Declining collection and transfusion of blood: Whither goest transfusion medicine?

In two back-to-back articles (see references 1 and 2), two separate surveys, one governmental and one from AABB, reviewed data from 2013 showing a decline in both blood collections and blood product administration over the previous two years. Because the United States has a decentralized blood system, unlike most other more developed countries in the world, including Canada, it has always been difficult to access enough complete data to get an accurate picture of what goes on regarding blood component production and transfusion in this country. This, in turn, makes central planning and coordination a complex task. The best we have been able to do is to complete a national survey every other year.

The AABB Blood Collection, Utilization, and Patient Blood Management Survey (BCUPBM) of 2013 was conducted by a thorough review/survey of appropriate data from all AABB approved institutions. The information was then compared to the similar survey of 2011, using only U.S. member institutions to provide these comparative data. The Department of Health and Human Services has conducted a biennial National Blood Collection and Utilization Survey (NBCUS) every 2 years since 1997. Sometimes, but not always, the AABB survey has been used for that purpose. The hospitals surveyed by the NBCUS include all hospitals in the American Hospital Association Annual Survey database hospital registry that perform at least 100 inpatient surgical procedures a year, not just those who are members of AABB.

Despite the fact that the surveys serve two different masters, and for different reasons, the data are pretty similar, after adjusting for the models, data weightings, and comparisons with prior surveys. Both of the articles cover the same period of time, and compare 2013 to 2011. Compared to 2011, whole blood and red cell collections have declined significantly in 2013, even more so than the decline in actual transfusions. Distribution of platelets and plasma declined, and plasma transfusions did as well, while platelet transfusions increased. These facts are similar between the two surveys. The gap between collection and utilization is narrowing, which suggests improvements in product and inventory management.

Why do we have two surveys, how did we end up with this duplication of effort and expense? Dr. Harvey Klein from the Department of Transfusion Medicine, NIH, (see reference 3), wrote an editorial providing perspective. He notes that the government imperative is to promote a safe and available national blood supply by supporting comprehensive data collection and analysis. The AABB Survey is designed to...
meet membership wants and needs, to gather information about AABB members, to support AABB policy decisions and provide members with benchmarks.

Those of us who have been around a while will recall the 1972 report, conducted under contract to Booz, Allen, Hamilton, commissioned by the National Heart and Lung Institute (now called, tellingly, the National Heart, Lung and Blood Institute) that reported on our fragmented blood system. It especially noted a lack of effective regulation, underserved patients, and a lack of comprehensive and reliable data on which to base policy decisions. A test for HBV was being implemented at that time, but post-transfusion hepatitis was still a big problem, and HIV remained unknown/unrecognized for almost another decade.

Dr. Klein reviews the events that followed and how they have improved our blood services in fits and starts. The development of a national blood policy, the American Blood Commission begun in 1974, and the National Blood Data Center that became the National Blood Data Resource Center, were all part of the story, but not all survived the clash of the private and public cultures that were involved. Nonetheless, transfusion of whole blood diminished and blood component production flourished, as did the concept of an all-volunteer blood donor base. DHHS’s Blood Data Resource Center has (unfortunately, perhaps) morphed into two entities, DHHS’s biennial National Blood Collection and Utilization Survey, and the AABB’s Blood Collection, Utilization, and Patient Blood Management Survey.

It is lamentable that two similar, but different, surveys are carried out, and consume time and resources. At NIH’s Clinical Center, which collects, processes and transfuses blood components for their clinical studies, it took a week for an experienced supervisor to complete both surveys, since the questionnaires differed so much in need and structure. He makes the case for a single entity/survey, but notes the problems to be overcome. He believes 10 cents per unit of blood collected would pay for the whole thing, which data should then be published in peer-reviewed journals where it will be forever available. You would be enlightened by reading this editorial.


