Sickle Hb polymerization in RBC components from donors with sickle cell trait prevents effective WBC reduction by filtration

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BACKGROUND: RBC components collected from donors with sickle cell trait frequently occlude WBC-reduction filters. In vitro, sickle trait RBCs have the potential for sickle Hb (Hb S) polymerization at low oxygen saturations and high Hb concentrations.

STUDY DESIGN AND METHOD: To determine if the low pH and high osmolarity of the CP2D used in the collection contributed to filter failures, the filterability of sickle trait donor RBCs collected in CP2D was compared with RBCs from the same donors collected in heparin.

RESULTS: Five of six sickle trait components collected in CP2D did not complete filtration, but all six RBC components collected in heparin filtered completely. RBC components collected in CP2D from four other sickle trait donors were divided in two, and one-half was treated with carbon monoxide to convert Hb S to its liganded form to prevent Hb S polymerization. All four carbon monoxide-treated components filtered within 9 minutes, but only one untreated component filtered completely. RBC components collected by apheresis contained less CP2D, and five of seven sickle trait apheresis components filtered completely; four of the five filtered rapidly (<15 min) and one filtered in 100 minutes. Hb oxygen saturation was greater in the four rapidly filtering apheresis RBC components (68 ± 9%) than in the three that filtered slowly or incompletely (37 ± 5%, p = 0.03).

CONCLUSIONS: Hb S polymerization appears responsible for RBC WBC-reduction filter failures. Citrate anticoagulant and low oxygen saturation are responsible in part for Hb S polymerization in this setting.

The reduction of WBCs in cellular blood components may benefit transfusion recipients by preventing alloimmunization, febrile reactions, cytomegalovirus infections, and possibly transfusion-associated immune suppression.1,2 As a result, many blood centers and transfusion services have begun to provide only WBC-reduced RBC and platelet components.

WBCs are removed from RBC components by filters specially designed for this purpose.3 Although these filters are highly effective, approximately 1 percent of filtered RBC units do not meet the FDA criteria for WBC reduction because the quantity of WBCs remaining in the unit or the loss of RBCs is too high.4 Several recent preliminary reports indicate that RBC components that do not meet the criteria for WBC reduction are more likely to be derived from people with sickle cell trait (Hb AS).5-8 One study found that approximately half of the RBC components collected from people with sickle trait occlude WBC-reduction filters, one-quarter pass completely through the filter but the quantity of WBCs remaining exceeds criteria for WBC reduction, and one-quarter are successfully WBC reduced.7

People with sickle cell disease (Hb SS) are homozy-

ABBREVIATIONS: Hb AA = without Hb S; Hb AS = with sickle cell trait; Hb S = sickle Hb; Hb SS = with sickle cell disease; HPLC = high-performance liquid chromatography.

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gous for Hb S and experience chronic anemia, acute chest syndrome, stroke, pain crises, splenic dysfunction, and renal dysfunction due to the intracellular polymerization of Hb S and resulting pathophysiologic processes.\textsuperscript{9-11} In contrast, people with Hb AS are heterozygous for Hb S and have approximately 40 to 60 percent Hb A in each RBC. They experience no sickle cell crises and few other manifestations of sickle cell disease.\textsuperscript{9-11} Hb S in RBCs from people with Hb AS can polymerize and cause cell sickling at low oxygen tension, very low pH, and/or markedly increased cellular Hb concentration, but under physiologic conditions the concentration of Hb S in RBCs from people with Hb AS is not great enough to cause polymerization.\textsuperscript{12-15} One exception is in the hyperosmotic kidney and renal concentrating defect in sickle trait that is dependent on the percentage of Hb S.\textsuperscript{16}

Blood collected by phlebotomy flows into bags containing 63 mL of citrate anticoagulant preservative, which is hyperosmotic and has a low pH. The extreme conditions of the citrate anticoagulant may damage the first portion of the blood collected and result in a so-called “citrate collection lesion.”\textsuperscript{17} We hypothesized that the ineffective filtration of RBCs from people with Hb AS is due largely to the collection of blood into citrate anticoagulant preservative, which results in the intracellular polymerization of Hb S. The low oxygen tension that is found in collected blood or that results from subsequent deoxygenation during storage and low pH and high osmolarity of the citrate anticoagulant solution could promote Hb S polymerization. The purpose of this study was to determine the role of Hb S polymerization in citrate-anticoagulated RBC components from Hb AS individuals in the performance of WBC reduction by filtration.

