3).

Similarly only the saline group had a significant elevation in serum chloride levels during surgery (104.2 ± 3.2 – 125.4 ± 3.7 mM/L). Chloride levels in these patients continued to be significantly elevated until POD 3 (Table 3).

Patients receiving Ringer's lactate showed a significant, progressive increase in lactate levels (0.48–1.95 mM/L) without a change in pH. Lactate was not significantly changed in the other groups. No groups experienced significant changes in serum potassium during the surgery. Bicarbonate, pH, lactate, base excess and potassium levels are shown in Figure 1.

No patients received insulin during the procedure, and blood glucoses ranged between 100 and 140 mg/dL. $CaCl_2$ was administered as needed to maintain ionized calcium above 0.7 mM/L.

The cumulative postoperative urine output was significantly larger in normal saline group on POD 1, 2, 3 compared with Ringer's lactate group and Plasmalyte group (Table 4). There were no significant differences between groups in postoperative renal function tests or the need for hemodialysis (Table 4).

DISCUSSION

Isotonic crystalloid solutions are the first choice for volume restoration during renal transplantation, but various crystalloid solutions can impact electrolyte and acid-base balance differently.[7] Our study indicates that Plasmalyte did not result in decreased pH, bicarbonate or increased chloride, as seen in patients who received saline. Lactate levels were also unchanged in patients who received Plasmalyte whereas patients receiving Ringer's lactate did have increased lactate levels at the end of surgery.

Renal impairment is associated with hyperchloremic metabolic acidosis, which can be exacerbated by saline infusion.[7–9] Normal saline is significantly hypertonic (osmolality 308 mOsm/L) and contains a very high content of chloride (Table 1).The incidence of hyperchloremic metabolic acidosis in patients who receive saline infusions is not well-established.[10,11] This acidosis may contribute to decreased splanchnic perfusion, as judged by reduced urine flow and abdominal discomfort in healthy volunteers.[12] Chloride ions have been shown to regulate renal vascular

Table 3. Continued

Bicarbonate (mM/L)	Potassium (mM/L)	CO_2 (mm Hg)	Chloride (mM/L)
22.2 ± 4.4 (18–31)	4.2 ± 0.4 (2.6–5.7)	34.5 ± 5.0 (27–46)	104.2 ± 3.2
21.5 ± 4.0 (16.1–28)	3.7 ± 0.6 (2.4–5.1)	29.2 ± 4.8 (27–38.2)	107.5 ± 3.4
21.1 ± 3.3 (14.7–26)	3.5 ± 0.3 (2.5–5.6)	28.5 ± 4.4 (23.6–35.8)	114.6 ± 4.1
20.1 ± 3.3 (16.2–33.9)	3.6 ± 0.5 (2.5–5.8)	28.8 ± 5.0 (25.6–37.8)	121.4 ± 2.9
18.2 ± 2.9*† (17–27)	3.9 ± 0.4 (3–5.8)	29.0 ± 4.0 (26–40.2)	125.4 ± 3.7*†
22.12 ± 3.45 (19.2–28.1)	4.1 ± 0.50 (2.9–4.9)	34.3 ± 5.0 (27.5–41)	102.4 ± 2.1
21.80 ± 3.51 (19.1–28.0)	3.8 ± 0.70 (2.6–5.5)	31.2 ± 5.7 (22–40)	102.5 ± 2.4
21.20 ± 3.00 (18.8–28)	3.9 ± 0.35 (2.6–4.6)	30.5 ± 3.9 (24.2–37.3)	103.3 ± 3.0
20.94 ± 2.84 (18.8–25.8)	3.4 ± 0.25 (2.9–5.9)	29.1 ± 3.5 (23.3–35.3)	104.7 ± 1.9
21.41 ± 3.70 (18.7–28.3)	3.8 ± 0.40(2.9–5.7)	29.2 ± 3.8 (27–43.5)	105.7 ± 1.3
22.11 ± 3.37 (16.6–29)	4.2 ± 0.20 (2.8–5.0)	30.3 ± 6.4 (27.1–44)	104.8 ± 1
22.10 ± 3.81 (14.0–26.6)	3.9 ± 0.25 (2.8–5.7)	29.7 ± 3.0 (25–39)	104.8 ± 1.6
22.13 ± 3.27 (14.2–29.3)	3.7 ± 0.30 (2.9–5.5)	28.7 ± 3.5 (23.4–36)	105.6 ± 2.1
22.16 ± 3.18 (14.5–30.5)	3.3 ± 0.20 (2.9–5.2)	28.8 ± 4.0 (23–39.7)	105.8 ± 2.5
22.61 ± 3.58 (14.0–26.4)	3.8 ± 0.25 (2.8–5.0)	29.3 ± 7.1 (25–46)	106.5 ± 2.2

