Cardiac Arrests Associated with Hyperkalemia During Red Blood Cell Transfusion: A Case Series

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BACKGROUND: Transfusion-associated hyperkalemic cardiac arrest is a serious complication of rapid red blood cell (RBC) administration. We examined the clinical scenarios and outcomes of patients who developed hyperkalemia and cardiac arrest during rapid RBC transfusion.

METHODS: We retrospectively reviewed the Mayo Clinic Anesthesia Database between November 1, 1988, and December 31, 2006, for all patients who developed intraoperative transfusion-associated hyperkalemic cardiac arrest.

RESULTS: We identified 16 patients with transfusion-associated hyperkalemic cardiac arrest, 11 adult and 5 pediatric. The majority of patients underwent three types of surgery: cancer, major vascular, and trauma. The mean serum potassium concentration measured during cardiac arrest was 7.2 ± 1.4 mEq/L (range, 5.9–9.2 mEq/L). The number of RBC units administered before cardiac arrest ranged between 1 (in a 2.7 kg neonate) and 54. Nearly all patients were acidotic, hyperglycemic, hypocalcemic, and hypothermic at the time of arrest. Fourteen (87.5%) patients received RBC via central venous access. Commercial rapid infusion devices (pumps) were used in 8 of 11 (72.7%) of the adult patients, but RBC units were rapidly administered (pressure bags, syringe pumped) in all remaining patients. Mean resuscitation duration was 32 min (range, 2–127 min). The in-hospital survival rate was 12.5%.

CONCLUSION: The pathogenesis of transfusion-associated hyperkalemic cardiac arrest is multifactorial and potassium increase from RBC administration is complicated by low cardiac output, acidosis, hyperglycemia, hypocalcemia, and hypothermia. Large transfusion of banked RBCs and conditions associated with massive hemorrhage should raise awareness of the potential for hyperkalemia and trigger preventative measures.

(Because red blood cell (RBC) membranes are only slightly permeable to potassium, their movement is largely dependent on energy-dependent transport mechanisms (glycolytically derived adenosine triphosphate). During storage, RBC membranes age, adenosine triphosphate synthesis, and potassium pumping decrease, and intracellular potassium leaks into the supernatant. The supernatant of stored RBC units may contain more than 60 mEq/L of potassium. However, hyperkalemia related to transfusion depends not only on the potassium concentration in the RBC unit, but also on volume and rate of RBC administration. A number of reports describing transfusion-related hyperkalemia and cardiac arrest have been published. In elective cases, hyperkalemia from RBC transfusion may be prevented through washing of stored blood units before administration. However, in most instances, rapid transfusion of large volumes takes place during emergencies, which are less amenable to preventative techniques. Therefore, early recognition and prompt pharmacologic intervention may be facilitated by greater awareness and anticipation. It is critical to develop a better understanding of the conditions that lead to hyperkalemia during RBC transfusion. In the present report, we examine the clinical scenarios and outcomes of 16 patients who suffered cardiac arrest after developing hyperkalemia during RBC transfusion.