\section*{MATERIALS AND METHODS}

\textbf{Study design}

The goal of the first part of the study was to determine if a CP2D “collection lesion,” caused by the low pH (5.7) and high osmolarity (585 mOsm/kg) of the CP2D, contributed to filter failures. From each healthy donor studied, 250 mL of blood was collected into 31 mL of CP2D, and 250 mL of blood was collected into 2.5 mL (2500 units) of heparin. Both donors with Hb AS and without Hb S (Hb AA genotype) were studied. RBCs were prepared, AS-3 was introduced, and the RBC components were filtered. Blood counts, pH, oxygen tension, and osmolarity were assessed before and after filtration. Filtration time, filtration volume, RBC recovery, and residual WBC counts were compared.

To determine the role of Hb S polymerization in filter failure, 500 mL of blood was collected by phlebotomy into 63 mL of CP2D. RBCs were prepared and AS-3 was added. The component was divided into two parts; one half was treated with carbon monoxide gas for 60 minutes before filtration to convert Hb S to its liganded configuration, which prevents Hb S polymerization, and the other half was filtered without further treatment.

In the second part of the study, RBCs were collected by apheresis to determine if reduced exposure to CP2D would prevent filter failures. Each unit of apheresis RBCs was split in half. One half of the unit was filtered immediately, and its properties and filterability were compared with those of components collected by phlebotomy. The other one-half unit of apheresis RBCs was used for other studies.

\textbf{Donors}

These studies were approved by a National Institutes of Health Institutional Review Board, and informed consent was obtained before the blood was collected. Donors met all AABB criteria for donating whole blood. Sickle cell trait was confirmed by ion exchange high-performance liquid chromatography (HPLC) analysis of donor RBCs (Varian HPLC system [\textbeta-thalassemia short program], Bio-Rad Diagnostics Group, Hercules, CA). All donors were asked to disclose their current smoking status.

\textbf{Collection and filtration of phlebotomy}

\textbf{RBC components}

RBCs were collected into a modified collection bag set that included CP2D, AS-3, and an RBC WBC-reduction filter (RCM1, Leukotrap RC System, Medsep Corporation, Pall Medical, Covina, CA). The set was altered by removing half (31 mL) of the CP2D from the collection bag, removing half (50 mL) of the AS-3, adding an additional collection bag that contained sodium heparin (2.5 mL, 1000 units/mL; Elkins-Sinn, Cherry Hill, NJ), adding a bag containing AS-3 (50 mL), and adding a second WBC-reduction filter (RCM1, Pall Medical). From each donor, a volume of 250 mL of whole blood was collected into the bag containing CP2D, and 250 mL was collected into the bag with heparin. The bags were rocked during collection (Sebra, Tucson, AZ).

RBCs were prepared by centrifugation of whole blood collected in CP2D and heparin, and RBCs were filtered according to the manufacturer’s instructions, with the exception that only one half of the AS-3 (50 mL) was added to the RBCs. Components were defined as filtering completely if all RBCs drained out of the upper prefiltration bag and into the final RBC storage bag. Samples were taken before and after the addition of AS-3 and after filtration for measurement of complete blood counts, osmolarity, blood gases, and pH.

\textbf{Carbon monoxide treatment of phlebotomy}

\textbf{RBC components}

One unit of blood was collected in CP2D, RBCs were prepared, and AS-3 was added. The RBC component was...
then divided in half. One half was filtered as described above (RCM1, Pall Medical), and the other half was treated with carbon monoxide before filtration. To treat the component with carbon monoxide, the half unit was added to a tonometer (Fisherbrand septum-port gas sampling tube, 250 mL, Fisher Scientific, Pittsburgh, PA), and carbon monoxide (Aldrich, St. Louis, MO) was allowed to flow slowly through the tonometer at room temperature for 60 minutes as the tonometer was gently rocked in an exhaust hood. After 1 hour of incubation with carbon monoxide, the RBCs were transferred from the tonometer to a bag and filtered with a RBC WBC-reduction filter (RCM1). The filter was primed with AS-3, but rather than adding the AS-3 to the RBCs after the AS-3 passed through the filter, the AS-3 was diverted to an empty bag as it left the filter.

