



ILLINOIS ASSOCIATION OF BLOOD BANKS

**The Phyllis Unger
Annual Case Studies Meeting
Thursday, February 15, 2018**



This case studies meeting is dedicated to Phyllis Unger in memory of all the wonderful case studies she presented to students, technicians, technologists, residents, fellows and physicians. Phyllis was a dedicated blood banker who spent much of her time teaching and educating whether she was at Michael Reese, University of Illinois or LifeSource. She was always willing to answer questions or test a sample if you sent it to her. She inspired many blood bankers and gave them the desire to look further into a problem. Phyllis was the first Medical Technologist to be President of the ILABB. Prior to this only a physician could hold the office. She wrote many papers and contributed to a few books including “Blood Group System: MN and Gerbich.” She had many things outside of blood banking that she enjoyed as well such as travel, music and bridge. Even these things helped give her blood banking perspective. She was known for never saying anything bad about anyone and always finding the best in them. We hope that this annual meeting will serve as a lasting memory to the knowledge she shared with all who came in contact with her over the years. Thank you Phyllis.

Agenda:

Social hour: 6-7pm

Presentations will begin promptly at 7pm.

1. HIPPA vs the Need to Know
J. Perkins
2. HU5F9-G4 monoclonal Anti-CD47 therapy: A First Experience with Interference in Antibody identification
C. Howard-Menck and J. Crane
3. Wide Variability in the Effect of Anti-CD47 Agents on Pre-transfusion Testing
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4. A novel X-chromosome deletion detected in two male half-siblings with X-linked chronic granulomatous disease and the McLeod phenotype
J. Zinni, P. DeChristopher, M. Saint Martin, R. Lapadat, K. Lannert, J. Johnsen, B. Patel
5. A Novel Type of Glycophorin A-Null Variant in a Rediscovered Patient with Anti-Ena
P. Lindholm, J. Drouillard, K. Hartman, S. Vege, C. Lomas-Francis, C.M. Westhoff, AJ Carterson, G. Ramsey
6. Catastrophic Antiphospholipid Syndrome (CAPS) and Treatment with Therapeutic Plasma Exchange (TPE)
P. DeChristopher, M. Saint Martin, D. Zaporowski, N. Pistryek, M. Miernik
7. Treatment of Rh D Alloimmunization in Pregnancy with Therapeutic Plasma Exchange and IVIgG: Two Cases at an Academic Center
D. Allison, J. Crane, S. Campbell-Lee, V. Vidanovic
8. Improving the Massive Transfusion Protocol using Lean Methodology and High-fidelity Simulation
A. King, R. Burgin, G. Wool

HIPPA vs the Need to Know

Jim Perkins, MD

NorthShore University HealthSystem Blood Banks

Case: An 82 year old man was admitted to the hospital for a Trans-Apical Valve Replacement (TAVR) for aortic stenosis causing heart failure with an ejection fraction of 38%. He had a history of hypertension as well as type II diabetes with lower extremity microvascular disease complicated by multiple episodes of cellulitis. More significantly, the patient had myelodysplastic syndrome rendering him partially transfusion dependent with a requirement of 2 to 3 units of RBCs a month. On admission his hgb/hct was 8.4/25.1 and his platelet count was 83,000.

A type-and-screen received on admission (the day of surgery) revealed him to be group A positive. His antibody screen was positive as was his DAT with polyspecific AHG and anti-IgG. Antibody identification demonstrated clear-cut anti-K, and an eluate was non-reactive. Also detected was a weak antibody thought to be of anti-HLA specificity based on the fact that it "adsorbed away" with Human Platelet Concentrate (HPC). Common, clinically significant antibodies other than anti-K were all ruled out. Of note, however, both cells reacting with the putative anti-HLA expressed Jk^a in a "double dose".

The patient's TAVR went relatively well, but on the late afternoon of the first post-operative day (Friday) the patient's hemoglobin was trending downward (H/H = 7.6/22.6), and the service was considering transfusion. In view of the somewhat complicated immunohematologic circumstances I contacted the blood bank at the patient's primary hospital, where he received his transfusions, was regarding any previously-demonstrated antibodies that might not be completely "showing" at this time.

