Safety and Efficacy of Regional Citrate Anticoagulation During 8-Hour Sustained Low-Efficiency Dialysis

John A. Clark, Gerald Schulman, and Thomas A. Golper
Division of Nephrology and Hypertension, Department of Medicine, Vanderbilt University, Nashville, Tennessee

Background and objectives: Patients who may benefit from sustained low-efficiency dialysis therapy are often at risk for bleeding. A safe and simple “regional” anticoagulation strategy would be beneficial. The modification of existing regional citrate anticoagulation protocols to typically performed 8-h sustained low-efficiency dialysis is necessary.

Design, setting, participants, & measurements: Sustained low-efficiency dialysis was performed at blood and dialysate rates of 250 and 300 ml/min, respectively. The circuit was anticoagulated with 4% sodium citrate (citrate 136, sodium 408 mmol/L) and reversed with CaCl2. Every 2 h, electrolytes, ionized circuit, and patient calcium were monitored during the first two versions. The second version differed by an increased infusion of CaCl2 and lower infusion of citrate, both by 10%. The third version measured only laboratory values before and after sustained low-efficiency dialysis.

Results: There were 41 treatments in the first iteration, 42 in the second, and 34 in the final iteration. All versions were titrated to maintain patient ionized calcium of 4.0 to 4.8 mg/dl (1.0 to 1.2 mmol/L) and the circuit ionized calcium between 0.8 and 1.6 mg/dl (0.2 and 0.4 mmol/L). The final protocol infusion was 31 mmol/h citrate and 41 mmol/h elemental calcium, which kept circuit and patient ionized calcium at targets. No unexpected metabolic complications occurred.

Conclusions: Compared with continuous renal replacement therapy, one must increase the calcium infusion because of the more efficient removal of the calcium citrate complex. Safe and effective regional citrate anticoagulation can be performed in 8-h sustained low-efficiency dialysis without metabolic complications with laboratory surveillance only before and after sustained low-efficiency dialysis treatment; however, certain safeguards are mandatory.


Since regional citrate anticoagulation (RCA) for hemodialysis (HD) was first introduced in 1961, it has been modified throughout the world to apply to many dialysis modalities (1–4). It is an ideal alternative to heparin in patients who are at increased risk for bleeding (5,6). The technical application and concentration of citrate does vary (7,8). The pharmacokinetics of citrate anticoagulation are well understood as citrate chelates free calcium in the blood, essentially removing Ca2+ from its role as a co-factor in the coagulation cascade. To date, the main application of citrate has been to continuous renal replacement therapy (CRRT), including continuous sustained low-efficiency dialysis (SLED), but it has not been shown to have a role or application in intermittent hybrid therapies such as 8-h SLED, as performed in the United States.

The major metabolic complications of RCA are due to excess retained sodium and citrate ions (hypernatremia and metabolic alkalosis) (9,10). Trisodium citrate (TSC) is metabolized to bicarbonate in skeletal muscle and liver and, when metabolism is complete, results in the generation of three molecules of bicarbonate from one citrate molecule. Because TSC contains on a molar basis three times as many sodium ions as citrate ions, the sodium load can increase the serum sodium concentration. Thus, hypernatremia has been observed in our experience, as well as others using RCA during continuous venovenous HD. The correction often requires the addition of hemodiafiltration with 0.45% NaCl replacement fluid to remove excess sodium and bicarbonate (HCO3-) (11). Other metabolic complications and adverse effects of RCA have been due to the difficulty of correcting the decalcification and impaired citrate metabolism that accompanies liver failure (12–14). Thus, RCA is not without potential problems. Developing a protocol that is safe and effective is in itself time-consuming and will not be undertaken unless there is a significant demand for SLED for patients who are at high risk for bleeding. Thus, we believe that describing the protocol that we developed for this population will have broad utility. Furthermore, the development of the protocol itself is instructive.

Finkel and Foringer (15) described the use of RCA during continuous SLED (C-SLED), demonstrating that in >2200 h of therapy, none of the 20 patients had significant derangements in serum sodium concentration or acid base status to require cessation of RCA during this therapy. Their explanation for the lack of hypernatremia and metabolic alkalosis was that the higher dialysis dosage delivered by C-SLED likely corrected the problems as they developed and the problems never became clinically evident. Finkel and Foringer used different blood flow (Qb) and dialysate flow (Qd) than are used in our 8-h SLED, so the RCA regimen was only theoretically applicable. Marshall et al. (16) simulated RCA during SLED using a crys-
talloid solution instead of blood. Again, this is only of theoretical benefit as applied to 8-h SLED. RCA was also applied to a hybrid therapy similar to 8-h SLED using the Genius system (17); however, the Genius system is not available worldwide, and 8-h SLED with the operating conditions that we apply is available almost everywhere.