METHODS

After Mayo Clinic IRB (Rochester, MN) approval, we retrospectively examined the medical records of patients who developed intraoperative cardiac arrest associated with hyperkalemia during RBC transfusion. Cases were identified by electronic search of the Anesthesia Performance Improvement Database from November 1, 1988, to December 31, 2006. This database was introduced on the November 1, 1988, and serves as a repository of all critical incidents, including perioperative cardiac arrests and deaths.
We defined transfusion-associated hyperkalemic cardiac arrest as an intraoperative arrest requiring cardiopulmonary resuscitation during administration of either large volume or fast RBC transfusion. Our criteria for defining transfusion-associated hyperkalemic cardiac arrest included arrest temporally related to large or fast RBC transfusion, laboratory evidence of hyperkalemia (≥5.5 mEq/L), and/or an anesthesiologist’s note that the arrest was due to hyperkalemia. Large or fast RBC transfusion occurred when several RBC units were given in sequence, multiple lines were used to simultaneously transfuse RBC, and/or commercial pressure devices were used to accelerate transfusion. We analyzed only cases that occurred under continuous care of the anesthesia team, i.e., those in the operating room or in the postanesthesia care unit. Using “death” and “cardiac arrest” as keywords, our initial search of the Performance Improvement database identified 1420 patients. This group was further searched with specific key words: external defibrillation, chest compressions, change in cardiac status, arrhythmia requiring intervention, hypothermia, rapid transfusion pump, RBC transfusion, and hyperkalemia. We identified 222 cases, and all charts were reviewed by a single reviewer (S.J.F.). We excluded all events that happened during cardiopulmonary bypass operations, liver transplant operations, and patients who required extracorporeal membrane oxygenation. Physician documentation of the arrests was also scrutinized for statements relating to evidence of electrocardiographic (ECG) patterns consistent with hyperkalemia during RBC transfusion. The charts of 16 patients that fit our search criteria were independently reviewed by the three authors (H.M.S., J.A., S.J.F.), and any inconsistencies were resolved by the consensus of the senior authors (J.S., H.M.S.).

The following information was collected for each patient: age, gender, ASA physical status, type and urgency of surgery (elective versus emergency). Relevant comorbid conditions were also recorded including kidney disease (creatinine ≥ 2.0 mg/dL), coronary artery disease, congestive heart failure, and diabetes mellitus. Laboratory data collected included serum potassium concentrations (before surgery, last value before cardiac arrest, and the highest level during cardiac arrest), ionized calcium levels (before cardiac arrest or calcium administration), hemoglobin concentration, blood glucose concentrations, and pH (last measurements immediately before cardiac arrest). RBC transfusion was calculated as number of RBC units before the cardiac arrest, and total RBC units for the entire surgery. The transfusion of any other non-RBC products (platelets, fresh frozen plasma, cryoprecipitate) are not reported. Additional data included lowest intraoperative body temperature and ECG presentation of the cardiac arrest (ventricular fibrillation, asystole, pulseless electrical activity, wide complex QRS). Resuscitation measures were divided into mechanical (chest compressions, external or internal cardiac massage, defibrillation, pacemaker placement) and pharmacological (atropine, epinephrine, bicarbonate). Data were collected for specific management of recognized or presumed hyperkalemia, including calcium, insulin, bicarbonate, glucose, albuterol, and hyperventilation. We also collected information regarding administered RBC units: leukoreduction, irradiation status, and type of storage solution. Outcomes in terms of operating room survival and in-hospital survival were also recorded. Only descriptive statistics (mean and standard deviation [sd], median and range) were used.

Data were also collected regarding supernatant potassium concentrations of banked packed RBC units as part of routine institutional quality assurance testing. Randomly selected RBC units were tested during the past 5 yr and the age of the unit and potassium concentrations (mEq/L) were recorded. Values reported in Figure 1 are unrelated to the RBC units given to patients who had cardiac arrest in the present case series. Normal potassium range at our institution is 3.6–4.8 mEq/L. Potassium concentrations were determined by the Mayo Clinic Central Laboratory using the Roche/Hitachi Modular Analytic System E170, Roche Diagnostics, Indianapolis, IN.

RESULTS

We identified 16 patients, 11 adult and 5 pediatric, in whom RBC transfusion-associated hyperkalemia was found to be a significant factor in precipitating cardiac arrest. The majority of patients underwent three major types of surgeries: cancer, major vascular, and trauma. All were receiving rapid blood transfusion before cardiac arrest. All transfused units were packed RBCs and none received whole blood. Three patients received leukoreduced RBC units and only one patient (2 day old) received irradiated blood (Table 2). All patients received normal saline during RBC administration, although lactated Ringer’s solution (with potassium content) was given to some patients before the events. Six (37.5%) patients underwent emergency surgery. Table 1 summarizes the demographics, comorbidities, and other intraoperative and laboratory characteristics of these patients.