The blood bank at the patient's hospital refused to provide any information regarding his past history citing "hospital policy".

Discussion: The Health Insurance Portability and Privacy act (HIPPA) was enacted by congress in 1996 to regulate the then-growing importance of information technology in healthcare and the consequent electronic transactions which included patient medical information. Multiple regulatory "rules" were introduced under HIPPA by the department of Health and Human Services (HHS) over the years, but the rule most identified with HIPPA is the Privacy Rule, which regulates disclosure of "individually identifiable health information". An individual may authorize disclosure of her health information, and certain disclosures may be required without authorization, such as those needed by HHS for compliance investigations or enforcement actions. Other disclosures are *permitted* without authorization but not required. These permitted disclosures include those for the purposes of **Treatment, Payment, and Operations (TPO)**, and patients being admitted to the hospital are typically informed of, or asked to consent to, such disclosures on entering a hospital. In choosing to implement permitted disclosures we may "rely on professional ethics and best judgments in deciding which of these permissive uses and disclosures to make."

It is the opinion of the author that disclosure of identified patient information regarding the past history of transfusion and immunohematologic information is legal, ethical, and typically in the best interest of the patient.

**HU5F9-G4 monoclonal Anti-CD47 therapy:
A First Experience with Interference in Antibody identification**

C. Howard-Menk, J. Crane
LifeSource Reference Lab

Background: Hu5F9 is a human monoclonal IgG4 antibody targeting CD47 for the treatment of B-cell lymphomas and some solid tumors. CD47 has been identified as a cell surface protein which regulates phagocytosis and is broadly expressed, including in red cells.

Case report: Our patient is a 58 year old female with a history of B-cell lymphoma, status post peripheral stem cell transplant from a sibling, admitted to the hospital with a 7.0 g/dL hemoglobin. Patient had a previous history of a cold and warm autoantibody. A panagglutinin using gel technology was detected at the hospital and the sample was referred to LifeSource for a complete workup. Transfusion history and medications were not readily available at the time of initial testing.

Initial testing with gel showed a reverse ABO discrepancy, a panagglutinin, and a negative DAT. Tube testing using PeG, LISS, and enzymes was selected to continue the identification. Strong agglutination (3-4+) strength was seen at IS and AGT using all reagents.

Patient plasma was tested against cells treated with Ficin, DTT, glycine acid-EDTA, cord cells, aged cells, and a battery of rare cells based on patient race and antibody reactivity. All reactions were 3-4+ against all cells tested. Though the patient DAT was negative, an eluate was performed to determine if there was antibody specificity due to autoantibody. It demonstrated a panagglutinin. Titration of the plasma versus a phenotypically matched cell showed reactivity greater than 4096. An alloabsorption using human stroma (ficin treated) was performed and the panagglutinin was removed to the point that the ABO discrepancy could be resolved. Four cold/allo absorptions removed all reactivity at IS. Reactivity remained when tested at 37/AGT. Additional alloabsorptions performed at 37C (x4) removed the remaining activity when tested with IgG Coombs.

The above results suggest a potent antibody to a high frequency antigen. Many tests and several rare phenotypes of cells were tested along these lines. When complete history was obtained, it was determined that the patient had been in a clinical trial for a monoclonal antibody therapy: anti-CD47. A literature review of the antibody identified a specific IgG Coombs sera clone is required to resolve the reactivity. If a clone without anti-IgG4 is used (Gamma clone, monoclonal), the cells which previously reacted 3-4+ with a clone containing IgG4 no longer react. Antibody testing was successfully completed using Gamma clone anti-IgG sera at AGT, omitting the IS and 37C phase. ABO testing was completed with x4 cold alloabsorbed stroma.

Conclusion: Additional monoclonal therapies will continue to emerge. This case highlights the need for extensive communication between the Blood Bank and the clinical service regarding the use of novel monoclonal antibody treatment.

