Review article

Intraoperative pediatric blood transfusion therapy: a review of common issues. Part I: hematologic and physiologic differences from adults; metabolic and infectious risks

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Summary
Pediatric intraoperative transfusion therapy, particularly the approach to massive blood transfusion (blood loss ≥ one blood volume) can be quite complex because of the unique relationship between the patient’s blood volume and the volume of the individual blood product transfused. This paper is divided into two parts: part 1 presents an overview of the physiologic and hematologic differences between children and adults as well as an overview of the metabolic consequences of blood transfusions, risks of disease transmission, and blood compatibility issues.

Keywords: transfusion therapy; massive blood transfusion; hypocalcemia; hypomagnesemia; hyperkalemia; hypothermia; disease transmission

Hematologic and physiologic differences from adults

The physiologic and hematologic differences between adults and children dictate different guidelines regarding perioperative blood transfusions. The threshold to transfuse a child may be altered secondary to these physiologic considerations. Although many of the complications associated with pediatric blood transfusions are similar to those encountered in adults, some complications occur more readily and with a greater frequency in children. Familiarity with the differences between adult and pediatric transfusion management is of utmost importance when planning transfusion strategies.

Children have higher oxygen consumption and a higher cardiac output to blood volume ratio than adults (1,2). The neonatal myocardium operates at near maximum level of performance as a baseline. Therefore, the newborn’s heart may be unable to compensate for a decreased oxygen carrying capacity by increasing cardiac output. The neonatal myocardium will also suffer a greater degree of decompensation when exposed to decreased oxygen delivery; intraoperative cardiac ischemia has been
reported in neonates (3). Therefore the optimal hemoglobin values in the newborn are generally higher than those of older patients. The normal term neonate has hemoglobin values in the 14–20 g·dl⁻¹ range depending in part upon how long it takes to clamp the umbilical cord. These elevated hemoglobin levels gradually decrease over the first several months of life because of decreased erythropoietin and reduced red blood cell half-life such that the physiologic nadir for hemoglobin occurs at approximately 2–3 months of age (Table 1). Term infants with hemoglobin levels <9 g·dl⁻¹ and preterm infants with hemoglobin values <7 g·dl⁻¹ at the time of this nadir should be investigated for the presence of hemoglobinopathy or other pathology. If concern over adequate oxygen carrying capacity exists, an elective procedure may be postponed pending evaluation and treatment. Raising the hemoglobin level may be accomplished by transfusion, exogenous erythropoietin administration, or simply postponing the case until the child’s natural hematopoietic mechanisms have taken effect. Higher hemoglobin levels will increase oxygen carrying capacity and in the premature infant, may protect from postanesthetic apnea of prematurity although transfusion for this purpose alone is not generally indicated (4). Transfusions are usually withheld unless clinically important blood loss is likely to occur.

Fetal hemoglobin (HbF) comprises 70% of full term and 97% of premature infants’ total hemoglobin at birth (5). Red blood cells (RBCs) containing HbF have a shorter life span (90 days) than those containing primarily adult hemoglobin (HbA) (120 days). HbF interacts poorly with 2–3-diphosphoglycerate (2–3 DPG). Therefore the P₅₀ (the partial pressure of oxygen at which hemoglobin is 50% saturated) decreases from 26 mmHg with HbA to 19 mmHg with HbF. This leftward displacement of the oxygen–hemoglobin dissociation curve results in decreased oxygen delivery to tissue because of the high affinity of HbF for oxygen. HbF production diminishes during the first few months of life until only a trace is present at 6 months of age (Fig. 1) (5). In clinical terms, the younger the infant, the higher the fraction of HbF and thus the lower the oxygen carrying capacity. Premature infants have higher percentages of HbF than their full-term counterparts and decreased erythropoietin production which inhibits them from responding to anemia appropriately (6). Another concern in all neonates is that they may have a decreased ability to oxygenate blood because of lung disease or congenital heart disease. It is for these reasons that hemoglobin levels that are adequate for the older patient may be suboptimal in the younger infant or neonate. The threshold for transfusing RBCs to a neonate should be at a higher hemoglobin trigger than an older child or healthy adult. In the operating room the decision to initiate RBC transfusion is based upon a constellation of factors such as the rapidity of the blood loss, the presence of impaired oxygenation (pulmonary or cardiac in origin), and the general medical condition of the patient.