Modeling the risks of emerging threats to the blood supply

For some time we have all been commenting and discussing the fact that more and more transfusion-transmitted emerging infectious diseases (EIDs) are being identified, and they are still “coming ashore”. Until we have some sort of method to render all prions, viruses, protozoa, bacteria and fungi non-infectious in ALL our blood components, it isn’t always easy to decide which of them pose significant problems that we should strongly address. Or as workers in this paper from the Netherlands say: How do we value the risk from a yet unknown emerging threat? They worked to develop a model for doing so, a model that requires estimates of four disease characteristics and their accuracy.

Using several panels of experts, the authors first developed a list of hypothetical EIDs and their characteristics: transfusion transmissibility (high, medium, low); mortality (high, moderate, low); disease severity (high, moderate, low);
asymptomatic pathogenemia (years, months, days); and, prevalence of infection in the population (high, low). A more detailed list of definitions was developed, which led to a list of 162 possible combinations and a definition of 16 hypothetical EIDs that seemed reasonable. Experts were asked to assign a weight, or score, to each hypothetical EID, and the relative risk of a particular disease compared with the others. As a second attempt, they combined mortality and disease severity into a single factor. As a sort of control, they applied the same parameters to some known EIDs.

The four characteristics now were transfusion transmissibility, an asymptomatic phase, prevalence of the infection, and disease impact. Various exercises involving numerous experts were carried out using mathematical modeling too detailed to include here (which partly means this writer didn’t really understand it!) Their results indicated that prevalence and transmissibility were the most important risk drivers, but if disease characteristics were not known, transmissibility and impact were more important. Interestingly, when ranking known EIDs that pose a serious risk to blood transfusion, they appeared at the top of the list, suggesting the model is useful.

The authors propose that EID risks can be systematically modelled and that further refinements can be developed. One problem with this method is that it doesn’t account for political muscling or public concern/hysteria which may have a real effect on the funding and implementation of preventive or corrective efforts.


What’s in a word?

Last year there were two articles in Transfusion in which the authors proposed changing the descriptions/definitions of “possible TRALI” associated with acute lung injury but without the presence of HLA-antibodies and/or human neutrophilic antibodies, HNA. These antibodies define TRALI. However, clinically very similar cases were occurring in their absence, and so “possible TRALI” was used, in which a two-hit, or threshold model was postulated. A first “hit” might be cardiac surgery, or severe sepsis, which made the patient more susceptible to lung injury, followed by the transfusion of blood, the second hit. There are no HLA or HNA antibodies in this blood. So there was antibody-mediated TRALI, and non-antibody related TRALI. This latter morphed into “possible TRALI”, said to occur in about 20% of apparent cases of TRALI.

The two articles suggested that “possible TRALI” not be used, but preferred (in one case) to say “transfused ARDS”. The other paper, comparing the causes of heparin-induced thrombocytopenia (HIT) with those of TRALI, suggested that TRALI and “non-immune” TRALI might be used, since it has been established that there is apparent HIT that is not associated with the presence of platelet antibodies in cases clinically similar to immune HIT.

In a recent article (see references), Swedish authors reviewed the history and definition of TRALI. These cases were associated with blood or plasma from (predominantly) women sensitized from pregnancy or prior transfusion. Symptoms classically began within 6 hours of transfusion, with signs of lung infiltrations by chest X-ray in the absence of pulmonary edema. As time passed, the immune causes of TRALI and the incidence of reported TRALI cases increased and it became the most significant cause of transfusion-related death, despite the fact that avoidance of blood/plasma from previously pregnant women became widespread. The authors believe it is too early to propose that antibody-mediated TRALI is the only true TRALI. They believe that more sensitive assays for HLA and HNA, and perhaps antibodies related to red cell or platelet...
storage times seen in some animal models might be among the causes for “possible TRALI”.

In response, the authors of one of the prior papers discussed by the Swedish authors cited evidence from a study where the age of the stored platelets, even 4 days and older, provided no evidence as a risk factor for TRALI, nor did storage time of a single unit of RBCs. They urge that “possible TRALI” cases must be carefully evaluated when both transfusion and an alternate ARDS factor are present within 6 hours of transfusion, and if no antibodies are found using current methods, no investigation for donor antibodies should be done and the cases labeled as “transfused ARDS”.

