Many African Americans with sickle cell disease (SCD) develop alloantibodies to antigens in the Rh blood group system. Others have shown that from D– individuals, those lacking the high-incidence hrB antigen (> 98% prevalence) may be found among r'r African Americans. We describe an algorithm to locate units for African Americans with SCD and anti-hrB and -D. From 46,539 donations, 5136 listed African American as race. Our primary reference laboratory performed Rh phenotyping (D, C, c, E, e) for first-time donors and those not tested previously. Specimens typing r'r were sent to a secondary reference laboratory for hrB phenotyping after each donation. Hemoglobin S screening was performed. Of 24 donors (27 donations) who phenotyped r'r, seven donors, 29.2 percent (nine donations) were hrB–. Two of seven who donated twice consistently tested hrB–. One of 24 donors initially tested hrB–, but hrB+ on repeat donation. The donor tested hrB– by a second reference laboratory. We felt this was the most judicious use of resources and provided the greatest opportunity to find compatible components for individuals with SCD and anti-hrB and -D.

Materials and Methods
During a 10-month period, 46,539 volunteers at our blood collection center were asked to designate their race. The software in use in our center allowed classification of race into one of six categories. Donors did not have the option to designate more than one race. During the study, 5136 donors listed themselves as African American. The primary reference laboratory for our blood collection center performed an Rh phenotypic evaluation (using anti-D, -C, -E, -c, -e) for first-time African American donors or for those for whom no previous phenotype was available. Rh phenotyping was performed by tube testing according to manufacturers’ instructions.

Specimens from donors who typed as r'r were sent to a secondary reference laboratory for hrB phenotyping. Specimens from r'r donors were typed for hrB after each donation. Rh phenotyping was repeated only for those Rh antigens that typed negative on the previous donation. At least two different sources of anti-hrB were used for evaluation.

Donors who phenotyped as r'r were screened for the presence of hemoglobin S, using a solubility assay (SickleScreen, Pacific Hemostasis, Huntsville, NC).

If a unit typed hrB–, hemoglobin S negative, it was inventoried and stored frozen for the primary reference laboratory at the discretion of the Technical Director. Information on the donor was entered into a database for future recruitment (Fig. 1).

Due to our patients’ need, our study was limited to hrB–, D– donors.
Results

Twenty-four African American donors (from 27 donations) were phenotyped as r'r. Seven donors, 29.2 percent (from nine donations) were found to be hr B–. Two of the seven donors who presented twice consistently tested hr B–. One of the 24 donors initially tested hrB–, but tested hrB+ on repeat donation. Because of discrepant test results, that sample was sent to an independent reference laboratory, which reported the donor to be hrB–. Future units from this donor were not transfused to individuals with anti-hrB. Using the algorithm, the probability that an hrB– donor would be found increased from 2 percent to 29.2 percent (Fig. 2).

Discussion

High-incidence antigens are observed in more than 90 percent of most random populations. Individuals with congenital hemolytic anemias, such as SCD, who require repeated transfusions often develop multiple antibodies, some of which may be to high-incidence antigens. In some situations, because of the rarity of blood that is negative for the antigen, as may be the case with hr⁺, it may be necessary to go beyond local inventories and institute a nationwide search. These actions may result in compatible units not being available to satisfy clinical urgency.

To expedite transfusion when an individual develops an antibody to a high-incidence antigen, reference laboratories often keep inventories of frozen RBCs with uncommon phenotypes.
Our primary reference laboratory continually screens donors in an attempt to maintain an inventory that would meet the clinical needs of those individuals. The inventory in most cases is an actual unit, frozen at the discretion of the Technical Director of the reference laboratory, but it may also be a virtual unit in that the donor's name is added to a database. The donor is recruited as the need arises and the unit is captured for inventory.

Using our algorithm, the probability that an hr\(^a\)-donor would be found increased 15-fold, from 2 percent to 29.2 percent (Fig. 2). We feel that this allowed for the most judicious use of scarce reagents and provided the greatest opportunity for us to find compatible blood components for those individuals with SCD and anti-D and -hr\(^a\).

Although we limited our study to those patients with SCD, this algorithm could be applied to other chronically transfused populations at risk for alloantibody formation, such as those with congenital hemolytic anemias and chemotherapy patients.

References

Richard R. Gammon, M.D. (corresponding author), Associate Medical Director, South Florida Blood Banks, Inc., 933 45th Street, West Palm Beach, Florida 33407, e-mail: rgammon@sfbb.org; and Norberto D. Velasquez, Jr., MT, BB, (ASCP)SBB, Technical Director, Reference Laboratory, Association of Independent Blood Centers, Inc., West Palm Beach, Florida.