Thrombocytopenia. References.

1. Tepler I, *et al.* A randomized placebo-controlled trial of recombinant human interleukin-11 in cancer patients with severe thrombocytopenia due to chemotherapy. *Blood* 1996; **87:** 3607–14.
2. Isaacs C, *et al.* Randomized placebo-controlled study of recombinant human interleukin-11 to prevent chemotherapy-induced thrombocytopenia in patients with breast cancer receiving dose-intensive cyclophosphamide and doxorubicin. *J Clin Oncol* 1997; **15:** 3368–77.
3. Reynolds CH. Clinical efficacy of rhIL-11. *Oncology (Huntingt)* 2000; **14** (suppl 8): 32–40.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Neumega†; ***Braz.:*** Neumega; ***Chile:*** Neumega†; ***Mex.:*** Neumega†; ***USA:*** Neumega; ***Venez.:*** Neumega.

Oxidised Cellulose

Cellulosic Acid; Celulosa oxidada; Oxidized Cellulose.

CAS — 9032-53-5.
ATC — B02BC02.
ATC Vet — QB02BC02.

Description. Oxidised cellulose is a sterile polyanhydroglucuronic acid, prepared by the oxidation of a suitable form of cellulose.

Pharmacopoeias. In *US* which also includes Oxidized Regenerated Cellulose.
USP 31 (Oxidized Cellulose). It contains not less than 16% and not more than 24% of carboxyl groups, calculated on the dried basis. It is a slightly off-white gauze or lint with a slight, charred odour. Insoluble in water and in acids; soluble in dilute alkalis. Store at a temperature not exceeding 8°. Protect from direct sunlight.
USP 31 (Oxidized Regenerated Cellulose). It contains 18 to 24% of carboxyl groups calculated on the dried basis. It is a slightly off-white knit fabric, with a slight odour. Insoluble in water and in dilute acids; soluble in dilute alkalis. Store at a temperature between 15° and 30°. Protect from direct sunlight.

Adverse Effects and Precautions

Foreign body reactions may occur after the use of oxidised cellulose or oxidised regenerated cellulose. Headache, burning, and stinging have been reported and sneezing has been noted after use in epistaxis. Oxidised cellulose swells on contact with a bleeding surface; this could result in tissue necrosis, nerve damage, obstruction, or vascular stenosis if packed closely, especially into bony cavities, or if wrapped tightly around blood vessels. To minimise such complications the removal of excess material should be considered after haemostasis is achieved, and oxidised cellulose should always be removed after use near the spinal cord or optic nerve. Oxidised cellulose should not be used in packing or implantation for fractures since it may interfere with bone regeneration or cause cyst formation. It should not be used as a surface dressing, except for immediate control of haemorrhage, as it inhibits epithelialisation.

Oxidised cellulose should be used as the dry material since moistening will reduce its ability to absorb blood. Silver nitrate or other escharotic chemicals should not be applied before use as cauterisation might inhibit absorption of oxidised cellulose. Thrombin is inactivated by the low pH of oxidised cellulose; it is recommended that oxidised cellulose should not be impregnated with other haemostatics or antibacterials.

Uses and Administration

Oxidised cellulose and oxidised regenerated cellulose are absorbable haemostatics (p.1045). When applied to a bleeding surface, they swell to form a gelatinous mass which aids in the formation of a clot. It is gradually absorbed by the tissues, usually within 7 to 14 days. These materials also have a weak bactericidal action. They are used in surgery as adjuncts in the control of moderate bleeding where suturing or ligation is impracticable or ineffective; they should not be used to control haemorrhage from large arteries. The gauze, lint, or knitted material should be laid on the bleeding surface or held firmly against the tissues until haemostasis is achieved; removal should then be considered (see Adverse Effects and Precautions, above). Oxidised cellulose should be used as the dry material as moistening will reduce its ability to absorb blood.

Preparations

Proprietary Preparations (details are given in Part 3)
Fr.: Surgicel; ***Ger.:*** Tabotamp; ***Hong Kong:*** Seal On†; ***Irl.:*** Alltracel P†; Premdoc†; Seal-On; Traumacel P; ***Ital.:*** Tabotamp; ***UK:*** Oxycel; StopBleed; ***USA:*** Oxycel; Surgicel.