Mean preoperative serum potassium of 16 patients was 4.2 ± 0.6 mEq/L (mean ± sd) with a range from 3.1 to 5.1 mEq/L (Table 1, Fig. 1). Mean serum potassium measured immediately before cardiac arrest was 5.1 ± 1.0 mEq/L (n = 16 patients), whereas that measured during or immediately after cardiac arrest was 7.2 ± 1.4 mEq/L (range, 5.9–9.2 mEq/L) (these were the highest potassium measurements recorded). In two cases, the laboratory reported the potassium as >8 mEq/L and this value was used in our calculations, although this clearly under-estimates the true serum concentration.

The volume of RBC units administered before cardiac arrest in all patients ranged between 1 (in a 2.7 kg
neonate) and 54. Average hemoglobin at the time of cardiac arrest was 9.6 ± 3.2 g/dL. Nearly all patients were found to be hyperglycemic at the time of the arrest, with mean glucose concentrations of 288 ± 136 mg/dL. Almost all patients were acidic (pH 7.18 ± 0.12), hypocalcemic, and hypothermic at the time of arrest (Table 1). In all patients, cardiac arrest occurred in the operating room. One patient experienced acute postthoracotomy bleeding in the recovery room and was immediately taken back to the operating room where he received 6 U of RBC and suffered fatal hyperkalemic cardiac arrest with a potassium concentration of 9.2 mEq/L. Fourteen (87.5%) patients received RBC via central venous access. Commercial rapid infusion devices were used in 8 of 11 adult patients (72.7%), and in the remainder, RBC was rapidly administered using pressure bags or syringe pumping. Mean resuscitation efforts lasted 32 min with a range from 2 to 127 min. Four patients survived the initial event to be transferred to the intensive care unit, but subsequently died from multiorgan failure (n = 3) or “intractable hypotension unresponsive to treatment” (n = 1). Only two patients survived to hospital discharge (12.5%) (Table 2). Table 2 shows ECG presentation of cardiac arrest, therapeutic measures, outcome, and type of RBC unit transfused.

Analysis of 74 blood bank RBC units (unrelated to the RBC units given to our patients in the present case series) revealed average potassium concentrations of 27.3 ± 13.5 mEq/L. Potassium levels ranged from 7.3 to 77.2 mEq/L (Fig. 2). Within the first week of storage (0–7 days), average measured potassium was 19.0 ± 7.8 mEq/L (n = 34), during the second week (8–14 days) 31.5 ± 14.1 mEq/L (n = 26), and between 15 and 28 days storage time 39.9 ± 10.3 mEq/L (n = 14).

**DISCUSSION**

Hyperkalemia associated with cardiac arrest may be a serious complication of massive blood administration. The main finding of our study is that transfusion-associated hyperkalemic cardiac arrest may develop with rapid RBC administration even with modest transfusion volume (Table 1). Other conditions associated with hemorrhagic shock (acidosis, hyperglycemia) likely contribute to increasing serum potassium levels. Similarly, hypothermia and hypocalcemia independently increase the risk of potassium cardiotoxicity. All this is underscored by the low survival rate in patients who experienced cardiac arrest after hyperkalemia during rapid blood administration (12.5%).