Collection and filtration of apheresis RBC components

RBCs were collected using a blood cell separator (MCS +, Haemonetics, Braintree, MA) according to the manufacturer’s recommendations, with the exception that only 1 unit of RBCs was collected. CP2D was added to whole blood as the blood was withdrawn from the donor’s vein, at a ratio of 1 part CP2D in 16 parts blood. Immediately after the RBCs were collected, the blood cell separator added the RBCs to a bag containing 100 mL of AS-3.

The apheresis RBC component was divided into two parts. One half was filtered within 2 hours of collection using an RBC WBC-reduction filter (RCM1). Before the RBCs were filtered, the filter was primed with AS-3 (as described above). The second half was used for other studies.

Blood counts, blood gases, and osmolarities

Blood counts were measured with an automated cell counter (Cell-Dyn 4000, Abbott Diagnostics, Santa Clara, CA). Blood gases, pH, sodium, potassium, chloride, bicarbonate, and glucose levels were measured with a blood gas analyzer (Radiometer ABL 700 Series, Radiometer Analytical SA, Lyon, France). Osmolality measurements were performed with a PSI-Multi-Osmette model 2430 instrument (Precision Systems, Natick, MA).

Statistical analysis

Values represent the mean ± 1 SD. Groups were compared using the students t-test. In some cases, paired t-tests were used.

RESULTS

Filtration of phlebotomy RBC components collected in CP2D

RBC components from six donors with HB AS were studied. All donors were healthy and met eligibility criteria for allogeneic blood donation. The donor’s mean age was 46 years (range, 32-53), one was male, and all were African American. The percentage of Hb S ranged from 33.7 to 39.0 percent. One Hb AS donor smoked cigarettes.

CP2D RBC components collected from five of the six Hb AS donors occluded the filter before all the RBCs passed through (Table 1). In two of these five donors, flow through the filter was completely blocked before any RBCs could pass into the collection bag. The Hb AS donor whose CP2D RBC component filtered completely was the only one who smoked cigarettes, and filtration time was 72 minutes. The RBC recovery of this donor’s component was 71 percent, and the residual WBC count was 0.11 x 10^6 cells.

RBC components were collected from six Hb AA healthy African Americans; their mean age was 39 years (range 32-49) and three were male. All six control Hb AA RBC components collected in CP2D passed through (data not shown). In two of these five donors, flow through the filter was completely blocked before any RBCs could pass into the collection bag. The Hb AS donor whose CP2D RBC component filtered completely was the only one who smoked cigarettes, and filtration time was 72 minutes. The RBC recovery of this donor’s component was 71 percent, and the residual WBC count was 0.11 x 10^6 cells.

RBC components were collected from six Hb AA healthy African Americans; their mean age was 39 years (range 32-49) and three were male. All six control Hb AA RBC components collected in CP2D filtered completely (data not shown). The mean filtration time was 18 ± 5 minutes and ranged from 11 to 26 minutes. The residual WBC counts in all six components were less than 0.15 x 10^6 cells. The RBC recovery of the CP2D components collected from the Hb AA donors was greater than the RBC recovery of components collected from the Hb AS donors (82 ± 4% vs. 26 ± 27%, respectively; p < 0.004).
Filtration of phlebotomy RBC components collected in heparin

To determine if CP2D contributed to filter occlusion in donors with Hb AS, one-half unit of blood was collected into heparin by phlebotomy from the same six donors with Hb AS. All six Hb AS RBC components collected in heparin filtered completely (Table 1). The residual WBC count in all six components was less than $1 \times 10^6$ cells. The RBC recoveries of the six components collected in heparin were greater than the RBC recoveries of the six components collected in CP2D from the same donors (78 ± 10% vs. 26 ± 27%, p < 0.005).

RBC components from the six donors with Hb AA (normal African Americans) were also collected in heparin and filtered. When the RBC recoveries and filtration times in the Hb AA donors were compared with those of the six components collected in heparin from Hb AS donors, no differences were found in filtration time (26 ± 15 min vs. 11 ± 5 min, p = 0.07) or RBC recoveries (78 ± 10% vs. 91 ± 10%, p = 0.10).

Comparison of the properties of RBC components collected in CP2D and heparin

There was no difference in pH, osmolarity, oxygen tension, Hb oxygen saturation, or MCHC between the Hb AS components collected in CP2D and heparin (Table 2). The similarities of these parameters among the RBC components collected in CP2D and heparin suggest that a citrate collection lesion was contributing to the filter failures.