The authors of the second paper, which viewed TRALI through the HIT perspective, also had specific facts to support their argument. However, they agree with the Swedish authors that TRALI be regarded as a clinical syndrome that is caused by an antibody-mediated and a non-antibody mediated one which cannot be distinguished at the bedside. They urge that the presence or absence of detectable HLA or HNA antibodies be used in any definition of TRALI.

So, what’s in a word? Stay tuned!


When to transfuse and with which unit of red cells

Over the past year or so, we have discussed several randomized controlled studies that related to both of the questions: “What is the safe threshold of Hgb to determine when to pull the red cell transfusion trigger in patients in various clinical settings?” and, “Is there an indication for using “fresh” blood vs “old” for transfusion in certain circumstances?” Many clinicians, especially surgeons, believed that fresher blood was better, while most blood banks, and blood collection agencies in particular, preferred to use the “oldest” blood in their inventories first, perhaps most especially to retard outdating of a precious, and expensive therapeutic product.

Now we have a masterful review of clinical practice guidelines related to both thresholds for transfusion and storage times, from the work of a distinguished expert panel which contains many of the authors whose work we have looked at in the past few years. (See reference.) The review included a literature search (not restricted to the English language) by reference librarians for randomized clinical trials (RCTs) that evaluated Hgb transfusion thresholds from 1950 to May of 2016, and RCTs on storage duration from 1948 to May, 2016. For transfusion thresholds, they found 31 RCTs that included 12,587 recipients. For the red cell storage duration, there were 13 RCTs that included 5,515 participants.

In 2012, AABB published guidelines based on 19 RCTs that included 6,264 patients, many of which were from pretty small studies. During the past 4 years the number of patients enrolled in RCTs has more than doubled; many of these later studies have included methods to reduce the bias introduced by studies with small numbers and have enrolled populations of patients known to receive frequent transfusions. Seven of the 13 RCTs related to the effect of storage duration on patient outcomes have been reported since 2012. The authors state, however, that there is as yet no formal guidance on the optimal length of RBC storage time prior to transfusion.

Continues on next page
When making transfusion decisions on an individual patient, it is good practice to consider the Hgb level, the overall clinical context, patient preferences where applicable, and alternative treatments. The authors have some recommendations. First, a restrictive RBC transfusion threshold in which transfusion is not indicated until the Hgb level is 7g/dl is recommended for hospitalized adults who are hemodynamically stable, including critically ill patients, rather than the “traditional” threshold of 10 g/dl. This not only saves money and blood, but in many studies the rates of infection and other complications were reduced. For patients having orthopedic surgery, cardiac surgery or with pre-existing cardiac disease, a restrictive threshold of 8g/dl is indicated. Many, if not most, procedures in this category could likely be done at 7g/dl, but most of the studies used an 8g/dl threshold. Neither of these recommendations should be applied to patients with acute coronary syndromes, severe thrombocytopenia (generally patients undergoing hematologic or oncologic treatments at risk of bleeding), and chronic transfusion-dependent anemia—the evidence was insufficient to make a specific recommendation.

A last recommendation was that all patients, including neonates, should receive red cells selected at any point during their licensed storage period, as opposed to the use of “fresh” blood, with less than 10 days of storage.


Cryoprecipitate, a cold-insoluble fraction of fresh frozen plasma, was discovered and developed as a therapeutic tool by Dr. Judith Poole and colleagues in the late 1950s. It is made in the blood bank and contains a concentrated amount of Factor VIII (FVIII), Factor XIII, (FXIII) von Willebrand Factor (vWF), fibrinogen and fibronectin. It is now very rarely used for FVIII, FXIII or vWF deficiency, as more effective and safer factor concentrates are available. The authors, from New York City and Zurich (see reference), performed a retrospective review of cryo use in their hospitals over a recent 4.5 year period.