Thus, because of the unique properties of 8-h SLED therapy and its widespread use, we proposed an RCA protocol to fit this expanding modality. We used standard commercially available solutions and implemented a step-wise evolution of titration and safety monitoring of RCA adverse effects. We now are convinced that RCA can be applied to our 8-h SLED therapies in a safe and efficacious manner without the measurement of intradialytic ionized calcium, sodium, or bicarbonate concentrations.

**Materials and Methods**

**Study Protocols and Design**

We used Fresenius 2008H (retrofitted to allow a Qd rate as low as 100 ml/min) or K machine for 8-h SLED treatments in our intensive care units or the dialysis unit at Vanderbilt University Hospital. We preferred the K machine for its touchscreen interface and the added feature of the Diasafe, which filters dialysate water just before the dialysate enters the hemodialyzer. The flow rates were rigidly set at Qb of 250 ml/min and Qd of 300 ml/min, which differ considerably from that of Finkel and Foringer when they performed C-SLED (15). The Qb of 250 ml/min was selected because of the theoretical antithrombotic benefit of higher Qb causing less stasis within the blood pathway. The “low efficiency” term is descriptive because both Qb and Qd are much lower than in standard intermittent HD. When the mandated Qb or Qd were not achieved, the RCA protocol was not applied. This is because the dosages of citrate and calcium depend on Qb and dialytic clearance. During the treatment, switching among heparin, saline flushes, and RCA was not allowed. The standard dialysate [Na⁺] was 140 mEq/L, and did not vary. Dialysis machine set-up, hemodynamic and technical monitoring, and laboratory surveillance were exclusively performed by dialysis staff.

TSC (4% TSC solution manufactured by Baxter [Deerfield, IL], citrate 136 mmol/L, and sodium 408 mmol/L) was infused by a volumetric pump into a three-way stopcock at the blood pick-up site, and CaCl₂ (pharmacy formulated to make the 39-g/L CaCl₂ final solution [1 mmol elemental Ca²⁺/ml] from a standard stock 10% calcium chloride concentrate) was infused in the same manner into a three-way stopcock at the blood return site. The dialysis machines generate dialysate from concentrate, and there is flexibility in the concentration of many constituents. The calcium-free dialysate contained 33 mEq/L bicarbonate. Net ultrafiltration rates were determined by individual patient needs.

**Table 1. Standard titration protocols for RCA with CRRT**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>If patient iCa²⁺ &lt;0.8 mg/dl (&lt;0.2 mmol/L), then decrease citrate rate by 5 ml/h.</td>
<td></td>
</tr>
<tr>
<td>If patient iCa²⁺ 0.8 to 1.6 mg/dl (0.2 to 0.4 mmol/L), then do not change citrate rate.</td>
<td></td>
</tr>
<tr>
<td>If patient iCa²⁺ 1.6 to 2.0 mg/dl (0.4 to 0.5 mmol/L), then increase citrate rate by 5 ml/h.</td>
<td></td>
</tr>
<tr>
<td>If patient iCa²⁺ &gt;2.0 mg/dl (&gt;0.5 mmol/L), then increase citrate rate by 10 ml/h.</td>
<td></td>
</tr>
</tbody>
</table>

The patient sample is drawn from a peripheral site or before citrate infusion site every 2 h (0, 2, 4, 6, and 8) or before and after as in the final protocol.

The patient’s serum HCO₃⁻ rises >10 mEq/L.

Sample sizes were not predetermined because this was a clinical protocol development strategy. Thus, after acceptable safety parameters were met during the first version of the protocol, the second version protocol was initiated using the mean infusion rates for TSC and CaCl₂, as that seen for the mean of the eighth-hour infusion rate from protocol version 1. After consistent safety parameters were met in the second protocol version without significant change in infusions, the final protocol was started using the same approach as the first set of changes. The final protocol was designed to have no change in infusion rate during the course of the SLED treatment unless initial laboratory values were very unusual and further stipulated that the only laboratory surveillance was pre- and post-SLED treatment.
Surveillance and Safety Monitoring

Surveillance of circuit and patient laboratory values was managed by the dialysis nurse and the physician. Standard titration protocols from RCA with CRRT were applied to the SLED monitoring (Table 1).