Blood chemistry levels, pH, osmolarities, and oxygen saturations measured in whole blood were compared among components collected from Hb AS donors and from Hb AA donors (Table 2). When components were collected in CP2D, sodium, glucose, and osmolarity were lower and chloride, potassium, and pH were higher in components collected from Hb AS donors than in control Hb AA donors. There was no difference between the two groups in sodium, chloride, glucose, osmolarity, and pH among components collected in heparin. There was no difference in oxygen tension or oxygen saturation among Hb AS and Hb AA components collected in CP2D and heparin. The differences in laboratory parameters among Hb AS and Hb AA components collected in CP2D, but not those collected in heparin, suggest that CP2D had a greater effect on RBCs from donors with Hb AS than with Hb AA donors.

When blood is collected into CPD, the RBCs swell.16 As expected, when RBC indices were compared among RBC components collected from Hb AA donors in CP2D and from Hb AA donors in heparin, MCV was greater, and MCHC was less in CP2D components (Table 2). In contrast, no increase in RBC volume occurred when Hb AS RBCs were collected into CP2D. There was no difference in Hb AS donor MCV or MCHC between RBC components collected in CP2D and those collected in heparin.

Filtration of carbon monoxide-treated RBC components

To determine if Hb S polymerization was responsible for the occlusion of WBC-reduction filters, RBC components were treated with carbon monoxide to convert Hb S to its liganded form, which prevents polymerization. RBC components collected in CP2D from four Hb AS donors were divided in half; one half was treated with carbon monoxide before filtering and the other half was filtered without further treatment (Table 3). All four RBC components treated with carbon monoxide filtered completely, but only one of the four untreated components filtered completely. In addition, two of the six CP2D Hb AS RBC components collected in the first part of these studies did not filter at all. One component was hemolyzed and was not tested further, but the other component was treated with carbon monoxide and filtered again. After carbon monoxide treatment, this component filtered completely in 7 minutes, with a RBC recovery of 85 percent and a residual WBC count of 0.04 × 10^6 cells. The RBC recoveries of the five carbon monoxide-treated Hb AS components were significantly greater than those of the untreated components from the same donors (84 ± 4% vs. 32 ± 36%, p < 0.04).

Filtration of apheresis RBC components

Apheresis RBC components were collected from seven donors with Hb AS. All seven donors were African American, their median age was 39 years (range, 20-50), and

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**TABLE 2. Comparison of chemistry levels* in whole-blood components collected in CP2D and heparin in Hb AS and Hb AA donors**

<table>
<thead>
<tr>
<th>Number</th>
<th>CP2D</th>
<th>Heparin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hb AS</td>
<td>Hb AA</td>
</tr>
<tr>
<td>Sodium (mM)</td>
<td>142 ± 9</td>
<td>152 ± 4 †</td>
</tr>
<tr>
<td>Potassium (mM)</td>
<td>4.5 ± 0.9</td>
<td>3.2 ± 0.2 ‡</td>
</tr>
<tr>
<td>Chloride (mM)</td>
<td>97 ± 9</td>
<td>80 ± 3 †</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>277 ± 197</td>
<td>603 ± 88 †</td>
</tr>
<tr>
<td>Osmolarity (mOsm/kg)</td>
<td>300 ± 20</td>
<td>335 ± 7 †</td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>58.2 ± 23.4</td>
<td>53.7 ± 17.2</td>
</tr>
<tr>
<td>pH</td>
<td>7.22 ± 0.14</td>
<td>6.97 ± 0.08 †</td>
</tr>
<tr>
<td>MCV (fL)</td>
<td>84.3 ± 3.6</td>
<td>91.2 ± 6.7 ‡</td>
</tr>
<tr>
<td>MCH (pg)</td>
<td>28.7 ± 1.2</td>
<td>28.8 ± 2.0</td>
</tr>
<tr>
<td>MCHC (g/dL)</td>
<td>33.4 ± 9</td>
<td>31.6 ± 1.5 ‡</td>
</tr>
</tbody>
</table>

* Chemistries and RBC indices were measured in components at the time of collection.
† p < 0.02 for comparison of CP2D blood collected from Hb AS and Hb AA donors.
‡ p < 0.02 for comparison of CP2D and heparin components collected from Hb AA donors.
two were men. Five of the apheresis RBC components passed completely through the WBC-reduction filter. Four of these five apheresis RBC components filtered in less than 15 minutes and one filtered in 100 minutes. The residual WBC count in all five components filtering completely was less than $0.9 \times 10^6$ cells, and RBC recovery in all five was 85 percent or greater (Table 4). When the RBC recovery of components collected from Hb AS donors by apheresis was compared with components collected from Hb AS donors by phlebotomy into CP2D, RBC recovery was greater in the apheresis RBC components (67% vs. 28% to 27%, $p < 0.05$).