**Table 1**

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Full term (g·dl⁻¹ of blood)</th>
<th>Premature (g·dl⁻¹ of blood)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth</td>
<td>19.3</td>
<td>Slightly less than full term</td>
</tr>
<tr>
<td>0.5 months</td>
<td>16.6</td>
<td>15.4</td>
</tr>
<tr>
<td>1 month</td>
<td>13.9</td>
<td>11.6</td>
</tr>
<tr>
<td>Age at hemoglobin nadir</td>
<td>9–12 weeks</td>
<td>6–10 weeks</td>
</tr>
<tr>
<td>Mean hemoglobin at nadir</td>
<td>11.2</td>
<td>9.4</td>
</tr>
<tr>
<td>4 months</td>
<td>12.2</td>
<td>11.7</td>
</tr>
<tr>
<td>6 months</td>
<td>12.5</td>
<td>12.4</td>
</tr>
</tbody>
</table>

Modified with permission from Barcelona & Coté (10).

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**Metabolic consequences and infectious disease risks of transfusion therapy**

The anesthesiologist must be well versed in the infectious disease risks and metabolic consequences
of blood transfusions. The common metabolic consequences of transfusion include: hypocalcemia, hyperkalemia, hypomagnesemia, hypothermia, changes in acid–base status, and shifts in the oxygen–hemoglobin dissociation curve. These metabolic disturbances may occur to a greater degree in children than with adults because of the larger transfusion to blood volume ratio. For example, a unit of whole blood may represent the entire circulating blood volume of an infant whereas that same unit of blood transfused into an adult represents only one-tenth of the circulating blood volume.

**Metabolic implications**

**Hypocalcemia**

Ionized calcium is essential for the successful initiation of the coagulation cascade (7). Citrate is present in stored blood as a chelating agent for calcium to prevent clot formation. However, upon transfusion, the deleterious effect of citrate is in vivo ionized hypocalcemia. All blood products contain some citrate but the resulting degree of ionized hypocalcemia depends upon the blood product transfused, the rate of transfusion, and hepatic blood flow/function (8–11). Hypocalcemia is most often seen during transfusion of fresh frozen plasma (FFP) and whole blood because these components contain the greatest concentration of citrate per unit volume. Patients with hepatic dysfunction and neonates are at high risk secondary to their decreased ability to metabolize citrate (9–11). As the neonate’s heart has reduced sarcoplasmic reticulum, it is greatly dependent on ionized calcium (calcium flux) for normal contraction and relaxation. Therefore the neonatal heart is particularly vulnerable to myocardial dysfunction in the presence of citrate-induced ionized hypocalcemia. This is further confounded by the concomitant administration of potent inhalational anesthetic agents because all of the potent inhalational agents cause myocardial depression secondary to blocking of calcium channels (Figure 2) (9,12–14).

Severe cardiac dysfunction may be prevented by slowing the rate of infusion of citrate containing products to <1 ml·kg⁻¹·min⁻¹, prophylactically infusing calcium during the transfusion, and by reducing the concentration of inhaled potent anesthetic agent. If a patient becomes hemodynamically unstable during rapid blood transfusion, and the circulating blood volume has been maintained, then ionized hypocalcemia is the most likely cause of the hypotension. The treatment for ionized hypocalcemia is administration of exogenous calcium. There is no difference in the rate of ionization or the change in blood ionized calcium concentrations if equivalent doses of calcium chloride vs calcium gluconate are administered. Generally three times more calcium gluconate is required than calcium chloride, e.g. 2.5 mg·kg⁻¹ calcium chloride = 7.5 mg·kg⁻¹ calcium gluconate (Figure 3) (15). Calcium should not be administered in the blood container or even in the same intravenous line because this will rapidly allow clot formation. A reasonable starting dose for calcium chloride is 5–10 mg·kg⁻¹ or 15–30 mg·kg⁻¹ for calcium gluconate to treat ionized hypocalcemia during rapid infusions of FFP or whole blood (>1 ml·kg⁻¹·min⁻¹). Alternatively, with blood loss that is moderate but continuing over an extended period of time, e.g. redo liver transplantation, a baseline infusion of calcium particularly during the anhepatic phase may be useful. Our practice in this situation is to begin calcium

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**Figure 2**

The effects of an infusion of citrate on ionized calcium and mean arterial pressure (MAP) in a dog model. Note that with the higher expired concentration of halothane there is a greater fall in ionized calcium and MAP. This suggests that the calcium channel blocking properties of inhalation agents are potentiated by the reduced ionized calcium while at the same time the reduced MAP decreases citrate metabolism by reducing hepatic blood flow. Although this study examined halothane, the interaction between ionized hypocalcemia and any potent anesthetic agent would be similar as all potent anesthetics depress cardiac function through calcium channel blocking activity. [Modified from data in Coté (13)].

chloride at 10–15 mg·kg\(^{-1}\)·h\(^{-1}\) with frequent measurement of ionized calcium values and appropriate titration of the infusion to the desired value.