There were only 44 patients (median age 1.7 years) at a large tertiary medical center who received cryo during that period, the only indication for which was fibrinogen replacement. Most of the cases involved recent cardiac surgery (39%) or disseminated intravascular coagulation (DIC) due to sepsis (32%). Cryo was often transfused at a higher than recommended dose and without a defined pre-transfusion fibrinogen level. Of the 44 patients 61% were considered to be transfused inappropriately. 41% were transfused prophylactically. When the indication selected when ordering by the physician was compared to the clinical scenario the order was often incorrect. Most of the noted indications were for fibrinogen replacement. One patient who received cryo for DIC had a pretransfusion fibrinogen level of 233mg/dL. Of two patients with a selected indication of von Willebrand Disease, neither had it. Of the 24 patients for which “fibrinogen less than 100 mg/dL with bleeding” was selected, 8 of them either had a higher fibrinogen level, 3 of which were greater than 300 mg/dL, or no fibrinogen level had actually been measured.

In only 18% of the patients did the indications for cryo selected by the physicians actually match the clinical scenario as assessed by chart and laboratory review.

This is an example, of course, of improper use; however, it also points out the fact that despite the blood bank’s guidelines there is a lack of standardization and adherence to proper protocols for the use of cryoprecipitate in critically ill children. Perhaps it’s time for more randomized clinical trials in the use of coagulation therapy in this patient population.

Factor V deficiency

In addition to the rare (1 in a million) cases of congenital Factor V deficiency there are acquired cases thought to be mostly due to autoantibodies that arise spontaneously or from exposure to bovine thrombin used as a topical agent for hemostasis. It is thought that the bovine Factor V present with bovine thrombin causes the development of Factor V antibodies that cross-react with human Factor V. Factor V is primarily synthesized in the liver, but about 25% of circulating Factor V is stored in platelet alpha granules, which are released at sites of clot formation. Since this is an action specifically located at a bleeding site, some think that platelet transfusion is a more effective way to treat bleeding due to Factor V deficiency. HCV infection and antibiotic exposure have also been linked to autoantibody formation.

Physicians from the University of Texas’ Southwestern Medical Center in Dallas reported on three cases of Factor V deficiency, one congenital and two acquired, illustrating the need to identify specific etiologies to account for a low level of Factor V. The first case was that of a 30 y/o man who was known to be congenitally deficient in Factor V and whose bleeding problems had improved to some degree as he aged. He presented with a growing post-traumatic hematoma. He had generally been treated with platelet transfusions, and was again, this time with 5 units of platelets over 48 hours. His level was 1%, on admission and mixing studies showed no inhibition of Factor V in normal plasma.

The second patient had end stage renal disease (hypertension) and was admitted with volume overload and pneumonia. She underwent dialysis and antibiotic therapy with piperacillin-tazobactam and vancomycin and was discharged on moxifloxacin. She returned shortly thereafter with volume overload. PT and PTT levels were normal, but blood cultures were positive for Enterobacter species. She went home with two weeks of ciprofloxacin, but returned again with persistence of right arm pain, which she noted on first admission but for which no cause was found by venogram. This time, her venogram showed a partial occlusion of the right brachiocephalic vein, and her PT and PTT were both prolonged. She was found to have a Factor V inhibitor. After steroid therapy, her clotting studies became normal and the venous occlusion was resolved.

The third patient was an elderly woman with Stage 3 chronic kidney disease who presented with pneumonia and shortness of breath and was treated with several antibiotics. Her PT and PTT were both prolonged, her Factor V level was less than 1% and a mixing study showed inhibition. She also responded to a short course of prednisone. The message here is that congenital V deficiency is pretty rare, and it’s important to rule out an acquired inhibitor, since the treatment for this is considerably different.

