**DEFINITIONS:**

- The goal of transfusion is to provide minimum O₂ and CO₂ buffering and clotting factors to prevent hypothermia.
- Although RBC transfusions increase CaO₂, they may not be sufficient to keep core temperature above the mid 35°C threshold.

**Type and screen** – determines blood type and detects antibodies in recipient (e.g., indirect Coombs test) or donor (direct Coombs test).

**Crossmatch** – involving patient blood and specific donor units for compatibility. Crossmatch takes ~45 min.

In emergencies crossmatch can be skipped.

**EVIDENCE BASED TRANSFUSION THRESHOLDS:**

- Restrictive transfusion strategies (Hb > 7) are comparable/superior to liberal strategies in most settings including GI bleed, septic shock, cardiac surgery, TBI, and most ICU patients.
- Massive transfusion protocols (MTP) (e.g., trauma pts or massive GI bleed) target hemodynamic stability not a specific Hb. Among patients receiving MTP, **balanced ratio** (e.g., 1 RBC : 1 FFP : 1 platelet) is superior.
- Platelet transfusion thresholds are disease dependent: For most diseases 10k is adequate, if bleeding or needing surgery 50k may be required. Limited evidence for higher targets (e.g., 100k for CNS bleed).

**SPECIAL BLOOD PRODUCT TYPES:**

- **Leukocyte reduced RBC:** decreases incidence of febrile transfusion reaction. Also makes blood CMV-safe.
- **Gamma-irradiated RBC:** reduces incidence of GVHD during transfusions; important in very immunosuppressed patients.
- **Volume Reduced RBC:** each unit comes in ~100 mL (instead of ~350 mL), can reduce the incidence of febrile transfusion reaction because there are fewer plasma proteins; can also be used in volume overloaded patients (though giving diuretic is probably better).
- **Washed RBC:** plasma is replaced with crystalloid; this should be done only if there was a previous allergic reaction or in IgA deficient patients (if no IgA deficient donors).
- **Single donor (apheresis) platelets:** a full unit of platelets obtained from a single donor via apheresis (in contrast to pooled platelets typically combining 5 donors). Single donor limits antigen exposure.

**STRATEGIES IN PEOPLE WHO DECLINE TRANSFUSION:**

- Discuss specific reasons/concerns, understand what tx is acceptable.
- Correct coagulopathy (consider amicar, TXA, other products).
- Stop and minimize blood loss: hormonally suppress menstruation, autotransfuse with cell-saver (OR) or hemotrans/heart tube (ICU).
- Minimize iatrogenic blood loss (fewer labs, less frequently, drawn in pediatric tubes); no "routine" labs; every test should be thoughtful and drawn in pediatric tubes to minimize volume lost.
- Optimize hematopoiesis (IV iron infusion, folate supplementation, EPO administration).
- Consider blood substitute (poly-HEME).

**TRANSFUSIONS:**

- **Whole blood** (stored at 4°C up to 35 days): ~450 mL + 60 mL citrate. Good at achieving hemostasis (contains all factors) but limited availability (autologous, military).
- **RBCs** (stored at 4°C up to 42 days): ~350 mL. ↑ Hb ~1 gm/dl*
- **Pooled Platelet** (stored at RT up to 5 days): ~300 mL. ↑ Plat by ~5-7k*.
- **FFP** (frozen -25°C up to 3 years): ~225 mL (in 70 kg pt).