Wide Variability in the Effect of Anti-CD47 Agents on Pre-transfusion Testing

Zhen Wei Mei, Geoffrey Wool
University of Chicago
Department of Pathology

Background: CD47 is a cell surface transmembrane molecule that is expressed universally on all human cells. In addition to its roles in angiogenesis, fibrosis, cell adhesion, and migration, it also interacts with a molecule found on the surface of myeloid cells, signal-regulatory protein alpha (SIRP α). This interaction allows cells expressing CD47 to modulate, and in this case, inhibit, the phagocytic activity of the macrophages expressing SIRP α . As cells age, the level of CD47 being expressed is reduced, thereby allowing macrophages to recognize and eliminate senescent cells. Tumor cells, however, overexpress CD47 to overcome this mechanism of cell death; anti-CD47 therapy removes this inhibition by allowing macrophages to resume their phagocytic attack on tumor cells. Multiple anti-CD47 agents are now in clinical trials. CD47 is expressed normal red cell membranes.

Case Report: Within our institution, there are two clinical trials for anti-CD47 agents: Hu5F9-G4 (Forty Seven, Inc.) for lymphoid malignancy, and CC-90002 (Celgene) for myeloid malignancy.

Hu5F9-G4 is an IgG4 humanized anti-CD47 antibody; CC-90002 is a humanized CD47-blocking antibody. Though both utilize a similar mechanism, differences exist between these two therapeutic agents.

Initial screen and panel testing was performed in gel. If panagglutinins detected, additional tube testing (PEG or LISS) was performed. Initial DAT was performed in gel. DAT were routinely ordered on CC-90002 patients as part of trial protocol.

Seven total patients are described (Table). All had negative antibody screens before starting anti-CD47 agent. In the four CC-90002 patients, the general pattern is a negative antibody screen with a strongly positive DAT with a panagglutinin eluted. In the three Hu5F9-G4 patients, the pattern is a panagglutinin in the plasma with a less strongly positive DAT. All plasma reactivity is eliminated when using the Immucor AHG which does not bind IgG4.

Conclusion: CC-90002 generally does not affect antibody screen testing, while plasma from patients receiving Hu5F9-G4 shows significant interference. Since most antibody screen are negative for those receiving CC-90002, the standard blood bank testing protocols are followed. However, those receiving Hu5F9-G4 must have significant antibodies ruled out using an alternate reagent, Immucor AHG, before appropriate blood products may be issued. Performing timely pretransfusion testing and providing appropriate blood products for patients receiving anti-CD47 agents is strongly dependent on the particular anti-CD47 agent used. As always, rapid and clear communication with the clinical services is necessary for optimal patient care.

A novel X-chromosome deletion detected in two male half-siblings with X-linked chronic granulomatous disease and the McLeod phenotype

Authors: Jules G. Zinni, MLS(ASCP)MB1, Phillip J. DeChristopher, MD, PhD1, Marisa C. Saint Martin, MD1, Razvan Lapadat, MD1, Kerry W. Lannert, MT(ASCP)2, Jill M. Johnsen, MD2,3, and Bhakti Patel, MLS(ASCP)SBB4

1Loyola University Health System, 2Bloodworks Northwest Research Institute, Seattle, WA, 3Department of Medicine, University of Washington, Seattle, WA, 4American Red Cross IRL, Chicago, IL

Background: Genetic variation involving the Xp21.1 region has been associated with X-linked chronic granulomatous disease (CGD). In X-linked CGD, deleterious variants affecting the *CYBB* gene encoding cytochrome b-245 beta polypeptide result in loss of NADPH functionality, leading to loss of the neutrophil respiratory burst needed to kill phagocytized pathogens. The *XK* gene that encodes the Kx transmembrane protein on rbc's lies near *CYBB* on the X-chromosome. Absence of the Kx protein reduces expression of the Kell system antigens so that transfused Kell antigens appear foreign.