Most published cases of transfusion-associated hyperkalemic cardiac arrest have occurred in pediatric patients, but little is known about how hyperkalemic cardiac arrests related to transfusion partition in total perioperative cardiac arrests across ages. In our previous study, 223 noncardiac surgery patients experienced perioperative cardiac arrests, and 4% (9 of 223) were associated with hyperkalemia during RBC transfusion. In a more recent study of perioperative pediatric cardiac arrests during noncardiac operations, 19.2% (5 of 26) of arrests were associated with hyperkalemia during RBC transfusion. These estimates suggest that this etiology of cardiac arrest is more frequently the cause of arrest in pediatric patients compared with adult patients in whom other causes are more prevalent. Smaller circulating blood volumes, immature renal function and potassium handling, and differences in autonomic tone may account for some of the discrepancy in prevalence of cardiac arrests between pediatric and adult patients after rapid RBC administration.
The pathogenesis of hyperkalemia during massive RBC transfusion is complex, and depends on numerous alterations related to hemorrhage (tissue hypoperfusion), RBC unit factors, and factors related to the rate and route of administration (Table 3). Serious physiologic derangement, such as uncontrollable hemorrhage with associated low cardiac output, may slow intracellular distribution of potassium administered with RBC units. It has been shown that the low cardiac output states per se (such as hemorrhage in our study) can be associated with hyperkalemia, providing that the potassium concentration in the rapidly transfused blood (with either packed RBCs or whole blood reconstituted with plasma) exceeds 10 mEq/L.\(^{19}\) We demonstrated that the mean potassium concentration in our banked RBC units is 27.3 mEq/L (10-day-old blood), well above the 10 mEq/L required to produce hyperkalemia in low cardiac output states (Fig. 2). Elevated serum potassium typically normalizes rapidly when the transfusion rate is slowed\(^{20}\); however, intracellular redistribution of potassium depends on adequate circulating blood volume and cardiac output,\(^{19}\) both of which are low in hemorrhagic shock. In addition, management of hemorrhagic shock requires uninterrupted resuscitation; therefore, potassium increases from transfusion continue until hemodynamic stability and effective homeostasis are achieved.

Other mechanisms contributing to the risks of RBC transfusion-induced hyperkalemia or increased potassium cardiotoxicity include hyperglycemia, hypocalcemia, hypothermia, and acidosis. First, surgical stress and shock are associated with hyperglycemia. This acute increase in serum osmolality causes potassium to exit cells.\(^{12,13}\) Second, massive transfusion of citrated blood is associated with hypocalcemia, which predisposes to cardiac membrane instability at lower potassium levels.\(^{15}\) Hypothermia also slows the metabolism of citrate, which exacerbates hypocalcemic states. Third, in hypothermia, the rat myocardium

### Table 1. Demographic and Other Characteristics of Patients with Red Blood Cell Transfusion-Related Hyperkalemic Cardiac Arrest