**Hyperkalemia**

Transfusion associated hyperkalemia also occurs during specific conditions in pediatric patients. Neonates and infants have experienced fatal cardiac arrhythmias during large volume or exchange transfusions as a result of hyperkalemia (16–19). The rate of rise of serum potassium values occurs quickly in children with small blood volumes (20). Blood components with the highest levels of potassium include whole blood, irradiated units and units approaching their expiration date (9,10,20–24). Washing of erythrocytes has been shown to markedly reduce the potassium concentration (16,25). As potassium leaks from older RBC as their cell membranes deteriorate, it may be advisable to use fresh or ‘newer’ (i.e. <7-day old) units, and to avoid the use of whole blood when transfusing neonates and small infants (16,20,26). It should be noted however that life-threatening hyperkalemia may occur even with packed RBC if large quantities of blood are rapidly infused (18,20,27). Should a life-threatening arrhythmia such as ventricular tachycardia develop, then higher doses of calcium chloride (15–20 mg·kg\(^{-1}\)) or calcium gluconate (45–60 mg·kg\(^{-1}\)) should be administered at 1–2-min intervals until the arrhythmia is resolved. It should be noted that the calcium therapy only opposes the electrophysiologic effects of hyperkalemia. Other measures to reduce circulating potassium values such as administration of glucose and insulin, hyperventilation, administration of albuterol, and Kayexalate will be needed to correct the hyperkalemia. Standard cardiopulmonary resuscitation (CPR measures) should be initiated as indicated.

**Hypomagnesemia**

Decreased ionized magnesium levels occur with massive blood transfusion. Similar to hypocalcemia, hypomagnesemia is the result of citrate toxicity and is seen with its greatest severity during the anhepatic phase of liver transplantation. Magnesium is essential in stabilizing resting membrane potential and thus is very important in maintaining cardiovascular stability, particularly from an electrophysiologic standpoint (28–30). Should a life-threatening arrhythmia such as ventricular tachycardia or ventricular fibrillation develop that does not respond to exogenous calcium therapy, then intravenous magnesium sulfate (25–50 mg·kg\(^{-1}\)) may be administered followed by an infusion of 30–60 mg·kg\(^{-1}\)·24 h\(^{-1}\).

**Acid–base changes**

The typical unit of donated whole blood contains approximately 450 ml of whole blood and about 50 ml of anticoagulant for a final volume of approximately 500 ml. After donation, the RBC continue to undergo metabolism and can elevate dissolved carbon dioxide to 180–210 mmHg within several hours (31). Once the available oxygen is utilized, the RBC change to anaerobic metabolism thus increasing the lactic acid content. Therefore, the rapid transfusion of whole blood may initially cause a transient combined respiratory and metabolic acidosis. However, the carbon dioxide is rapidly excreted through the lungs and the small amount of lactic acid is generally rapidly buffered so that there is no net effect upon acid–base status (32). Thus there is no need for exogenous bicarbonate therapy as long as the patient is adequately volume resuscitated. The development of a metabolic acidosis during massive blood transfusion generally
indicates inadequate perfusion, severe hypovolemia prior to initiation of transfusion therapy, or an underlying issue such as systemic infection or hypoxemia. In general, patients who experience massive blood transfusion will later develop a metabolic alkalosis secondary to the metabolism of citrate. This is another good reason to limit exogenous sodium bicarbonate therapy to treatment of measured acid–base abnormalities rather than by administering bicarbonate empirically.

**Hypothermia**

Children are predisposed to heat loss because of their large surface area to weight ratio and relatively large head size. The normal shift in heat distribution from core to periphery from general anesthesia, evaporative losses through ventilation and surgical incisions, use of irrigation solutions, and cold operating rooms further contribute to the development of hypothermia (33). Maintenance of normothermia is often challenging even without the administration of cold blood products.