Case Report: Two black male half-brothers, ages 9 and 18, were identified to have phenotypes consistent with McLeod. Both brothers had been hospitalized with recurrent pulmonary and other infections. The 18 year old, diagnosed with CGD and a previous recipient of granulocyte transfusions for a chronic fungal pneumonia. Anti-Kx was identified 3 months after these index transfusions. The antibody screen of the 9-y/o brother has remained negative despite two RBC transfusions at age two. Whereas both siblings clinically have CGD and the McLeod phenotype, it remains unclear if either has the complete McLeod Syndrome.

We analyzed the serological and genetic characteristics of these patients to clarify a more complete picture of the basis of their disorders.

Results / Methods: A fresh sample from the older brother was tested with a selected cell panel, revealing the presence of a panagglutinin reactive with all but autologous red cells tested. An extended high frequency panel using 0.2 M DTT treated cells was positive against the patient's plasma, supporting an anti-Kx. The polyspecific direct antiglobulin test was negative. Cellano and Kpb phenotyping were found to be weakly reactive. The antibody was nonreactive with four McLeod phenotype reagent red cells. All other common clinically significant alloantibodies were excluded at PEG/IAT using alloadsorbed plasma.

A regional tiled genomic targeted PCR strategy was used to identify deletions affecting Xp21.1 using a screen comprised of sixteen genomic PCRs spanning a 2.8mbp region containing *CYBB* and *XK*. Amplicons were visualized by agarose gel electrophoresis. In these samples, contiguous amplicons failed to amplify consistent with a deletion beginning in *PRRG1* and ending in *SYTL5*. Custom fine mapping primers confirmed the location of the predicted deletion, identifying a ~570kb X-chromosome deletion affecting at least the genes *PRRG1*, *LANCL3*, *XK*, *CYBB*, *DYNLT3*, and *SYTL5*.

Conclusion: We studied two half-brothers with both the McLeod phenotype and CGD and identified a previously undescribed X-chromosome deletion impacting at least 6 genes including

XK and *CYBB*. Evaluation of patients with X-linked CGD or McLeod phenotype should consider evaluation for the presence of the other syndrome. Genetic evaluation should include a method capable of identifying the extent of X-chromosome deletions. Transfusion exposures should be minimized whenever possible in these cases due the risk of provoking or exacerbating anti-Kx and anti-Kell system responses.

A Novel Type of Glycophorin A-Null Variant in a Rediscovered Patient with Anti-En(a)

Authors: P. Lindholm, J. Drouillard, K. Hartman, S. Vege, C. Lomas-Francis, CM Westhoff, AJ Carterson, G. Ramsey
Northwestern University, Northwestern Memorial Hospital and Heartland Blood Centers, Chicago and Aurora, IL, and New York Blood Center, New York, NY

Background: In the En(a⁻) phenotype, RBCs lack normal expression of glycophorin (GP) A protein carrying M/N antigens.¹ The term anti-Ena encompasses various antibody specificities occurring in persons with absent or modified GYPA genes and reacting to high-prevalence GPA antigens.² Compatible En(a⁻) or MkMk (absent GPA and GPB) RBC units compatible with anti-Ena antibodies are extremely rare.

Methods and Case Report: A type-and-screen was requested on a 59-yr-old Pakistani-born woman with diabetes and remote history of broad RBC crossmatch incompatibility in obstetrical testing. She was group B, D+ with plasma pan-reactivity in testing by solid-phase (4+), polyethylene glycol (3+ anti-IgG), and low-ionic saline solution (LISS) (1+ 37C, 3+ anti-IgG) methods, and with ficin-treated RBCs (2+ 37C, 3+ anti-IgG). Direct antiglobulin testing and autocontrols were negative. Her RBCs were serologically phenotyped as M-N-S-s+, En(a⁻), Wr(a-b⁻) and her plasma was nonreactive to En(a⁻) RBCs. Her antibody reactivity was consistent with anti-EnaFR (ficin-resistant). DNA-based PreciseType HEA BeadChip (Immucor), which targets GYPA c.59C>T for M/N, predicted her phenotype as M+N-. Her RBCs were reactive with Glycine soja, indicating reduced levels of sialic acid which is consistent with loss or change of the glycophorins. She was advised to donate autologous RBCs to freeze in case of future need and has donated 1 unit to date. She has no US relatives for RBC testing.