The elucidation in the 16th and 17th centuries of the anatomy of the cardiovascular system and the circulation of blood by Vesalius, Harvey and Marpighi led directly to an interest in blood transfusion by physicians on both the British and French sides of the English Channel. (Note, however, that two of the above scientists were Italian.) Practitioners started with animal blood, but soon ran afoul of authorities and peers due to the reactions/deaths that ensued. One practitioner was accused of murder, and most activity stopped for a while. However, James Blundell (1790-1877), now known as the father of modern obstetrics, developed several tools to aid in direct human-human transfusion. He performed about 10 such transfusions on women afflicted with potentially fatal post-partum hemorrhage, and saved several. Karl Landsteiner’s work describing ABO blood groups didn’t occur until 1901, and work related to the Rh factors by another Italian, Carlo Moreschi, in 1911, was lost until it was rediscovered in 1945 after the war by R.R.A. Coombs. Peri-partum transfusions were now much safer, and the need was great. Even today, obstetrical hemorrhage is a leading cause of maternal mortality, with over 140,000 deaths from childbirth occurring annually worldwide. In many, if not most, less-developed countries, it is the single most frequent indication for transfusion; in fact peri-partum mortality worldwide is an indication of the lack of adequate health and blood services in many areas of the globe. Massive blood loss at parturition still occurs, and the frequency may be increasing due to the ongoing increase in second and third, or more, caesarian sections in previously sectioned mothers. Primary factors are abnormal placentaion (26.6%, see below), uterine atony (21.2%), placental abruption (16.7%), and postpartum hemorrhage due to a coagulopathy (15%).

More than 1.2 million women in the U.S. had a C-section in 2014. The incidence of placental accreta has increased 10-fold in the last 50 years, according to our authors from Boston (see reference), and occurs when there is abnormal adherence of placental villi to the myometrium. The most common cause for this is previous C-sections, particularly those associated with placenta previa, where the placenta implants over the internal mouth of the cervix. Placenta increta occurs when chorionic villi of the placenta invade the myometrium. Placenta percreta is when the villi invade through the myometrium to the serosal surface of the uterus, and in some cases invade nearby bowel or bladder. Thus, huge volumes of blood may be lost quite rapidly. Because of these concerns, and evidence that the problems are increasing with more C-section births, the authors from Beth Israel and Deaconess Medical Center centralized the care of these patients in a single comprehensive care center with a large number of specialists to discuss and carry out the care of these patients.

The authors, having laid this groundwork, go on to describe what their overall program consists of, including: the role of the blood bank; the need for available components; their massive transfusion protocol; the need for additional products or coagulation factors; and, the kind of specialized laboratory support required. After every event involving the use of their massive transfusion protocol, they have a post hoc review to discuss what worked, what didn’t go as well as planned and ways to improve the process. Specific transfusion service issues include the timeliness of blood product delivery to the delivery room, product wastage due to improper storage in the OR or delivery suite, or delayed return to the blood bank. The article needs careful reading of the details in the first part of the above paragraph, as there are many critical points in all those topics too numerous to detail here. Their massive transfusion protocol is important and of considerable interest, as is the description of the role of the blood bank. The authors emphasize that when a patient presents with one or more identifiable risk factors, a multidisciplinary plan of care must be rapidly developed and implemented, and that a massive transfusion protocol is critically important to care. Ongoing assessment and review of cases provides an opportunity to continuously fine tune the process and make changes based on various risk factors and outcomes. Although this paper is about obstetrical disasters, there are many excellent ideas that translate directly to any organized response to massive hemorrhage.

Herpesviruses comprise a large class of viruses, including varicella-zoster virus (chicken pox and shingles), herpes simplex viruses (HSV-1, oral, HSV-2, genital), Epstein-Barr virus (infectious mono, Burkitt’s lymphoma), human herpesviruses 6 and 8 (roseola, Kaposi’s sarcoma) and human cytomegalovirus (CMV). Congenital CMV causes severe anomalies in newborns, and severe, life-threatening illness in immunosuppressed individuals including in neonates and the elderly, the latter often on chemotherapy for lymphoma or other malignancy. CMV derives its name from the large, multi-nucleated cells with cytoplasmic as well as peri-nuclear inclusions. As with several of its herpesvirus cousins, it is very widespread, and after an acute infection – usually in childhood – antibodies remove the virus from the circulation, but it persists in latent form. Varicella-zoster remains dormant in sensory nerve root nuclei, as do herpes simplex agents, commonly the trigeminal nerve for HSV-1, and sacral nerve roots for HSV-2.