Patients

All patients presented with one or more indications to avoid systemic anticoagulation, such as recent surgical procedure, thrombocytopenia, or coagulopathy. We implemented a modified standard RCA protocol from our CRRT protocols (derived from that of Swartz et al. [18] from the University of Michigan Medical Center) into our SLED practice, because the operational characteristics for SLED were more similar to CRRT than they were for intermittent HD, which had a different RCA protocol. We calculated the mmol/h citrate infused during CRRT with a Qb rate of 180 ml/min. Our SLED operational Qb rate was 250 ml/min, so we increased the citrate infusion accordingly. The CRRT protocol uses anticoagulant citrate dextrose-formulation A (ACD-A) as the citrate source. We had to convert the source to 4% TSC because the protocol uses anticoagulant citrate dextrose-formulation A (ACD-A) as the citrate source. We used the same math reasoning for CaCl2 and eventually had to make a more concentrated solution there as well. Typically, the patients who required SLED therapy were in the intensive care unit, but an occasional treatment was performed in the dialysis unit. Only some patients were on mechanical ventilation, and management of the ventilators was often performed without obtaining arterial blood gases. The choice of 8-h SLED over intermittent HD or CRRT was determined by clinical, technical, or staffing criteria. Baseline characteristics of admitting diagnosis, age, race, and gender are shown in Table 2. Informed consent for the dialysis included a discussion of the anticoagulation regimen, which included the reasons to avoid systemic anticoagulation.

Statistical Analyses

Unpaired t test was used to assess the changes between time points within a protocol version and against other versions. A significant difference was defined as P < 0.05. All results are expressed as the means ± SD. Group sizes were not chosen/selected in advance for any statistical purposes because this was not a prospective study. When we felt comfortable that we understood the results of the protocols, we moved to the next protocol, each protocol with less laboratory surveillance.

Table 2. Baseline demographics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>First Protocol</th>
<th>Second Protocol</th>
<th>Third Protocol</th>
<th>P</th>
</tr>
</thead>
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<tr>
<td>Patients</td>
<td>9</td>
<td>11</td>
<td>10</td>
<td>NS</td>
</tr>
<tr>
<td>Treatments</td>
<td>41</td>
<td>42</td>
<td>34</td>
<td>NS</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>53.6 ± 4.8</td>
<td>53.5 ± 5.0</td>
<td>50 ± 5.0</td>
<td>0.04</td>
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<tr>
<td>Male gender</td>
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<td>7</td>
<td>4</td>
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<tr>
<td>Race</td>
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<td></td>
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<tr>
<td>white</td>
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<td>black</td>
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<td>3</td>
<td>2</td>
<td>NS</td>
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<td>6</td>
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<td>NS</td>
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<tr>
<td>chronic</td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>NS</td>
</tr>
<tr>
<td>Baseline laboratory values</td>
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<tr>
<td>Sodium (mEq/L)</td>
<td>138.8 ± 0.8</td>
<td>138.8 ± 0.9</td>
<td>139 ± 0.7</td>
<td>0.54</td>
</tr>
<tr>
<td>HCO3⁻ (mmol/L)</td>
<td>25.5 ± 0.6</td>
<td>25.9 ± 0.6</td>
<td>26.2 ± 0.9</td>
<td>0.48</td>
</tr>
<tr>
<td>Venous pH</td>
<td>7.33 ± 0.01</td>
<td>7.33 ± 0.01</td>
<td>7.33 ± 0.01</td>
<td>0.95</td>
</tr>
<tr>
<td>Ionized calcium (mg/dl)</td>
<td>4.6 ± 0.09</td>
<td>4.65 ± 0.09</td>
<td>4.33 ± 0.07</td>
<td>0.012</td>
</tr>
<tr>
<td>Total calcium (mg/dl)</td>
<td>9.1 ± 0.2</td>
<td>9.1 ± 0.2</td>
<td>8.0 ± 0.2</td>
<td>0.002</td>
</tr>
</tbody>
</table>

*aP values were compared from first protocol to final version. P < 0.05 is significant.
versions of the RCA protocol, as shown in Table 5. Concentrations did drop during the SLED treatment in all three

Evolution from First Protocol Version to Final Protocol Version

Table 4. Ionized circuit calciuma

<table>
<thead>
<tr>
<th>Parameter</th>
<th>First Protocol</th>
<th>Second Protocol</th>
<th>Final Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2 H</td>
<td>8 H</td>
<td>P</td>
</tr>
<tr>
<td>Ionized calcium (mg/dl)</td>
<td>0.81</td>
<td>0.84</td>
<td>0.07</td>
</tr>
</tbody>
</table>

a8 h P1 = first/final, 8 h P2 = first/second, 8 h P3 = second/final. To convert Ca concentration in mg/dl to mmol/L, divide by 4.

a8P values were compared from 0 to 8 h.