**TRANSFUSION REACTIONS:**

<table>
<thead>
<tr>
<th>REACTION</th>
<th>EXPLANATION</th>
<th>MANAGEMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Febrile Non-Hemolytic Transfusion Reaction (FNHTR)</td>
<td>Most common immune reaction to transfusion. Occurs within 4 hours of transfusion due to accumulated inflammatory cytokines in the banked donor blood. May recur; 25% of patients who had FNHTR once had another reaction subsequently.</td>
<td>Prevention: APAP + H2 blockers, consider 2nd line: methylprednisolone. Treatment: stop infusion, APAP, meperidine. R/o other causes. Notify blood bank.</td>
</tr>
<tr>
<td>Acute Hemolytic Transfusion Reaction (AHTR)</td>
<td>Occurs during or shortly after transfusion. Occurs due to mismatch of donor antigens (often AB0/Rh) &amp; recipient antibodies leading to hemolysis &amp; agglutination. S/sx: Fever, flank pain, dark urine, DIC, hypoT/Na, renal failure. Hemolysis on labs (J-agglutinin, T-LEH, etc).</td>
<td>A true emergency. Prevention: carefully check units. Treatment: Stop transfusion, notify blood bank, test for hemolysis &amp; DIC. aggressive IV hydration (goal UOP &gt; 100/hr).</td>
</tr>
<tr>
<td>Delayed Hemolytic Transfusion Reaction (DHTR)</td>
<td>Occurs 24 hours to 3 days after transfusion due to mismatch of minor antigens (often false negative crossmatch). 2nd exposure can be faster, more severe. May have drop in Hct, fever, minor hemolysis.</td>
<td>Treatment: Notify blood bank, repeat testing (DAT, type &amp; screen, etc).</td>
</tr>
<tr>
<td>Allergic reaction</td>
<td>Usually anaphylactoid (not IgE mediated). S/sx: urticaria, maculopapular rash, pruritis, fx &amp; hypoT/Na. Occurs minutes to hours after transfusion, due to antibodies against proteins on plt, leukocytes, or in plasma, including IgA in recipients w/ IgA deficiency.</td>
<td>Prevention: washed (or IgA deficient) RBCs. Check for IgA deficiency if recurrent anaphylaxis. Tx: epi, H2 blockers, steroids.</td>
</tr>
<tr>
<td>Post Transfusion Purpura (PTP)</td>
<td>Occurs 7-10 days after transfusion, due to anti-platelet antibodies in donor blood. Causes purpura &amp; severe thrombotic microangiopathy, may be life-threatening. More common in women (85%) &amp; Caucasians.</td>
<td>Treatment: IVIG, plasmapheresis</td>
</tr>
<tr>
<td>Transfusion Related Acute LUNg Injury (TRALI)</td>
<td>Leading cause of transfusion related death (15% mortality). TRALI resembles ARDS, onset is 4-6 hours after transfusion. Most common following platelet transfusion from multiparous female donors (due to anti-HLA or anti-HNAb).</td>
<td>Treatment: ventilatory support may be required (use LVP), use platelets from male donors for future transfusions.</td>
</tr>
<tr>
<td>Transfusion Associated Graft Versus Host Disease (TA-GVHD)</td>
<td>Occurs 8-10 days post transfusion, donor leukocytes attack immunosuppressed recipient. Sx include: fever, cutaneous eruptions, diarrhea, liver abnormalities. May progresses to panmyelopoenia due to marrow aplasia. High mortality.</td>
<td>Prevention: use irradiated and leukocyte reduced blood in immunosuppressed recipients. Treatment: no effective treatment</td>
</tr>
<tr>
<td>Transfusion Associated Cardiac Overload (TACO)</td>
<td>Occurs between 0-6 hrs after transfusion. Volume overload from transfusions, particularly in patients with CHF. Presents as dyspnea potentially progressing to severe hypoxemia.</td>
<td>Prevention: minimal units of, volume reduced units, diuresis.</td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>Citrate in RBCs binds to serum calcium. Blood products contain potassium from lysed cells.</td>
<td>Treatment: Replete calcium and monitor for hyperkalemia.</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>Due to low temp of transfused products. Iatrogenic hypothermia exacerbates coagulopathy &amp; ↑ bleeding</td>
<td>Prevention/Tx: Use a blood warmer for massive transfusions</td>
</tr>
<tr>
<td>Hypotension</td>
<td>People taking ACEi may develop hypotension due to inability to break down bradykinin in transfused blood</td>
<td>Does not require intervention. Rule out infection/hemolysis</td>
</tr>
</tbody>
</table>

**INFECTION:**

- Infection occurs due to **untested** organisms (rare), false negatives on testing (very rare), or bacterial contamination.
- Bacterial contamination: Platelets (stored at RT) are more likely to cause infections with skin flora (GPCs). RBCs (stored at 4°C), are more likely to be contaminated with GNs. Can lead to sepsis.
- Untested organisms: Organisms NOT tested include: Malaria, Borrelia (Lyme disease), Trypanosoma (Chagas disease), Babesiosis, & vCJD (varies by country).
- False negative: Extremely rare: HIV 1 in 2,000,000,000, HBV 1 in 100,000,000, HCV 1 in 2,000,000, HTLV 1 in 650,000.