<table>
<thead>
<tr>
<th>Year of event</th>
<th>Age, gender, ASA PS</th>
<th>Type of surgery</th>
<th>Comorbid conditions</th>
<th>Serum potassium (mEq/L)(^a)</th>
<th>Last Hb before arrest (g/dL)</th>
<th>RBC units given (n)</th>
<th>Last pH and Ca(^{2+}) (mg/dL) before arrest</th>
<th>Lowest temp (°C)</th>
<th>Glucose during arrest (mg/dL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1991 59/F/3</td>
<td>Urinary diversion</td>
<td>CRI, DM</td>
<td>4.6</td>
<td>5.1</td>
<td>7.2</td>
<td>9.2</td>
<td>20 27</td>
<td>7.07/3.0</td>
<td>33.1 283</td>
</tr>
<tr>
<td>1991 69/M/4E</td>
<td>TAA repair</td>
<td>CRI, CAD, DM</td>
<td>3.6</td>
<td>4.4</td>
<td>6.5</td>
<td>12.7</td>
<td>3 9</td>
<td>7.18/NA</td>
<td>35 476</td>
</tr>
<tr>
<td>1992 57/F/4</td>
<td>Hepatectomy</td>
<td>CAD</td>
<td>3.9</td>
<td>NA</td>
<td>6.0</td>
<td>8.7</td>
<td>54 57</td>
<td>7.19/4.8</td>
<td>33.4 233</td>
</tr>
<tr>
<td>1993 14/M/3</td>
<td>Liver CA</td>
<td>Healthy</td>
<td>4.6</td>
<td>4</td>
<td>7.9</td>
<td>9.8</td>
<td>3 3</td>
<td>7.38/2.88</td>
<td>35.3 176</td>
</tr>
<tr>
<td>1993 67/M/5E</td>
<td>Resection spinal</td>
<td>CRI, CAD, CHF, DM, sepsis</td>
<td>4.9</td>
<td>NA</td>
<td>6.9</td>
<td>9.2</td>
<td>6 6</td>
<td>7.04/4.8</td>
<td>NA 203</td>
</tr>
<tr>
<td>1994 9/F/3</td>
<td>Thoracotomy</td>
<td>CRI, DM</td>
<td>4.1</td>
<td>NA</td>
<td>7.9</td>
<td>6.9</td>
<td>5 5</td>
<td>7.31/4.12</td>
<td>36.5 119</td>
</tr>
<tr>
<td>1994 33/F/4E</td>
<td>Esophageal CA</td>
<td>Healthy</td>
<td>3.7</td>
<td>6.2</td>
<td>7.9</td>
<td>5.8</td>
<td>30 30</td>
<td>7.17/3.6</td>
<td>35 476</td>
</tr>
<tr>
<td>1995 67/M/3E</td>
<td>Thrombectomy</td>
<td>Healthy</td>
<td>4.9</td>
<td>5.5</td>
<td>9.2</td>
<td>8.4</td>
<td>6 6</td>
<td>7.31/NA</td>
<td>34.5 280</td>
</tr>
<tr>
<td>1995 29/F/4E</td>
<td>Trauma</td>
<td>CAD, severe pulmonary HTN</td>
<td>3.2</td>
<td>8.1</td>
<td>8.1</td>
<td>9.6</td>
<td>7 14</td>
<td>6.95/2.2</td>
<td>31.7 210</td>
</tr>
<tr>
<td>1996 69/F/5</td>
<td>AAA repair</td>
<td>CRI, CAD</td>
<td>4.7</td>
<td>3.8</td>
<td>6.0</td>
<td>8.2</td>
<td>4 29</td>
<td>7.41/NA</td>
<td>35 476</td>
</tr>
<tr>
<td>1999 68/M/3E</td>
<td>Osteogenic sarcoma</td>
<td>CAD, CABG, Prior lung CA</td>
<td>3.9</td>
<td>NA</td>
<td>6.1</td>
<td>7.7</td>
<td>13 13</td>
<td>7.19/4.8</td>
<td>35.3 152</td>
</tr>
<tr>
<td>2002 12/M/3</td>
<td>Anterior spine</td>
<td>Severe scoliosis</td>
<td>3.9</td>
<td>4.4</td>
<td>7.1</td>
<td>11.6</td>
<td>6 6</td>
<td>7.16/2.0</td>
<td>36 95</td>
</tr>
<tr>
<td>2003 39/M/3</td>
<td>Hemipelvectomy</td>
<td>Cachectic on</td>
<td>3.9</td>
<td>4.8</td>
<td>6.7</td>
<td>6.3</td>
<td>48 78</td>
<td>7.20/4.0</td>
<td>34.7 289</td>
</tr>
<tr>
<td>2004 61/F/2</td>
<td>Rectal CA</td>
<td>HTN</td>
<td>5.1</td>
<td>3.8</td>
<td>&gt;8.0</td>
<td>8.6</td>
<td>6 9</td>
<td>7.10/4.1</td>
<td>30.4 442</td>
</tr>
<tr>
<td>2005 2d/M/4</td>
<td>Resection pelvic angiosarcoma</td>
<td>Hyperthyroidism</td>
<td>3.6</td>
<td>NA</td>
<td>5.9</td>
<td>11.3</td>
<td>1 1 (350 mL)</td>
<td>7.02/2.2</td>
<td>36.5 420</td>
</tr>
<tr>
<td>2005 17/M/5E</td>
<td>Trauma</td>
<td>Healthy</td>
<td>3.1</td>
<td>6.4</td>
<td>&gt;8</td>
<td>19.3</td>
<td>39 39</td>
<td>7.21/3.3</td>
<td>NA &gt;400</td>
</tr>
</tbody>
</table>

\(F\) = female; \(M\) = male; \(NA\) = not available; \(d\) = days; \(N\) = number; \(ASA\) PS = American Society of Anesthesiologists Physical Status; \(E\) = emergency; \(Hb\) = hemoglobin concentration; \(TAA\) = thoracoabdominal aneurysm; \(AAA\) = abdominal aortic aneurysm; \(HTN\) = hypertension; \(CAD\) = coronary artery disease; \(CABG\) = coronary artery bypass grafting; \(DM\) = diabetes mellitus; \(CHF\) = congestive heart failure; \(CRI\) = chronic renal insufficiency; \(CPR\) = cardiopulmonary resuscitation; \(CA\) = cancer; \(TPN\) = total parenteral nutrition.

