Secondary hemostasis
The main purpose of secondary hemostasis is the production of fibrin to stabilize the platelet plug. Initiators of secondary hemostasis include the platelet plug and endothelial damage. It ends in the formation of thrombin, which cross-links fibrin and strengthens the clot. Thrombin also activates fibrinolysis, to limit excessive fibrin accumulation. Coagulopathies can be caused by a congenital defect or deficiency in any coagulation factor, lack of production of coagulation factors, excessive consumption of factors such as in the late phase of DIC, or by dilution.

Assessment of secondary hemostasis
Disorders of secondary hemostasis are recognized by hemorrhage into body cavities (e.g. hemothorax, hemoabdomen, hemomediastinum) or into organs (e.g. brain, urinary bladder, intestinal tract). Lower platelets counts generally can be found due to consumption, but they rarely are low enough (i.e. less than 20,000/µL) to suggest that they were the primary cause of the hemorrhage.

Treatment of impaired secondary hemostasis
Since most of the above tests are abnormal due to a lack of sufficient coagulation factors, the goal of therapy is to restore coagulation factors to levels that normalize these tests and, more importantly, reduce the chance for hemorrhage to persist. In general, 10-20 mL/kg of fresh frozen plasma will restore coagulation factors to a level to normalize the coagulation tests. This should be administered within 4 hours of the thaw at a rate at which the patient is not at risk for fluid overload.

Fresh plasma is obtained by centrifugation from FWB and separation into packed red blood cells (pRBC) and plasma within 6 hours of collection. After fresh plasma has been frozen, it is called FFP for up to 1 year. After one year of storage it is then referred to as Frozen Plasma due to the natural decrease in the labile factors (FV, FVIII, von Willebrand Factor) during storage.

The major indication is the treatment of coagulopathies, either acquired (e.g. rodenticide intoxication, DIC) or inherited (e.g. von Willebrand disease, hemophilias).

The required FFP dose in an individual patient is impossible to predict and must be based on response to therapy, availability of blood products and financial limitations. A general guideline is an initial dose between 10–20 mL/kg.

Fresh frozen plasma is not the fluid of choice for low albumin/low osmotic pressure in dogs and cats because it contains very low albumin (less than 5%, i.e. 5 mg/dL). It takes over 40ml/kg of plasma to raise albumin 1g/dL in the patient. Synthetic colloids can be used to increase the colloid osmotic pressure. Human serum albumin can be used to effectively increase albumin concentration in dogs and cats, but questions remain regarding safety of this product in our veterinary patients.

During the process of thawing fresh frozen plasma at 4°C, a white precipitate forms due to the run-off of thawed portion; that is the cryoprecipitate (CP), which can be separate and stored. The major indication for the use of CP is von Willebrand disease and Hemophilia A. The published dose is 10 mL/kg.

Treatment of anemia
Human patients are at risk of decrease oxygen delivery and organ failure with a hemoglobin of 3-4 g/dL (hematocrit around 9-12%). Transfusion decisions, however, cannot be based only on the level of anemia due to compensatory mechanisms for anemia. Thus, clinical signs and the underlying causes of anemia are also very important. The transfusion should be considered as soon as the patient cardiovascular status is jeopardized by the blood loss. Clinical signs include:

- Physical examination perfusion parameters: poor perfusion: tachycardia, pale mucous membranes, prolonged capillary refill time, weak pulses, cold extremities, altered mentation.
- Physical examination signs of anemia (pale mucous membrane color and narrow pulses) are difficult to differentiate from hypoperfusion/shock and need to be re-assessed after initial fluid resuscitation.
- Perfusion markers: increased lactate, decreased blood pressure and urine output.
- Respiratory function: increased respiratory rate and effort, blood gas analysis.

The variable clinical response to anemia in patients suggests different mechanisms of adaptation over time. Therefore, many parameters need to be determined prior to instituting red cell transfusion. A patient with an acute splenic rupture from abdominal trauma can manifest shock and severe blood loss with a small drop in hematocrit. Some patients walk in the Emergency Room with a hematocrit of 6% and appear clinically stable. When chronic anemia occurs, three principal compensatory mechanisms occur:

1. Decreased haemoglobin affinity for oxygen: A rightward shift of the oxyhemoglobin dissociation curve is caused by decreased pH (acidity), increased 2,3-DPG, increased temperature, and increased PCO2. Red blood cells produce more 2,3-DPG under conditions of chronic hypoxia (i.e. anemia); therefore, oxygen is more easily released at the tissue level.
2. Redistribution of blood flow: In anemia, selective vasoconstriction of blood vessels underserves certain non-vital areas (skin for example) and allows more blood to flow into critical areas like the heart or brain.

3. Increased cardiac output: The increased output is matched by decreased peripheral vascular resistance and decreased blood viscosity, so that cardiac output can rise without an increase in blood pressure. Generally, anemia must be fairly severe (hemoglobin < 7 g/dL) before cardiac output rises.

Thus, duration (acute versus chronic) of anemia needs to be taken into account because of normal adaptation of the body to the blood loss.

The transfusion trigger
The threshold values for the clinical use of blood products to treat anemia are not well defined in the veterinary literature. For many years in human medicine, the transfusion trigger had been set at a hemoglobin (Hgb) of 10 g/dL (hematocrit [HCT] of 30%). However, transmissible infections, immunologic risks, transfusion-related acute lung injury, the cost of blood, and component availability are current disadvantages of transfusions. In 1999, the Transfusion Requirements in Critical Care (TRICC) trial showed that maintenance of a Hgb between 7 and 9 g/dL in ICU patients is as effective with less adverse consequences as the maintenance of a Hgb above 9 g/dL. Based on that information, hemoglobin-based threshold values have recently been reviewed for humans and are currently between 6 and 8 g/dL of Hgb (HCT or packed cell volume [PCV] around 20%).

The time it took to achieve that particular Hgb/HCT/PCV value is also an important factor in the determination of when to give red blood cells. Animals with chronic disease may have suppression in the production of, or an insensitivity to, erythropoietin. This more chronic anemia will often go clinically undetected for weeks to months as the patient compensates. Animals with an acute anemia, i.e. from trauma/hemorrhage into body cavities, have little physiologic compensation for this abrupt change in oxygen carrying capacity.

Other objective measures that will help to decide if a transfusion is warranted include: venous oxygen tensions and lactate. As oxygen delivery to the tissues falls but the tissue extraction remains the same, less venous oxygen is present when measuring PVO2. This however may reflect increased oxygen demand by the tissues. Alternatively, an increase in lactate is a readily available marker of tissue anaerobiosis or poor perfusion.

Red blood cell dosage
As a rule of thumb, 2 mL/kg of FWB will increase the PCV by 1% point. So, 20 mL/kg are used to increase the PCV by 10 percentage points.

In normovolemic, anemic patients, such as autoimmune hemolytic anemia, the volume required to increase the PCV may place the patient at risk of fluid overload. This is a particular concern in the cardiac patient.

Packed red blood cells (pRBC) will have a very small amount of remaining plasma. The hematocrit of pRBC may exceed 80%. In that case, dilution with 0.9% NaCl can be helpful to avoid hyperviscosity. Fluids containing calcium, like LRS, should not be used in order to decrease the risk of citrate chelation and coagulation.

As a rule of thumb, 1 mL/kg of pRBC will increase the PCV by 1% point. The post-transfusion PCV goal is usually between 18 - 25%.