In healthy infants or adults, the acute infection is pretty benign. Acute CMV infection is similar to infectious mono, and after it is cleared from the blood it becomes latent and persists in mononuclear cells, primarily lymphocytes. Acute or reactivation infections can cause encephalitis, pneumonitis, pericarditis, hepatitis and nephritis in immunocompromised children or adults, with severe consequences. Included in this category are intrauterine transfusions of CMV-positive blood. Because of the ubiquity of CMV, and the fact that most blood recipients (of any age) are seriously ill, universal leukocyte reduction has become widespread, thereby additionally reducing the side effects of leukocyte administration: fever, chills, non-hemolytic transfusion reactions.

Leuko-reduced blood products have very successfully reduced CMV transmission, some reports show it being eliminated altogether. This is NOT true of bedside filtration. Some controversy has existed between filtration-only fans and others who feel that blood transfused to children, neonates especially, should be CMV seronegative, as well, a position some call a “belt and suspenders” opinion. Many hospitals have used the techniques additively, making for serious availability and timeliness problems. Their argument was that someone could be viremic with CMV for the first time, have very few symptoms but have circulating virus in the plasma that had not yet been incorporated into mononuclear cells. But it could also be said they had not seroconverted. Careful studies of seroconverting donors found that the number of “free” plasma virions was very low when the infection begins and no antibody can be found. As antibodies develop, they rapidly clear out the virions that now go to “hibernate” in the cells.

In a small study in the same issue as the Editorial by Strauss (see references), a study designed to compare leukocyte reduction only to leukoreduction plus CMV seronegativity in transfused blood in neonates, found filtration alone to be equal to leukocyte reduction plus seronegativity. Several were transfused with 1 or more seropositive units that had been leukocyte reduced.

Looking at all the available information, Strauss and an AABB committee have stated the case for leukoreduction alone, using modern LR technology, as their recommendation for avoiding CMV transmission from transfusion.


Finding an antidote for direct oral anticoagulants: of -mabs, -bans and -nets

Many clinicians have been reluctant over the past few years to use the direct-acting anticoagulants that bind to thrombin and to activated Factor X, Factor Xa. These drugs – dabigatran, rivaroxaban, apixaban, edoxaban, and betrixaban - are very effective and generally have a very good safety record. Many feel they are safer than the traditional long-term anticoagulant, warfarin, which acts to inhibit the production of Factors II, V, VII and X. It should be noted, though, that warfarin is still the anticoagulant of choice when dealing with artificial surfaces, such as heart valves.

There are many variables that affect the absorption and efficacy of warfarin, since many drugs and foods interfere with it to the degree that fairly frequent monitoring (every 2-4 weeks, once stable), and in some cases many dose adjustments, are required. Bleeding and bruising are not uncommon, but not often life-threatening, for example, causing intracranial hemorrhage at a rate of about 0.2% per year. The prothrombin time can be corrected with intravenous vitamin K, or with heat-treated plasma-derived prothrombin complex concentrates. The use of fresh frozen plasma is controversial. Simply omitting doses takes several days to show an effect on the PT/INR.

Until recently there have not been clinically practical agents that can be used to reverse the effects of the Xa inhibitors. In 2015, idarucizumab, a monoclonal antibody that targets the active site of the thrombin inhibitor was licensed, but it is only effective in patients receiving dabigatran, which works as a thrombin inhibitor. The other four anticoagulants work by binding to Factor Xa. The authors of the first reference demonstrated effective reversal by a new agent, andexanet, of the anticoagulant effects of rivaroxaban, apixaban, edoxaban, and betrixaban. It also works to reverse the effect of low molecular weight heparins. The Connoly group found that within 12 hours after administration of andexanet, more than 80% of patients had normal hemostasis.

The authors of the editorial point out, though, that the administration of andexanet, combined with cessation of the Xa inhibitor, led to high rate of thrombosis over the next 30 days, 12 of 67 patients. This raises an etiological question: does andexanet have an intrinsic prothrombotic effect, or do these results simply reflect the absence of antithrombosis in a high-risk group of patients? The authors of the editorial, while noting the importance of this new agent, point out that the actual need for such an antidote to these direct anticoagulants is very small, and that simple cessation of treatment may be all that is needed in most cases.