Efficacy of Citrate Anticoagulation

No dialyzers clotted, and no treatments were terminated as a result of dialyzer clotting. Also of significance, no bleeding was recognized. During the titration of each version of the RCA protocol, the circuit ionized calcium levels were maintained in an acceptable range (Table 4). The first and second protocol mean ionized calcium concentration was unchanged from the second to the eighth hour. The final protocol ionized calcium concentration was 0.99 mg/dl at 8 h, and, by protocol, no sample was obtained at 2 h. There was no statistical difference in circuit ionized calcium concentration from the first version to the final version (P = 0.23). The patient’s ionized calcium concentrations did drop during the SLED treatment in all three versions of the RCA protocol, as shown in Table 5.

Dosage of Citrate Anticoagulation

The infusion dosages of 4% TSC (Figure 1) and CaCl2 (Figure 2) were able to maintain therapeutic circuit and patient’s ionized calcium concentrations.

The mean initial CaCl2 infusion rate was 37.5 mmol/h elemental Ca2+ but was statistically elevated at 8 h to 43.3 mmol/h elemental Ca2+. We explain this dosing in both ml/h and mmol/h elemental Ca2+ so that the reader can prepare the concentration of CaCl2 to address the local facility’s desires.

The second protocol version had very similar decrease in TSC infusion rate with subsequent increase in CaCl2 infusion rate. The initial TSC infusion rate was 241 ml/h (32.8 mmol/h citrate), which statistically decreased to 231 ml/h (31 mmol/h citrate) by 8 h (P = 0.02). Mean CaCl2 infusion rates also expectedly increased as well from 38.5 mmol/h elemental Ca2+ to 43.4 mmol/h elemental Ca2+ (P = 0.006) at 8 h. The final protocol version infusion rates were set for TSC at 231 ml/h (31 mmol/h citrate) and for CaCl2 (41 mmol/h elemental Ca2+). There were no significant changes in requirements of infusion of TSC or CaCl2.

Complications

We encountered several technical and staffing issues during the evolution of the protocol. There was some initial confusion regarding which citrate solution to use (4% TSC versus ACD-A) because ACD-A is commonly used as the anticoagulant during apheresis and CRRT. We also initially struggled with supplying a large enough volume of TSC to avoid having to change the initial set-up of solutions during treatment. Using 500-ml bags of TSC is preferred. The CaCl2 concentration had to be increased from its original 20-mg/dl solution because it required such large volumes, which complicated the net ultrafiltration goals. Nursing staff had to remember to increase the hourly fluid removal to meet this extra intravenous fluid intake. Another serious problem was in order entry and set-up of the calcium-free dialysate baths. Three SLED treatments during the final protocol were initiated with a 2.5-mEq/L calcium bath instead of a calcium-free dialysate bath. This caused hypercal-

Table 3. Safety

<table>
<thead>
<tr>
<th>Parameter</th>
<th>First Protocol</th>
<th>Second Protocol</th>
<th>Final Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 H</td>
<td>8 H</td>
<td>P</td>
</tr>
<tr>
<td>Sodium (mEq/L)</td>
<td>138.80</td>
<td>139.80</td>
<td>0.420</td>
</tr>
<tr>
<td>HCO3 (mEq/L)</td>
<td>25.50</td>
<td>29.40</td>
<td>0.002</td>
</tr>
<tr>
<td>Venous pH</td>
<td>7.33</td>
<td>7.40</td>
<td>0.009</td>
</tr>
<tr>
<td>Calcium (mg/dl)</td>
<td>9.12</td>
<td>8.62</td>
<td>0.250</td>
</tr>
<tr>
<td>HCO3 (mEq/L)</td>
<td>8.16</td>
<td>8.40</td>
<td>0.002</td>
</tr>
<tr>
<td>Venous pH</td>
<td>7.17</td>
<td>7.52</td>
<td>0.002</td>
</tr>
<tr>
<td>Calcium (mg/dl)</td>
<td>9.12</td>
<td>8.62</td>
<td>0.250</td>
</tr>
<tr>
<td>Ionized calcium (mg/dl)</td>
<td>0.81</td>
<td>0.84</td>
<td>0.07</td>
</tr>
</tbody>
</table>