One of two published En(a⁻) cases in the US was a Pakistani-born woman reported from Cook County Hospital in 1993-95.^{3,4} That patient's initials, demographics, clinical history, serologic RBC phenotypes and anti-EnaFR reactivity matched our patient. No GPA was detected in her RBCs by immunoblotting or with monoclonal anti-GPA. She was presumed to have the 'Finnish' En(a⁻) phenotype with deletion of GYPA exons 2-7, although no DNA testing was done then. Detection of c.59C>T by HEA ruled out deletion of GYPA exon 2 as the molecular basis. Long-range PCR 5 and sequencing of GYPA exons 2-7 was performed and confirmed a GYPA *M/M background and homozygosity for a novel indel (insertion/deletion) variant. In the gene region coding for the transmembrane helical domain,⁶ a guanine nucleotide was inserted at position c.314-315 in exon 5, which causes a frameshift in the protein and a stop codon 19 amino acids downstream [c.314_315insG (p.Thr106Asnfs*19)]. This GYPA mutation has not been previously reported.

Discussion: Ena antibodies have caused mild to fatal hemolytic transfusion reactions and severe fetal hemolysis.^{1,2} In recently summarized rare-donor registries in 20 countries including the US, the only reported En(a-) or Mkmk RBC donors were in Canada and Japan.⁷ The Wr(a-b-) phenotype (Diego blood group, band 3 protein) and formation of anti-Wrb alongside anti-Ena in some patients with GPA-deficient RBCs is attributed to disruption of the GPA-band 3 RBC membrane protein complex. Previously described En(a-) cases have had either deletion of GYPA exons 2-7 or GP(A-B) hybrid proteins ('UK' type).¹ This patient is the first reported case of an indel glycophorin A-null variant. Modern-day molecular characterization of this unusual patient's GYPA genes has advanced our knowledge of the MNS system 25 years after she was first reported.

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Catastrophic Antiphospholipid Syndrome (CAPS) and Treatment with Therapeutic Plasma Exchange (TPE)

Authors: Phillip J. DeChristopher, MD, PhD, Marisa C. Saint Martin, MD, Danuta Zaporowski, RN, Nataliya Pistryek, BSN, RN, Magda Miernik, BSN, RN, CMSRN
Loyola University Health System, Maywood, IL 60153

BACKGROUND: Antiphospholipid syndrome (APS) is characterized by thromboses and/or pregnancy morbidity associated with persistently positive antiphospholipid antibodies (aPL). Uncommonly, a small percentage of APS patients develop a potentially lethal complication known as catastrophic APS (CAPS). The low incidence of CAPS renders prospective or controlled studies unavailable, making optimal treatment uncertain. Early treatment is vital for survival with combinations of anticoagulation, corticosteroids and TPE being recommended. Even with this approach, the mortality rate remains high (30 – 50%).

CASE REPORT: A 35-y/o woman, with a 15-year h/o APS associated and at least 2 prior pregnancy losses, was transferred to our care. She had 1 term delivery 3 years PTA, but currently was 10 days postpartum from term delivering of twin boys. There was no evidence of preeclampsia or HELLP Syndrome. 4 days postpartum, she developed an acute DVT below the right knee. Serology was positive for 3 aPLs: anticardiolipin (aCL, 48.3 GPL), β -2-microglobulin (4.9 mg/L) and anti- β -2-glycoprotein I (IgG, 37 SGU). Heparin anticoagulation, placement of an IVC filter and TPE was instituted for 3 days. Progressive active hemolysis and severe thrombocytopenia raised concern for CAPS, prompting transfer. On admission to LUMC,