Multi-ingredient: ***Fr.:*** Promogran; ***Irl.:*** Alltracel S†; ***Ital.:*** Promogran; ***UK:*** Seal-On.

Oxypolygelatin ⊗

Oxipoligelatina.

Profile

Oxypolygelatin is a polymer derived from gelatin (p.1072). It is used as a 5.5% solution as a plasma volume expander. There have been reports of anaphylaxis.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Gelifundol†; ***Austria:*** Gelifundol; ***Cz.:*** Gelifundol†; ***Ger.:*** Gelifundol†; ***Hong Kong:*** Gelifundol†; ***Hung.:*** Gelifundol†; ***S.Afr.:*** Gelifundol†; ***Thai.:*** Gelifundol.

Pegzerepoetin Alfa ⊗

Methoxy Polyethylene Glycol-Epoetin Beta; Pegserepoetin Alfa; R-744; Ro-50-3821. 1-165-Erythropoietin (human) monoamide with α-(3-carboxypropyl)-ω-methoxypoly(oxy-1,2-ethanediyl).

CAS — 677324-53-7.
ATC — B03XA03.
ATC Vet — QB03XA03.

Adverse Effects and Precautions

As for Epoetins, p.1061.

Pharmacokinetics

In patients with chronic renal impairment, pegzerepoetin alfa is absorbed after subcutaneous injection with an absolute bioavailability of about 60%. It has a terminal elimination half-life of about 134 hours after intravenous injection and about 140 hours when given subcutaneously. Haemodialysis does not affect the pharmacokinetics of pegzerepoetin alfa.

Uses and Administration

Pegzerepoetin alfa is described as a continuous erythropoietin receptor activator (CERA). It has similar properties to the epoetins (p.1062), but a longer duration of action. Pegzerepoetin alfa is used in the treatment of anaemia associated with chronic renal failure (see Normocytic-normochromic Anaemia, p.1044). A starting dose of 600 nanograms/kg is given once every 2 weeks as a single intravenous or subcutaneous injection. The dose may be adjusted by about 25%, at monthly intervals, so that the rate of rise of haemoglobin is between 1 and 2 g per 100 mL each month. When the target haemoglobin concentration of between 11 and 12 g per 100 mL has been achieved, a maintenance dose of pegzerepoetin alfa may be given once monthly; this is equal to twice the dose that had been given once every 2 weeks.

Pegzerepoetin alfa is also under investigation in the treatment of anaemia in patients with non-myeloid malignant disease receiving chemotherapy.

◊ References.

1. de Francisco ALM, *et al.* BA16260 Study Investigators. Continuous Erythropoietin Receptor Activator (C.E.R.A.) administered at extended administration intervals corrects anaemia in patients with chronic kidney disease on dialysis: a randomised, multicentre, multiple-dose, phase II study. *Int J Clin Pract* 2006; **60:** 1687–96.
2. Sulowicz W, *et al.* PROTOS Study Investigators. Once-monthly subcutaneous C.E.R.A. maintains stable hemoglobin control in patients with chronic kidney disease on dialysis and converted directly from epoetin one to three times weekly. *Clin J Am Soc Nephrol* 2007; **2:** 637–46.
3. Levin NW, *et al.* MAXIMA study investigators. Intravenous methoxy polyethylene glycol-epoetin beta for haemoglobin control in patients with chronic kidney disease who are on dialysis: a randomised non-inferiority trial (MAXIMA). *Lancet* 2007; **370:** 1415–21.
4. Österborg A, *et al.* Phase II study of three dose levels of continuous erythropoietin receptor activator (C.E.R.A.) in anaemic patients with aggressive non-Hodgkin's lymphoma receiving combination chemotherapy. *Br J Haematol* 2007; **136:** 736–44.

Preparations

Proprietary Preparations (details are given in Part 3)
Cz.: Mircera; ***Fr.:*** Mircera; ***Port.:*** Mircera; ***UK:*** Mircera; ***USA:*** Mircera.