\(a\) Normal potassium values are 3.6–4.8 mEq/L.

\(b\) Ionized calcium (normal values, 4.8–5.7 mg/dL).

\(c\) This patient first developed acute hemorrhagic cardiac arrest (severe hypotension), and then hyperkalemic cardiac arrest during fast administration of blood.
becomes more sensitive to the toxic effects of potassium. Finally, almost all our patients had metabolic acidosis before cardiac arrest and this condition significantly contributes to extracellular potassium shift. The majority of our patients were hyperglycemic, hypocalcemic, hypothermic, and acidotic (Table 1).

Release of endogenous sources of potassium may also exacerbate hyperkalemia from blood transfusion. For example, massive tissue injury, rhabdomyolysis after trauma, manipulation of tumor tissues, or protracted ischemia during major vascular surgery, can all increase serum potassium. The majority of our cases fit...
Transfused unit

Mode of blood

Central line

administration

Fast

administration

Large RBC

volume

administration

Pressure infusing
devices

Mechanism

H+ /K+ exchange leads to shift of potassium extracellularly
Alteration of transmembrane potential
Myocardium appears to be more sensitive to hyperkalemia
Osmotic shift of potassium out of cells
Impairs extrarenal disposal of an acute potassium load
Low cardiac output alters potassium distribution
Low volume of distribution

Load of potassium (undiluted) directly delivered to the heart
Shortened time allowed for the potassium equilibration between body compartments
Large potassium load
Red blood cell membrane rupture from shear stress

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Shortened time allowed for the potassium equilibration between body compartments
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Load of potassium (undiluted) directly delivered to the heart
Shortened time allowed for the potassium equilibration between body compartments
Large potassium load
Red blood cell membrane rupture from shear stress

Table 3. Factors Which May Contribute to Hyperkalemia During Blood Transfusion

<table>
<thead>
<tr>
<th>Patient factors</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidosis11</td>
<td>H+ /K+ exchange leads to shift of potassium extracellularly</td>
</tr>
<tr>
<td>Hypocalcemia15,34</td>
<td>Alteration of transmembrane potential</td>
</tr>
<tr>
<td>Hypothermia14</td>
<td>Myocardium appears to be more sensitive to hyperkalemia</td>
</tr>
<tr>
<td>Hyperglycemia12,13</td>
<td>Osmotic shift of potassium out of cells</td>
</tr>
<tr>
<td>Beta-adrenergic blockade35</td>
<td>Impairs extrarenal disposal of an acute potassium load</td>
</tr>
<tr>
<td>Shock/low cardiac output state19,36</td>
<td>Low cardiac output alters potassium distribution</td>
</tr>
<tr>
<td>Low weight (pediatric)37,38</td>
<td>Low volume of distribution</td>
</tr>
<tr>
<td>Transfused unit factors</td>
<td>Erythrocyte membrane rupture and potassium leakage</td>
</tr>
<tr>
<td>Hemolysis39–41</td>
<td>Increased potassium load from transfused supernatant</td>
</tr>
<tr>
<td>Old blood42</td>
<td>Gamma irradiation-induced damaged of erythrocyte membrane</td>
</tr>
<tr>
<td>Irradiated blood43,44</td>
<td></td>
</tr>
</tbody>
</table>

into one of these categories: 8 patients (50%) had tumor surgery, 3 (18.8%) had massive motor vehicle trauma, and 3 (18.8%) had major vascular operations. The route and rate of blood administration are important factors that must be considered in the pathogenesis of transfusion-associated hyperkalemic cardiac arrest. Most of the patients in our report received RBCs via central venous access and using high-pressure infusing devices. Any exogenous source of potassium would be carried directly to the right heart through the pulmonary circulation and to the left heart where coronary circulation occurs. Therefore, central venous access may deliver more concentrated potassium loads to the coronary circulation than peripheral venous access, and this could have contributed to cardiac arrest in some of our patients. At the same time, pressure-infusing devices can traumatize RBCs causing additional leak of potassium.24,25 Finally, Linko and Tigerstedt20 demonstrated that hyperkalemia correlated well with the rate of transfusion, and not with the actual amount of blood transfused.