The deleterious effects of hypothermia include apnea, hypoglycemia and decreased drug metabolism which can lead to prolonged effects of anesthesia and delayed emergence (34,35). A drop in patient temperature also leads to a leftward displacement of the oxygen dissociation curve, resulting in decreased oxygen delivery to tissues (31,32). Hypothermia increases oxygen consumption (secondary to shivering and nonshivering thermogenesis), worsens coagulopathy, and may even increase mortality (36–38). The warming of blood products transfused to children may require specific equipment and different techniques than used in adults. When priming any fluid warmer with blood products to be transfused to an infant or small child, it is important to disconnect the system from the patient so as to avoid a concomitant massive infusion of crystalloid. A standard ‘heating plate’ type blood warmer has a large dead space and high resistance to flow caused by the coiled tubing through which the blood must travel while traversing the heating plates. These devices can be used in most situations where blood loss is slow and a syringe can be used to both quantitate the volume transfused and facilitate flow of blood. For patients in whom blood loss is rapid, more sophisticated warming devices are often needed. Recent generations of blood warmers have taken advantage of counter current heat exchange (tube within a tube of hot water). For discussion purposes we will present information on systems available in the USA recognizing that other systems are available in other countries.

The Hot Line fluid warmer (Level-1 Technologies, Rockland, MA, USA) utilizes a circulating warm water bath and a simple system which has much less dead space and resistance to blood flow than heating plate type warmers (39). However, air bubbles have been observed in the transfusate raising concerns regarding the potential for air embolization (40). This system is not designed for rapid blood transfusions.

Level-1 system also uses counter current technology but with an automatic pressurization system that squeezes the blood or crystalloid containers (10,41). However, fatal air embolism has occurred with the use of Level-1 when intravenous fluid bags were not properly evacuated of air (42,43). Level-1 has now developed a new air detection system with further refinements in the air detection and flow cutoff that may be added on to the currently available systems. A recently released model of Level-1 technology is now equipped with an automatic air purging system with an alarm with flow cutoff should air bypass the air eliminator. Level-1 has also developed upgraded disposable blood administration systems (D-100 and D-300) which are capable of more rapid transfusion and superior heating of fluids compared with the original D-50 disposable blood administration system (44).

The Rapid Infusion System (RIS; Haemonetics, Inc., Braintree, MA, USA) has a much greater capacity and is based upon cardiotomy reservoir technology (41,45–47). The RIS has many safety features, including superior air elimination and warming capabilities compared with Level-1, but this device is large, expensive and too powerful for most pediatric cases. In addition, this system is quite difficult to set-up for the occasional user (41). Haemonetics Inc. is no longer producing this machine but will continue to service it until 2005.

The replacement for the RIS is the FMS 2000 (Belttoma Inc., Billerica, MA, USA). It uses magnetic induction for heating of fluids between 10 and 500 ml min⁻¹ (an upgraded system with the capacity to heat up to 750 ml min⁻¹ is in development).
device is smaller and easier to set up than the RIS, has excellent air detection and elimination capabilities, and may provide a middle ground between Level-1 and RIS for bigger pediatric patients. The disadvantage of the FMS for smaller pediatric patients is its inability to heat fluids at flows <10 ml min$^{-1}$. At present the FMS seems to have superior blood warming and air detection compared with Level-1, however a new add-on air detector for the Level-1 will likely correct the air detection problem (48).

Recently Belmont has introduced a new fluid warmer (Buddy) that heats fluids and blood just prior to entering the patient using the same magnetic induction heating method. This system mounts on an i.v. pole but the disposable 3 × 11 cm cartridge and warming unit are interfaced with the patient’s i.v. system close to entry to the patient. This plate is equipped with a pressure regulating valve and a ‘venting membrane’ that the manufacturer purports to ‘automatically remove air from crystalloid solutions’. This heating unit only weighs 105 g. Having the blood warmer so close to the patient eliminates the usual long distance between warmer and patient whereby warmed fluids cool to room temperature prior to entering the patient. The manufacturer states that room temperature crystalloid solutions can be warmed to 38°C at flow rates up to 100 ml min$^{-1}$. This device is not designed to administer blood products under pressure.

Each of these fluid-warming devices has advantages and disadvantages in terms of ease of setup, cost per setup, cost per device, and footprint in the operating room where space can be at a premium (Table 2).