*P values were compared from 0 to 8 h.
cemia that was not recognized until therapy ended because the only surveillance was before and after SLED. Each of the three affected patients required an extra intermittent dialysis treatment to correct the ensuing hypercalcemia. Another technical problem was on set-up of the infusion ports on one patient during the first protocol. The TSC and CaCl$_2$ infusions were reversed, thus causing systemic infused citrate and lowering the patient’s ionized calcium levels. This was rapidly corrected as a result of the second-hour surveillance laboratory values.

The accuracy of the infusion pumps for TSC and calcium was not assessed. At the end of the SLED treatment, the volume of infused solutions was consistent with the volumetric infusion pump settings and duration of treatment. This became so reliable that we could account for it in our ultrafiltration strategy and orders written before the treatment.

**Discussion**

We have not attempted very many 8-h SLED treatments with similar operating characteristics in the absence of some anticoagulant (e.g., heparin, argatroban); therefore, we can only state that we have developed an RCA protocol to decalcify effectively the extracorporeal circuit. The use of RCA in our SLED system is therefore probably effective at providing anticoagulation of the dialysis circuit while maintaining systemic calcium levels and thus not altering systemic coagulation. We did not experience any circuit failures during the evolution of the citrate protocol. We did not observe hypernatremia, metabolic alkalosis, or excess citrate during our development of an RCA protocol for 8-h SLED treatments. Other errors were made, and the reader should learn from our experience and consciously establish safeguards to prevent this from recurring.

RCA is applied only during the actual RRT. For CRRT and even C-SLED, because they are continuous and lower in per-minute clearance than SLED, the metabolic complications of RCA can gradually accumulate. In SLED, in which the per-minute clearance is at least twice that of CRRT, the SLED treatment actually corrects the potential metabolic complications of RCA as they occur; for example, not allowing an increase in bicarbonate or sodium concentration. Most CRRT modalities use a Qd of 32 ml/min, resulting in a total effluent volume of 48 L/d. In C-SLED, that rate is 100 ml/min, with a daily effluent of 144 L. Qd is increased to 300 ml/min with an 8- to 12-h SLED therapy. The increased removal of the calcium

### Table 5. Ionized patient calcium

<table>
<thead>
<tr>
<th>Parameter</th>
<th>First Protocol</th>
<th>Second Protocol</th>
<th>Final Protocol</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 H 8 H P</td>
<td>0 H 8 H P</td>
<td>0 H 8 H P</td>
</tr>
<tr>
<td>Ionized calcium (mg/dl)</td>
<td>4.63 4.21 0.009</td>
<td>4.66 4.21 0.007</td>
<td>4.33 3.91 0.002</td>
</tr>
</tbody>
</table>

*8 h first/final P = 0.07, 8 h first/second P = 0.80, 8 h second/final P = 0.12. To convert Ca concentration in mg/dl to mmol/L, divide by 4.

**Figure 1.** 4% Trisodium citrate infusion rates during all three protocol versions. Data are means ± SD. (A and B) The first two versions have rates every 2 h. The difference between the 0- and 8-h infusion rates in both A and B is statistically significant ($P = 0.02$). (C) The final protocol version had pre- and final infusion rates that were not statistically different.

**Figure 2.** Ca$^{2+}$ infusion rates during all three protocol versions. Data are means ± SD. (A and B) The first two versions have rates every 2 h. The difference between the 0- and 8-h infusion rates in both A and B is statistically significant ($P = 0.001$ and $P = 0.006$). (C) The final protocol version had pre- and final infusion rates that were not statistically different. There were no significant differences between the 8-h infusion rates in the first protocol and the final protocol.
Table 6. Infusion protocols for each successive version

First version protocol: 41 treatments and nine patients over 1 mo
4% TSC infusion prefilter initially started at 261 ml/h
136 mmol/L citrate × 261 ml/h = 35.5 mmol/h citrate
10.4 g/h = 35.5 mmol/h citrate = 106.5 mEq/h citrate
CaCl₂ infusion postfilter initially started at approximately 31 ml/h
1 mmol/ml elemental Ca²⁺ × 31 ml/h = 31 mmol/h or 62 mEq/h
39 mg/ml CaCl₂ × 31 ml/h = 1.2 g/h or 10.9 mmol/h CaCl₂