An associated clinical problem is that during rapid RBC administration, laboratory evidence of serum potassium concentrations lags in time with resuscitation efforts. Except for ECG monitoring, providers may have no evidence of increasing potassium levels until cardiac arrest ensues. Increases in serum potassium levels may or may not produce accompanying electrocardiographic changes. Patients can manifest peaked T waves, bradycardia, or changing QRS morphology, at serum potassium levels as low as 5.3 mEq/L, whereas patients with chronic renal failure may show no ECG changes, even at much higher potassium levels.26 Brown et al.19 reported that 6 of 18 cardiac arrests were associated with the plasma potassium levels of 6.0 mEq/L or higher. The potassium values in our patients (7.2 mEq/L) were within a range consistent with cardiac arrest reported by others.19,27–29

The quantity of RBC units associated with hyperkalemic cardiac arrest in our patients varied widely (Table 1). The most plausible explanation is that those who received less blood and experienced hyperkalemia either received RBC units with higher potassium content or had increased endogenous potassium release. Anesthesiologists rarely know the potassium content in administered RBC units and may be unaware of the wide variation of potassium in stored RBC units (Fig. 2). Potassium increases with time in stored RBC units. Our highest quality assurance RBC unit tested contained supernatant potassium of 77.0 mEq/L at 14 days of storage, only one-third through its usable shelf life (42 days). One published case of cardiac arrest during blood transfusion reported a potassium concentration of 120 mEq/L in the blood unit tested after the arrest.30 Also, irradiation of blood, by disrupting the RBC membrane, increases the RBC unit free potassium.31 One of our patients, a 2-day-old newborn, received irradiated blood.

Because of the potential to develop transfusion-associated hyperkalemic cardiac arrest during large RBC transfusion, determination of potassium in both RBC units and patients’ blood should be routinely considered. Point-of-care laboratory testing may allow for expedited reporting of potassium concentrations to providers. Other measures aimed at preventing the transfusion of hyperkalemic blood products include the preoperative washing of RBC units by transfusion medicine or intraoperative washing of RBC units using cell salvage equipment. A future option may be the use of in-line potassium absorption filters; however, their use has been rarely reported,32,33 because they are not yet commercially available world-wide. In addition, because of flow limitations of these filters, they may be less suitable for situations where fast RBC transfusion is required.

LIMITATIONS OF THE STUDY

Our study has one important limitation in addition to those associated with any retrospective study. Because neither the shelf-age nor the RBC unit potassium content are known before transfusion, we cannot
calculate the potassium load given to the patient for each transfused unit. Therefore, we cannot precisely determine the role of hyperkalemia from transfusion versus that of other associated conditions in the pathogenesis of cardiac arrest in our patients.

In conclusion, when dealing with patients undergoing massive hemorrhage and blood replacement, it is imperative to anticipate the potential for developing hyperkalemia. In addition to potassium load in transfused RBC units, other factors associated with bleeding, such as hypotension with low cardiac output, hypothermia, hypocalcemia, hyperglycemia, and acidosis, can contribute to hyperkalemia and/or increase the potential for potassium cardiotoxicity. Anticipation and early recognition of these aberrations, and their prompt correction may theoretically improve otherwise low survival from transfusion-associated hyperkalemic cardiac arrest.

REFERENCES

44. Weiskopf RB, Schnapp S, Rouine-Rapp K, Bostrom A, Toy P. Extracellular potassium concentrations in red blood cell suspensions after irradiation and washing. Transfusion 2005;45:1295–301