**Infectious disease transmission risk**

Infectious risks of transfusion have decreased dramatically thanks to improved screening, detection of infected units and advances in pathogen inactivation (Table 3). The incidence of infectious complications is now smaller than that of metabolic or immunologic consequences. Further improvements in safety are likely in the future with the more widespread use of viral RNA detection by means of nucleic acid amplification but this will not occur until the cost of this test is markedly reduced (49,50). Nonetheless,
the risk of infection, especially of human immunodeficiency virus and hepatitis C, is often the primary concern of parents whose child may need a blood transfusion. As the incidence data will vary from country to country, the anesthesiologist should be familiar with the most recent statistics in their country in order to have meaningful discussions with concerned parents (Table 4). Clearly the incidence of these viral pathogens in the blood pool is also dependent upon the prevalence of these infections within the donor community and the resources used to screen the blood products; unfortunately some less advantaged countries cannot afford to carry out all the necessary testing (51–59). In these settings it is likely that anemia (Hb $\leq$ 5 g dl$^{-1}$) is a better alternative than transfusion with contaminated blood provided that a stable cardiovascular and acid–base status can be maintained.

Besides the viral pathogens included in Table 4, the anesthesiologist should be aware that bacterial contamination may also occur, and is most common with platelets (60–63). Standards for testing of platelets for bacterial growth prior to release for administration are now being developed by the American Association of Blood Banks. Various other infectious diseases are potentially transmissible by transfusion including HTLV, West Nile virus, Babesiosis, Chagas disease, Lyme disease, malaria and Creutzfeldt–Jakob disease (64–70). Although no specific nucleic acid testing or antigen testing for many of these rare diseases exist, donor screening and deferral of those with potential symptoms helps to prevent transfusion-related transmission.

Another recent concern is the possibility of transmission of severe acute respiratory syndrome (SARS) through blood transfusion. The position of the American Association of Blood Banks (http://www.aabb.org/All_About_Blood/FAQs/aabb_faqs.htm) is that as live virus could be in the blood of a potential symptom free donor, they will ask if the donor has traveled to a SARS-affected region. They also inquire if the donor has had contact with a SARS-infected individual, and if so, they will not collect blood from that person.

### Incompatibility and other immunologic considerations

While transfusions of blood products can be highly beneficial in many clinical settings, side effects and other hazards may be associated with their use. Compatibility tests are standard procedures which are routinely performed to prevent hemolytic transfusion reactions which can be life-threatening. Table 5 displays the common blood types (USA) and Table 6 displays typical compatibilities for cross-match. Clerical error is the most common cause for a mismatched transfusion. These reactions can be decreased by scrupulous vigilance by the anesthesiologists and nurses responsible for checking blood products prior to patient administration (71–74). The most common clerical error we have observed is a careful check of the name on the unit and the name on the paperwork from the blood bank but not a cross check that the patient for whom it is intended is in fact the patient receiving it which then

### Table 3

Current blood screening tests used on donated blood in the United States

<table>
<thead>
<tr>
<th>Screening tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis B surface antigen (HBsAg)</td>
</tr>
<tr>
<td>Hepatitis B core antigen (anti-HBc)</td>
</tr>
<tr>
<td>Hepatitis C virus antibody (anti-HCV)</td>
</tr>
<tr>
<td>HIV-1 antibody (anti-HIV-1)</td>
</tr>
<tr>
<td>HIV-2 antibody (anti-HIV-2)</td>
</tr>
<tr>
<td>HTLV-I antibody (anti-HTLV-I)</td>
</tr>
<tr>
<td>HTLV-II antibody (anti-HTLV-II)</td>
</tr>
<tr>
<td>Nucleic acid amplification testing (NAT) for HIV-1 and HCV</td>
</tr>
<tr>
<td>Serologic test for syphilis</td>
</tr>
<tr>
<td>Nucleic acid amplification for West Nile virus</td>
</tr>
</tbody>
</table>

Abstracted from data in: http://www.aabb.org/All_About_Blood/FAQs/aabb_faqs.htm

### Table 4

Risk of transfusion related viral transmission and viral window for a negative screening test

<table>
<thead>
<tr>
<th>Virus</th>
<th>Days possible to transmit disease, i.e. false negative screen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis A</td>
<td>? ‘Occasional cases’</td>
</tr>
<tr>
<td>Hepatitis B</td>
<td>1 per 137 000</td>
</tr>
<tr>
<td>Hepatitis C</td>
<td>1 per 1 000 000</td>
</tr>
<tr>
<td>Human</td>
<td>1 per 641 000 or less</td>
</tr>
<tr>
<td>T-lymphotrophic virus I and II</td>
<td>1 per 1 900 000</td>
</tr>
<tr>
<td>HIV</td>
<td>1 per 1 900 000</td>
</tr>
</tbody>
</table>

Abstracted from data in: http://www.aabb.org/All_About_Blood/FAQs/aabb_faqs.htm

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results in administration of blood to the wrong recipient.