she had transaminitis, worsening renal injury (increasing creatinine, decreasing GFR, proteinuria), MAHA (schistocytosis, LDH 1478 IU/L, ahaptoglobinemia) and severe thrombocytopenia (22 K / μ L). She was treated with IV methylprednisolone, anticoagulated with IV bivalirudin and intensive TPE continued. For 11 consecutive days, we performed daily, 1.5-plasma-volume TPEs, replaced with thawed plasma, interrupted by dose #1 of rituximab. Six daily TPEs were continued until rituximab dose # 2 was given. Subsequently, TPE were continued every second or third day, with hiatuses for the next 2 doses of rituximab. After 19 TPE's, we tapered the volume exchanged, the proportion of plasma replaced and the frequency of TPE's. With normalizing laboratory parameters (drop in LDH, rise in platelet counts, detectable haptoglobins, and reticulocytosis), seven tapering TPEs were continued as an outpatient. We performed 26 TPEs (29, total, including 3 done at OSH). Steroids were tapered and anticoagulation transitioned from bivalirudin to heparin to oral Coumadin. Outpatient therapy continues with prednisone, Coumadin and hydroxychloroquine. Absent further thrombotic events, she has normal LFT's and normal hematologic parameters, however she suffered renal injury. She has Stage 3 CKD with BUN / Cre / Est. GFR at 28 mg/dL, 1.8 mg/dL and 32 mL / min / 1.73 me², respectively.

CONCLUSION: We present a case of a 35-y/o female with acute, postpartum "*triple-antibody-positive*" CAPS, complicated by MAHA and severe thrombocytopenia, thrombosis, hepatic and renal involvement. TPE for CAPS has never been investigated in a randomized trial, but observational data suggest that it improves survival; the international, retrospective CAPS Registry shows that the highest rate of recovery (78%) was achieved by the combination of anticoagulation, glucocorticoids, and TPE. Aggressive therapy with steroids, anticoagulation, intensive TPE and rituximab achieved a stable remission in this patient, but her renal function was remains compromised (Stage 3 Chronic Kidney Disease).

Treatment of Rh D Alloimmunization in Pregnancy with Therapeutic Plasma Exchange and IVIgG: Two Cases at an Academic Center

Allison D, Crane J, Campbell-Lee S, Vidanovic V

Transfusion Medicine, Department of Pathology, University of Illinois at Chicago

Background: Hemolytic disease of the fetus and newborn (HDFN) can occur when an Rh D-negative female with an anti-D alloantibody is pregnant with an Rh D-positive fetus. Maternal sensitization often occurs during a previous pregnancy with an Rh D positive fetus after maternal-fetal hemorrhage, but can occur after transfusion of red blood cells expressing Rh D. The effects of HDFN are catastrophic, including fetal demise, fetal hydrops, and kernicterus. The mainstay of treatment for fetal anemia to prevent fetal hydrops is intrauterine transfusion (IUT). Transcranial middle cerebral artery (MCA) ultrasounds are the gold standard test for fetal anemia. MCA measurements more than 1.5 multiples of the median (MoM) predict moderate – severe fetal anemia, and IUT is indicated. IUT is dangerous before 20 weeks gestation age (WGA), and therapeutic plasma exchange (TPE) and IVIgG can be employed to delay the onset of fetal anemia until IUT can be safely performed.

Case Report: Two Rh-alloimmunized pregnant patients are described. The first patient was a 28 year old female, G4P1112, with an initial anti-D titer of 1:128 in her first trimester. TPE three times per week, and IVIgG twice per week were initiated at 20 WGA. Her prenatal course was complicated by a central line infection requiring line replacement. She also developed a

pulmonary embolus and was anticoagulated with enoxaparin. TPE and IVIgG continued until 37 WGA, when the patient underwent scheduled caesarean delivery of a viable female. During pregnancy, the patient's anti-D titers ranged from 1:32 to 1:512. Her MCA velocities peaked at 1.22 MoM, and the infant was never transfused.