Second version protocol: 42 treatments and 11 patients over 2 mo
4% TSC infusion prefilter initially started at approximately 241 ml/h
136 mmol/L citrate × 241 ml/h = 32.8 mmol/h citrate
9.64 g/h = 32.8 mmol/h citrate = 98.3 mEq/h citrate
CaCl₂ infusion postfilter initially started at approximately 36 ml/h
1 mmol/ml elemental Ca²⁺ × 36 ml/h = 36 mmol/h or 72 mEq/h
39 mg/ml CaCl₂ × 36 ml/h = 1.4 g/h or 12.7 mmol/h CaCl₂

Third version protocol: 34 treatments and 10 patients over 1.5 mo
4% TSC infusion prefilter at exactly 231 ml/h
136 mmol/L citrate × 231 ml/h = 31 mmol/h citrate
9.24 g/h = 31 mmol/h citrate = 93 mEq/h citrate
CaCl₂ infusion postfilter at exactly 41 ml/h
1 mmol/ml elemental Ca²⁺ × 41 ml/h = 41 mmol/h or 82 mEq/h
39 mg/ml CaCl₂ × 41 ml/h = 1.6 g/h or 14.5 mmol/h CaCl₂

*Units are expressed in mEq or mmol. For trisodium citrate (TSC), there are 3 mmol of Na or each mmol of citrate. Only citrate is quantified. For Ca, the mmol and mg are expressed for CaCl₂ as well as for elemental Ca²⁺.

Citrate chelated complex in SLED was obvious from our first protocol version, which used infusion rates similar to our CRRT therapy with RCA. These rates were clearly inadequate because of the higher dialytic clearance during SLED. Even with a dialysate bicarbonate concentration of 33 mEq/L, we did not observe metabolic alkalosis during SLED with RCA. We also had to increase the rate of CaCl₂ infusion during SLED for the same reason.

The patients in the final protocol cohort had a higher incidence of predialysis hypocalcemia than either of the first two protocol cohorts. Thus, during the final protocol version, we started off with lower mean ionized and total calcium levels. This is important because our protocol results in lower ionized and total Ca levels after SLED with RCA. We believe that if the pre-SLED total Ca concentration (corrected for the serum albumin concentration) is <8 mg/dl, then surveillance during the SLED with RCA at 2 or 4 h would be prudent. If surveillance is not performed in patients with pre-SLED hypocalcemia, then the CaCl₂ infusion rate should be empirically increased. This is certainly a topic for further investigation. The ideal calcium level in the critically ill patient is not well understood (19–22).

The usual metabolic safety concerns (metabolic alkalosis and/or hypernatremia) are self-corrected with SLED at our dosage of dialysis, although the lowering of total and ionized calcium levels in some patients can become problematic. In those cases, we recommend frequent surveillance and continued adjustment of the infusions of TSC and CaCl₂. We think that a safety check-off list must be developed similar to the method used by airline pilots before take-off. Critical to this would be a second check that the TSC line is going into the blood pick-up line, the CaCl₂ line goes into the blood return line or delivered centrally, and the dialysate is calcium-free. We recently placed the citrate infusion into the arterial drip chamber bubble trap and the calcium infusion into the venous drip chamber bubble trap with general success. This has the obvious benefit of avoiding direct patient infusion of citrate, which could occur with catheter dysfunction. In addition, when the dialysis machine blood pump stops and the drip chambers fill, it will set off alarms on the RCA infusion pumps and they also will stop. If this safety redundancy is applied, then our RCA protocol can provide safe and effective extracorporeal circuit decalcification (and probably anticoagulation) with no routine intradialytic surveillance. Alternatively, surveillance laboratory values can be checked. This, of course, increases costs and uses nursing time. For inexperienced programs, this may be a prudent expense.

Conclusions

The area of RCA in SLED is a new and novel approach to anticoagulating the dialysis circuit. We believe that this therapy can be further modified into other SLED practices with further adjustment to meet specific dialysis needs of an individual patient.

Acknowledgments

Parts of the results were presented at the 12th Meeting of the International Conference on Continuous Renal Replacement Therapies (March 7–10, 2007; San Diego, CA) and the National Kidney Foundation Spring Meeting (April 10–14, 2007; Orlando, FL).

We thank our nursing staff, colleagues, and patients without whom we could not have developed the protocol described here. Judy Moss provided secretarial help.

Disclosures

T.A.G. has consulted for Fresenius-North America.

References

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