Plasma

Pharmacopoeias. Many pharmacopoeias have monographs, including *Eur.* (see p.vii).
Ph. Eur. 6.2 (Human Plasma for Fractionation; Plasma Humanum ad Separationem). The liquid part of human blood remaining after separation of the cellular elements from whole blood or collected in an apheresis procedure; it is intended for the manufacture of plasma-derived products. It is obtained from healthy donors and is tested for the absence of hepatitis B surface antigen and antibodies against HIV-1 and HIV-2 and hepatitis C virus.
A light yellow to green, clear or slightly turbid liquid, without visible signs of haemolysis. Frozen plasma should be stored at or below –20°; it may still be used for fractionation if the temperature is between –20° and –15° for not more than a total of 72 hours without exceeding –15° on more than one occasion as long as the temperature is at all times –5° or lower.
Ph. Eur. 6.2 (Human Plasma (Pooled and Treated for Virus Inactivation); Plasma Humanum Coagumentum Conditumque ad Exstinguendum Virum). A frozen or freeze-dried, sterile, non-pyrogenic preparation obtained from human plasma derived from donors belonging to the same ABO blood group. The plasma used complies with the requirements for Human Plasma for Fractionation (above). The method of preparation is designed to minimise activation of any coagulation factor and includes a step or steps that have been shown to inactivate known agents of infection.
The frozen preparation, after thawing, is a clear or slightly opalescent liquid free from solid and gelatinous particles. The freeze-dried preparation is an almost white or slightly yellow powder or friable solid.

Adverse Effects and Precautions

As for Blood, p.1056, though with a low risk of transmitting cell-associated viruses. However, the production of blood products using plasma from UK donors has been phased out due to the possible risk of transmission of Creutzfeldt-Jakob disease.

Uses and Administration

Fresh frozen plasma contains useful amounts of clotting factors. It should be reserved for patients with proven abnormalities in blood coagulation. Indications include congenital deficiencies in clotting factors for which specific concentrates are unavailable, severe multiple clotting factor deficiencies (for example in patients with liver disease), rapid reversal of the action of coumarin anticoagulants, and disseminated intravascular coagulation. It may be used after massive blood transfusion when there is evidence of coagulation deficiency but its value for routine prophylaxis against abnormal bleeding tendencies in patients receiving massive blood transfusions is contentious except where clotting abnormalities have been confirmed. It has also been used in the treatment of thrombotic thrombocytopenic purpura and as a source of plasma proteins.

The amount of fresh frozen plasma transfused depends on the required level of clotting factors. A unit of fresh frozen plasma refers to the quantity of plasma obtained from 1 unit of whole blood; this generally represents a volume of about 250 mL, including anticoagulant.

Fresh frozen plasma should not be used as a volume expander or as a nutritional source.

Therapeutic plasma exchange or plasmapheresis (see below) are used in a wide variety of disorders.

Plasma is used to prepare blood products including albumin, antithrombin III, blood clotting factors, immunoglobulins, and platelets. Other preparations include cryoprecipitate depleted plasma, which is deficient in fibrinogen, factor VIII, von Willebrand factor, cryoglobulin, and fibronectin, and single donor plasma, which is not frozen. A solvent-detergent-treated plasma preparation is available.

Guidelines and reviews. General references to the use of plasma.

1. Fresh-frozen Plasma, Cryoprecipitate, and Platelets Administration Practice Guidelines Development Task Force of the College of American Pathologists. Practice parameter for the use of fresh-frozen plasma, cryoprecipitate, and platelets. *JAMA* 1994; **271:** 777–81.
2. Cohen H, *et al.* Plasma, plasma products, and indications for their use. In: Contreras M, ed. *ABC of transfusion*. 3rd ed. London; BMJ Books, 1998: 40–44.
3. British Committee for Standards in Haematology, Blood Transfusion Task Force. Guidelines for the use of fresh-frozen plasma, cryoprecipitate and cryosupernatant. *Br J Haematol* 2004; **126:** 11–28. Also available at: http://www.bcshguidelines.com/pdf/freshfrozen_280604.pdf (accessed 27/10/05) Addenda, amendments, and corrections (4 sets) at http://www.bcshguidelines.com/pdf/Amendments_FFP_091205.pdf (issued 07/12/05), *ibid.* 2007; **136:** 514–16, at http://www.bcshguidelines.com/pdf/FFPAmendment_1_17_Oct_2007.pdf (issued 17/10/07), at http://www.bcshguidelines.com/pdf/FFPAmendment_2_17_Oct_2007.pdf (issued 17/10/07) (accessed 19/06/08)
4. Stanworth SJ, *et al.* Is fresh frozen plasma clinically effective? A systematic review of randomized controlled trials. *Br J Haematol* 2004; **126:** 139–52.