The second patient was a 28 year old female, G5P2, with anti-D and anti-C alloantibodies. The initial anti-D titer was 1:2048, and anti-C was undetectable. TPE and IVIgG were initiated at 11 WGA, and TPE continued until 25 WGA. The patient's anti-D titers ranged from 1:256 to 1:2048. The fetus received four IUT's due to rising MCA velocities, the first at 22 6/7 WGA. The patient underwent urgent caesarean delivery of a viable male at 30 WGA.

Conclusion:

Due to the widespread use of prophylactic Rh immune globulin doses in pregnant women who are Rh D-negative, the incidence of HDFN has decreased dramatically. As a result, the recent literature addressing management of pregnant women with Rh alloimmunization and HDFN consists of case reports and small case series. Published treatment regimens utilize TPE and IVIgG not to prevent HDFN completely, but to delay IUT as long as possible, particularly until after 20 WGA, before which IUT is too risky to attempt. Our experience treating these two patients is concordant with the recent published case reports, and supports TPE and IVIgG as a safe and effective adjunctive therapy in Rh alloimmunized pregnant women.

Improving the Massive Transfusion Protocol using Lean Methodology and High-fidelity Simulation

Ariana King, MT, MPH¹, Robert Burgin², Geoffrey Wool, MD, PhD³.

University of Chicago Medicine. 1) Laboratory Administration; 2) Simulation Center; 3)
Department of Pathology

Background: The Massive Transfusion Protocol (MTP) is a standardized process which ensures safe and timely delivery of a large quantity of blood products for hemorrhaging patients. At University of Chicago Medicine (UCM), all blood products were delivered to the care team through the pneumatic tube system (PTS), including MTP blood product packs. Each adult MTP pack contains six RBCs, four FFP, and one plateletpheresis. A keep-ahead MTP pack is immediately allocated for each patient after issuing an MTP pack to minimize processing delays for ongoing hemorrhages. Transport through the PTS resulted in occasional delays due to planned and unexpected PTS downtimes, and plastic carriers being re-routed, stuck, or overlooked by the care team.

In 2016, the Blood Bank began stocking several pre-made MTP packs to minimize the turn-around-time from MTP activation to issue of blood products. However, electronic allocation and labeling beyond pre-made packs required additional processing, resulting in extended turn-around times for subsequent MTP packs.

Case Report: Hospital leadership agreed to transition to coolers for MTP pack transport with a member of the care team serving as the runner. Each unit identified a preferred primary and

secondary runner along with the hospital directive that all staff must transport MTP packs if necessary. Walking in-services were provided to staff in all critical care areas, which included a tour to the Blood Bank. Large coolers and insulated pouches were purchased and validated to store one adult MTP pack.

Value stream mapping was completed to redesign Blood Bank processes. The entire process was mapped through observation, then scrutinized to identify non-value add steps (waste). Non-value add steps were removed and critical tasks were redesigned to improve efficiency.

High-fidelity simulations were carried out in several adult critical care units; additional simulations were completed in pediatric critical care units. The artificial patient was a computer-controlled high-fidelity mannequin who exhibited vital signs, sounds, and allowed IV establishment. The patient was registered in the EMR, allowing providers to place transfusion orders. Blood Bank staff prepared several MTP packs with simulated blood products and followed the redesigned process. A care team runner transported each pack from the Blood Bank. Several clinical and Blood Bank leaders observed and timed the process, recording deviations and successes. Each simulation was concluded with a debrief including all participants. Several clinical and Blood Bank processes were modified based on simulation observations. Observation data will also be used to quantify the impact of Blood Bank improvements.

Conclusion: Simulation is an excellent methodology for testing and improving a new or existing process. Simulation is superior to tools such as FMEA that rely on staff recollection. Our group was able to test high-risk scenarios such as transporting a patient with an active MTP without any risk or exposure to our patients.

Participant feedback from scenarios indicated a strong positive reception to the new process, and improved learning and comfort with the new process. Planning and carrying out full simulations is resource intensive, but it will generate immense savings in improved patient safety and efficiency.

NOTES