As a control, apheresis RBC components were collected from four healthy Hb AA male donors; three were African Americans and one was Caucasian. Their mean age was 38 years (range, 28-46). For all four control apheresis RBC components, filtration time was less than 10 minutes, RBC recovery was greater than 87 percent, and residual WBC counts were less than $0.3 \times 10^6$ cells. RBC recoveries were similar for apheresis RBC components collected from Hb AS and Hb AA donors (67±35% vs. 91±32%, $p = 0.22$).

**Properties of RBC components collected by apheresis**

Although several differences were found in the properties of CP2D components collected by phlebotomy among Hb AS and control donors, there were no differences in chemistries, RBC indices, pH, osmolarity, or oxygen saturation (sodium, potassium, chloride, glucose, MCV, MCH, and MCHC) among apheresis units collected from Hb AS and Hb AA donors (data not shown).

**Comparison of apheresis components filtering rapidly with those filtering slowly**

The two Hb AS apheresis components that occluded the filter and the one that completed filtration in 100 minutes were considered “poorly” or “slowly” filtering components. The properties of the four apheresis Hb AS components that filtered “rapidly” (in less than 15 minutes) were compared with those of the three units that filtered “poorly.” There was no difference in Hb S fraction, pH, or MCHC between the two groups, but the oxygen saturation was greater in the rapidly filtering group ($68 \pm 9\%$ vs. $37 \pm 5\%$, $p < 0.03$) (Fig. 1).

**DISCUSSION**

Polymerization of Hb S during the collection and processing of blood appears to be responsible for the ineffective performance of RBC WBC-reduction filters with RBC components collected from Hb AS donors. Because oxygenated or liganded Hb S does not polymerize, the finding that Hb AS components treated with carbon monoxide can successfully undergo WBC reduction by filtration suggests that Hb S polymerization is responsible for filter performance problems. When Hb S polymerizes, RBC intracellular viscosity increases, reducing deformability and impairing filterability. The trapping of RBCs with polymerized Hb S in WBC-reduction filters leads to either complete obstruction of flow or channeling of flow that makes filtration ineffective.

These findings are significant because the Hb S polymerization in RBCs from persons with Hb AS was not thought to be of clinical relevance during blood donation. In healthy donors, oxygen saturation is generally considered high enough to prevent Hb S polymerization. However, this study shows that nonphysiologic conditions present in blood collection bags can be associated with Hb S polymerization.

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**TABLE 3. Filtration of Hb AS RBC components collected in CP2D and treated with carbon monoxide**

<table>
<thead>
<tr>
<th>Component</th>
<th>Untreated components</th>
<th>Carbon monoxide-treated components</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Filtration outcome</td>
<td>Filtration time (min)</td>
</tr>
<tr>
<td>7</td>
<td>complete</td>
<td>26</td>
</tr>
<tr>
<td>8</td>
<td>obstructed</td>
<td>&gt;120</td>
</tr>
<tr>
<td>9</td>
<td>obstructed</td>
<td>&gt;120</td>
</tr>
<tr>
<td>10</td>
<td>obstructed</td>
<td>&gt;120</td>
</tr>
</tbody>
</table>