resistance markedly within the clinical range.[13] Studies in dogs suggest that hyperchloremia leads to renal arteriolar vasoconstriction by inhibiting the intrarenal release of renin and angiotensin II, reducing glomerular filtration and urine output.[14–16] In our study, despite hyperchloremia, urine output increased significantly postoperatively in the saline group, likely because improved function of the graft outweighed the effect of hyperchloremia, and perhaps because of lack of sympathetic supply to the graft.

No patient in the saline group developed frank acidosis, despite the hyperchloremia. Development of acidosis is a consequence of central filling volumes, the composition of plasma and extracellular fluids, as well as the rate and composition of fluid losses.[17] The short duration of surgery in our patients and low blood loss also likely contributed to the relatively stable pH during surgery.

The acid-base changes that accompany hyperchloremia are related to the changes in strong ion difference, the difference in strong cations (sodium, potassium) and strong anions (chloride, lactic acid) and albumin concentration.[18] Because albumin levels were not different between groups in our study, the hyperchloremia following saline infusion is likely the major factor in development of acidosis in that group of patients. These data as well as other studies[11,19] also suggest that chloride, rather than volume expansion, is the major factor in the development of acidosis after saline administration. Hyperchloremia is difficult to treat, and it took a week for chloride levels to return to normal after transplantation in the saline group.[20]

Lactated Ringer's is also not an ideal solution. It is moderately hypotonic (osmolarity 273 mOsm/L), the Cl-content is substantially higher than plasma chloride, and the Na^+ content is lower (Table 1). Furthermore, the incomplete ionization of lactate salts means that the measured osmolality is only 255 mOsm/L. Most authorities recommend limited use of Ringer's lactate in patients at risk of cerebral edema.[21,22]

In our study, patients receiving lactated Ringer's showed a significant, progressive increase in lactate levels. Lactate is rapidly metabolized to CO_2 and water, which may lead to metabolic alkalosis, despite the fact that the pH of lactated Ringer's is 6.5. The

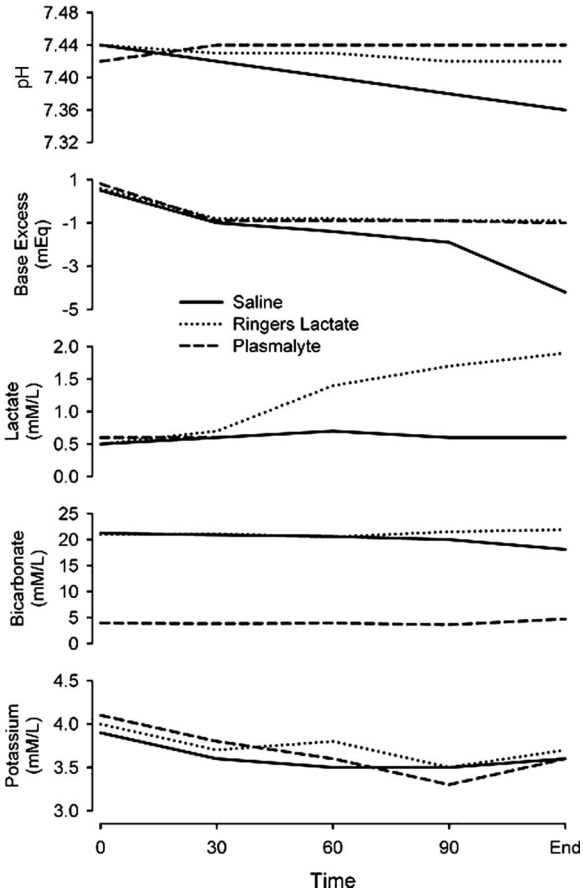

Figure 1. The mean values of pH, base excess, bicarbonate, lactate and potassium levels in groups.