For how long should we defer Babesia positive donors?

Since first recognized in 1980, there have been over 200 cases of Babesia microti reported as transmitted through infectious blood, and two-thirds of these occurred since 2000. At least 31 of them resulted in a fatality. People needing a blood transfusion are not always at their immunological best – newborns, older patients, asplenic individuals and the immunocompromised or immunosuppressed – and are far more likely to become ill from Babesia. In healthy adults, the disease may go unrecognized or be just a mild illness. The fact that there is no agreed upon licensed test for it makes blood donor screening difficult, so we are indefinitely deferring all donors with a history of infection so as to diminish transfusion-transmitted babesiosis (TTB). This strategy is not enough, as evidenced by the increasing rate of TTB.

In addition to the large numbers of infected ticks in most of New England, New York, and other northeastern states, there is moderate endemicity in the upper Midwest, as well as related illness to a lesser degree in parts of the upper west coast. The northeast, though, is where the incidence of infection in humans and ticks is the greatest, and seropositivity in the population...
is highest. It is true that a seropositive donor may have, on close examination, no parasitemia; however, it is also true that parasitemia can exist without seropositivity, making serological screening an uncertain predictor of infectivity.

The authors of this multi-institutional study (see reference) completed a prospective cohort study using donors from New York (13,688), Minnesota (4,583), and New Mexico (8,451), an unaffected region. Donors (37) with a repeat reactive EIA were then evaluated in a 12 month study that included seroreactivity, an immunofluorescent assay, PCR, blood smear and clinical questionnaire. Twenty of the 37 donors completed the 12 month study.

Of those twenty donors, 15 (75%) were still seroreactive. Of the 9 PCR-positive donors, 5 went on to participate in the study; 3 were positive at 6 months and 2 were positive at 1 year. Most of the repeat seroreactive donors had low levels of detectable antibody that were either stable or waning during follow-up. These data are consistent with an American Red Cross Blood Services study from 2015 involving 262 seroreactive or PCR-positive donors in which seroreactivity persisted past 1 year in 81% while PCR positivity was lost in 93%. The authors feel that their data, and that of others, support current policy of indefinite deferral for those with a positive test or positive history of disease, until such time as a reliable and effective test for re-entry becomes available.


Red cell distributions around the world in an era of Patient Blood Management

Much has been written about patient blood management (PBM), focusing on the appropriate use of red cells in various clinical situations by way of randomized clinical trials (RCTs). They show that most patients do not achieve benefit from a more liberal transfusion strategy. More restrictive strategies use less blood and cause fewer untoward events in patients receiving red cell transfusions. Physicians from the United States, Australia, Ireland, Canada, Wales, the United Kingdom, Israel and Japan, representing the “BEST” Collaborative, recently evaluated changes in blood center red cell distributions around the more developed world. There were 7 American blood centers and 8 national blood services represented. From fiscal year 2010 through fiscal year 2014, the number of RBC distributions in the U.S. dropped by 16.9%, and those of the other national programs together declined 8.0%.

These data represent distributions to hospitals, not actual transfusions, but it’s fair to say there are definitely analogous changes in transfusion, which are borne out in other studies. In another article in this edition (see “Declining collections and transfusion of blood” page 2) we note that both an AABB study group, as well as the Department of Health and Human Services, have reported on trends in U.S. in blood collection and utilization. This current article on PBM adds an international aspect to the growing body of information concerning these issues.

To most observers, the U.S. has lagged behind in developing a system that reduces the rate of blood component transfusion, perhaps because most other countries have a stronger governmental hand in data collection and regulation. Although there has been a marked decrease in distributions, the proportion of O-, and to some degree O+ red cell distributions, actually increased, although overall numbers decreased over the study period, which was from FY 2010 to 2014. This may be related to an increased use of massive transfusion protocols, which rely on having more group O blood available for un-crossmatched emergent use. In addition, distributions of B+ and AB+ units declined over the same period in all countries save Japan, where they have had long-standing programs to reduce the utilization of red cells.

Publications Corner

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