**TABLE 4. Filtration of RBC components collected by apheresis from Hb AS donors**

<table>
<thead>
<tr>
<th>Component</th>
<th>Hb S (%)</th>
<th>Filtration outcome</th>
<th>Initial volume (mL)</th>
<th>Filtration time (min)</th>
<th>RBC recovery (%)</th>
<th>Residual WBCs ($\times 10^6$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39.0</td>
<td>complete</td>
<td>162</td>
<td>12</td>
<td>86</td>
<td>0.41</td>
</tr>
<tr>
<td>2</td>
<td>37.3</td>
<td>complete</td>
<td>162</td>
<td>8</td>
<td>85</td>
<td>0.04</td>
</tr>
<tr>
<td>3</td>
<td>30.4</td>
<td>complete</td>
<td>160</td>
<td>10</td>
<td>91</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>39.2</td>
<td>obstructed</td>
<td>155</td>
<td>&gt;120</td>
<td>13</td>
<td>NA</td>
</tr>
<tr>
<td>5</td>
<td>38.3</td>
<td>complete</td>
<td>158</td>
<td>6</td>
<td>93</td>
<td>0.89</td>
</tr>
<tr>
<td>6</td>
<td>39.2</td>
<td>obstructed</td>
<td>160</td>
<td>&gt;120</td>
<td>17</td>
<td>NA</td>
</tr>
<tr>
<td>7</td>
<td>35.1</td>
<td>complete</td>
<td>160</td>
<td>100</td>
<td>85</td>
<td>0.24</td>
</tr>
<tr>
<td>Controls (n = 4)</td>
<td>0</td>
<td>complete</td>
<td>155-169</td>
<td>8 ± 2</td>
<td>91 ± 3</td>
<td>&lt;0.3</td>
</tr>
</tbody>
</table>
AS and Hb AA donors. We speculate that these differences in chemistries and RBC indices in CP2D components collected by phlebotomy between Hb AS and Hb AA donors resulted in Hb S polymerization in at least the first portion of blood collected, though it is not clear why this lesion does not rapidly reverse.

Further evidence for citrate solution-induced Hb S polymerization in Hb AS blood was provided by the difference in chemistries and RBC indices in CP2D blood collected from Hb AS and Hb AA donors. Sodium, potassium, chloride, glucose, osmolarity and pH differed in CP2D blood collected from Hb AS and Hb AA donors. We speculate that these differences are due to the polymerization of Hb S. The fact that no difference appeared in osmolarity or glucose levels in blood collected in heparin supports the conclusion that CP2D collection lesion is responsible for Hb S polymerization.

We found that apheresis RBC components collected from Hb AS donors filtered more effectively than RBC components collected by phlebotomy into CP2D. The improved filterability of apheresis RBC components is likely due to avoidance of the citrate collection lesion. In contrast to phlebotomy-collected CP2D components, no differences in the properties of apheresis components collected from Hb AS and Hb AA donors were noted, indicating that polymerization was less problematic in apheresis components. The reduction or elimination of the citrate collection lesion in apheresis RBC components is likely due to the method of addition of CP2D. During apheresis, CP2D is added to the blood at a carefully controlled rate proportional to the whole-blood collection rate. In contrast, blood collected by phlebotomy flows into a bag containing enough CP2D to anticoagulate an entire unit.

Factors other than citrate collection lesions also contributed to Hb S polymerization. Although filtration of apheresis RBC components was superior to phlebotomy components, some apheresis components did not filter effectively. These components had lower Hb oxygen saturations levels than those that filtered completely. It is unclear why Hb oxygen saturation levels vary among donors.

Blood from Hb AS donors collected in heparin filtered effectively, but heparin is not a suitable anticoagulant. An alternative to collecting blood in CP2D is to collect it in another citrate-based anticoagulant such as CPD or CPDA-1. The osmolarities of CPD and CPDA-1 are less than that of CP2D because they contain less dextrose. However, CPD and CPDA-1 are still hyperosmotic and acidic, and RBC filter performance problems have been reported with RBC components from Hb AS donors collected in CPDA-1.5,20

These studies have several implications. Because filter failures were due to changes in the rheologic properties of the Hb AS RBCs, it would be expected that without some intervention to prevent Hb S polymerization, RBC components from Hb AS donors would occlude most, if not all, WBC-reduction filters. The incidence of failure may vary among filters depending on the construction of the filter and filter material, the type of citrate anticoagulant, the temperature of the blood at the time of filtration, and the interval of storage before blood was filtered. It is likely that RBC components stored in gas-permeable bags before filtration would have increased oxygen tensions and would filter more effectively.

Our data show that when blood from donors with Hb AS is collected under the appropriate conditions, filters...
can be used to remove WBCs from RBC components. It is important to use existing collection systems, such as apheresis, or to develop new systems that permit WBC reduction of RBC components from Hb AS donors. Hb AS is most prevalent in African Americans, and because this population is underrepresented among blood donors, high priority should be given to future efforts to adapt RBC collection techniques such that donors with Hb AS are not excluded from blood donation.

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REFERENCES