Severe acute hemolytic reactions most often result from immunologic destruction of red cells because of ABO incompatibility. Less frequently, serologic incompatibilities not detectable by standard antibody screens can cause an acute hemolytic reaction. Symptoms in the anesthetized patient include fever, tachycardia, hypotension and abnormal bleeding. If suspected, treatment should include immediate cessation of transfusion, measures to maintain or correct arterial blood pressure, and attention to the maintenance of good urine output. Anaphylactoid reactions with bronchospasm, laryngeal edema and urticaria are dangerous but rare and typically occur in IgA-deficient individuals. Treatment includes corticosteroids, antihistamines and epinephrine (75).

Transfusion-related graft vs host disease occurs when lymphocytes contained in a transfused blood component proliferate and cause host tissue destruction (8,76,77). This is primarily a problem in immunocompromised patients who cannot mount a response to foreign lymphocytes before this lethal proliferation occurs. Immunosuppression of this degree has been reported in premature infants, children suffering from cancer or severe systemic illness, children experiencing rapid acute blood loss, and those undergoing cardiopulmonary bypass (8,77–79). More important however is that graft vs host disease may also occur in immunocompetent children who receive directed donor transfusion from a biological relative (8). The lymphocytes present in these units may not be recognized as foreign by the recipient secondary to the similarity of haplotypes between relatives (77,79). Therefore, the recipient does not mount an immune response to the lymphocytes which may then proliferate and destroy host tissue. This is a critically important consideration when families are considering directed donor blood as ultimately safe for administration to their children.

Graft vs host disease may be prevented by effectively reducing lymphocyte counts in transfused blood. Irradiation of directed donor units and those cross-matched for transfusion to immunocompromised patients has been shown to effectively inhibit the lymphocytes from proliferating and decreases the likelihood of graft vs host disease (8,77,78). Thus it is vitally important that the anesthesiologist assures that these units have been

<table>
<thead>
<tr>
<th>Recipient</th>
<th>Whole blood</th>
<th>Packed RBC&lt;sup&gt;a&lt;/sup&gt;</th>
<th>FFP</th>
<th>Platelets&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>AB+</td>
<td>AB+, AB−</td>
<td>AB+, AB−, A+, A−, B+, B−, O−, O+</td>
<td>AB</td>
<td>AB+, AB− (A+, A−, B+, B−, O−, O− if hyper-concentrated)</td>
</tr>
<tr>
<td>AB−</td>
<td>AB−</td>
<td>AB−, A−, B−, O−</td>
<td>AB</td>
<td>AB− (A−, B−, O− if hyper-concentrated)</td>
</tr>
<tr>
<td>A−</td>
<td>A−</td>
<td>A−, O−</td>
<td>A, AB</td>
<td>A−, AB− (B−, O− if hyper-concentrated)</td>
</tr>
<tr>
<td>B−</td>
<td>B−</td>
<td>B−, O−</td>
<td>B, AB</td>
<td>B−, AB− (A−, O− if hyper-concentrated)</td>
</tr>
<tr>
<td>O+</td>
<td>O+, O−</td>
<td>O−, O+</td>
<td>O, A, B, AB</td>
<td>O+, O−, A+, A−, B+, B−, AB+, AB−</td>
</tr>
<tr>
<td>O−</td>
<td>O−</td>
<td>O−</td>
<td>O, A, B, AB</td>
<td>O−, A−, B−, AB−</td>
</tr>
</tbody>
</table>

<sup>a</sup>Rh− patients should not receive Rh+ RBC unless in emergency, particularly in females to avoid Rh sensitization.

<sup>b</sup>Platelets have a minute amount of RBC that might potentially cause Rh sensitization in Rh− patients. If giving Rh+ platelets is necessary, Rh immunoglobulin should be considered (Rh+ patients can always receive any Rh− products).
irradiated prior to administration and to return nonirradiated units to the blood bank. The anesthesiologist should take note that irradiated units contain higher concentrations of potassium than nonirradiated units. It is vital to maintain a high degree of vigilance for the development of hyperkalemia when administering irradiated units (8,23,24,80).

Acknowledgements

We wish to thank the Blood Bank of Children’s Memorial Hospital, Chicago IL, USA for assembling Table 6.

References


48 Comunale ME. A laboratory evaluation of the level 1 rapid infuser (H1025) and the Belmont instrument fluid management system (FMS 2000) for rapid transfusion. Anesth Analg 2003; 97: 1064–1069.


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