Table 4. Postoperative Renal Function

	Creatinine (mg/dL)	BUN (mg/dL)	24-h urine output (L)	Creatinine clearance (mL/min)	Chloride level (mM/L)	Patients requiring postoperative dialysis (no)
Saline group						
POD1	4.4 ± 3.1	38.4 ± 20.3	11.4 ± 2.2†	85 ± 32.5	124.2 ± 2.9†	3
POD2	2.2 ± 2.7*	32.5 ± 22.2*	7.2 ± 3.0*†	106 ± 23.3*	120.3 ± 2.7†	
POD3	2.1 ± 2.2*	28.5 ± 18.4*	6.5 ± 2.5*†	109 ± 35.5*	113.2 ± 4.1†	
POD7	1.5 ± 1.0*	22.6 ± 10.9*	2.1 ± 1.6*	105 ± 29.1*	105.3 ± 3.3*	
Ringer's lactate group						
POD1	3.7 ± 1.9	34.0 ± 11.8	8.5 ± 2.9	77 ± 29.3	104.7 ± 1.2	2
POD2	2.2 ± 1.4*	26.0 ± 11.7*	6.7 ± 1.7	97 ± 40.4*	104 ± 3.2	
POD3	1.8 ± 1.4*	25.3 ± 12.2*	5.4 ± 1.6	103 ± 31.2*	102.4 ± 2.5	
POD7	1.5 ± 0.3*	20.5 ± 7.1*	1.8 ± 1.6*	110 ± 26.4*	101.4 ± 1.5	
Plasmalyte group						
POD1	3.8 ± 2.0	35.4 ± 11.6	7.9 ± 2.8	78 ± 30.3	104.8 ± 3.1	1
POD2	2.1 ± 1.8*	26.6 ± 14.1*	5.4 ± 2.5	98 ± 31.8*	103.4 ± 2.7	
POD3	1.9 ± 1.7*	26.3 ± 15.8*	4.4 ± 1.8	105 ± 27.3*	99.8 ± 2.9	
POD7	1.4 ± 0.9*	21.3 ± 9.7*	1.7 ± 1.4*	114 ± 25.3*	101.7 ± 2.9	

Data are expressed as mean ± standard deviation and (range).

* $P < 0.05$ (intragroup difference).

† $P < 0.05$ (intergroup difference).

conversion of lactate to glucose may impair glucose control in diabetics. Diabetes is a common underlying problem with renal transplant patients and lactated Ringer's should probably be avoided in patients taking metformin in whom lactate metabolism may be impaired.[23]

An ideal crystalloid resuscitation solution would resemble the electrolyte content of plasma. Lactated Ringer's is moderately hypo-osmolar, while 0.9% saline may have substantial adverse effects due to the chloride load. By contrast, Plasmalyte is a balanced salt solution having similar electrolyte constitution to that of plasma and is not associated with the same disturbance of the acid-base status caused by sodium chloride-based fluids.[11,24] This could be explained by the presence of weak acids, such as lactic acid, acetic acid, and gluconic acid. At pH 7.4 these acids are almost entirely in the base anion forms: lactate, acetate, and gluconate. Acetate and gluconate act as bicarbonate precursors, the conversion occurring predominantly in the liver, although acetate may be converted to bicarbonate in other tissues resulting in less acidosis.[25] Furthermore, the lower chloride content of Plasmalyte tends to attenuate the reduction in the strong ion difference compared with saline infusion.[26,27]

A previous randomized, double-blind study was conducted by O'Malley et al.[28] to compare 0.9% normal saline and lactated Ringer's solution during kidney transplantation. In their study, the normal saline group developed hyperkalemia (potassium >6 mM/L) in 19% of patients and metabolic acidosis in 31% of patients compared with 0% in lactated Ringer's group. In our study, potassium level was similar between all groups and no patient in the saline group developed metabolic acidosis. The difference in the two studies is likely due to the significantly shorter duration of surgery and lower total volume of fluids infused in our study. Furthermore, the total volume of the study fluids infused was less in our study, (2.8 ± 1.3 L) in the saline group, (2.7 ± 1.2 L) in lactated Ringer's group versus (6.1 ± 1.3 L), (5.6 ± 1.1 L) in their study respectively.

We concluded that all three crystalloid solutions can be safely used during uncomplicated, short-duration renal transplants; however the best metabolic profile is maintained in patients who receive Plasmalyte.

ACKNOWLEDGMENTS

The authors thank Akdeniz University Scientific Research Project Unit, Antalya, Turkey.

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