Hereditary angioedema. For a mention of fresh frozen plasma being used in hereditary angioedema, see p.1081.

Neonatal intraventricular haemorrhage. Plasma volume expansion in preterm neonates has been thought to help prevent neonatal intraventricular haemorrhage (p.1050). However, a study using plasma or gelatin as plasma volume expanders,[1,2] found no evidence of a decreased risk of such haemorrhage or subsequent death or disability.

1. The Northern Neonatal Nursing Initiative Trial Group. A randomized trial comparing the effect of prophylactic intravenous fresh frozen plasma, gelatin or glucose on early mortality and morbidity in preterm babies. *Eur J Pediatr* 1996; **155:** 580–8.
2. Northern Neonatal Nursing Initiative Trial Group. Randomised trial of prophylactic early fresh-frozen plasma or gelatin or glucose in preterm babies: outcome at 2 years. *Lancet* 1996; **348:** 229–32.

Plasma exchange. Therapeutic plasma exchange or plasmapheresis are procedures in which plasma is selectively removed from the body while the cellular constituents of blood are retained. Although the two terms are commonly used synonymously, plasmapheresis generally involves the removal of small volumes of plasma, whereas plasma exchange removes larger volumes which must be replaced with a suitable fluid.

They have been tried in a number of disorders, including many with an immunological basis, when conventional treatment has not been successful. The aim is removal or reduction of those constituents of plasma causing or aggravating a disease or replacement of deficient plasma factors if the deficiency is the cause of the disorder.

Volume and frequency of plasma exchange is determined by the pathophysiology of the undesirable plasma constituent. For example, removal of antibody usually requires exchange of 1.5 times the estimated plasma volume (3 to 4 litres) repeated daily or on alternate days until the desired reduction is obtained. The replacement fluid used depends on the volume and the condition being treated; albumin solutions, plasma expanders, or sodium chloride 0.9% are frequently used, whereas in conditions where there is deficiency of a plasma factor replacement of blood components such as immunoglobulins may be required. Fresh frozen plasma has been used as a replacement fluid but is associated with a high incidence of adverse effects and is generally reserved for the management of thrombotic thrombocytopenic purpura.

Technological developments, such as the use of specific adsorbents and the use of multiple filters with different pore sizes, may enable removal of only the desired constituent and avoid removal and subsequent replacement of total plasma.

References.

1. Urbaniak SJ, Robinson EA. Therapeutic apheresis. In: Contreras M, ed. *ABC of transfusion*. 3rd ed. London: BMJ Books, 1998: 67–70.
2. Michaud D, *et al.* Therapeutic plasma exchange. *Dynamics* 2001; **12:** 18–24.
3. Madore F. Plasmapheresis: technical aspects and indications. *Crit Care Clin* 2002; **18:** 375–92.
4. McLeod BC. Therapeutic apheresis: use of human serum albumin, fresh frozen plasma and cryosupernatant plasma in therapeutic plasma exchange. *Best Pract Res Clin Haematol* 2006; **19:** 157–67.

Thrombotic microangiopathies. Thrombotic thrombocytopenic purpura and haemolytic-uraemic syndrome are both syndromes characterised by intravascular platelet clumping.[1-6] Thrombocytopenia also occurs and fragmentation of erythrocytes, partly caused by the red cells passing through areas of the microvasculature occluded by the platelet aggregation, leads to microvascular haemolytic anaemia. In **thrombotic thrombocytopenic purpura** (TTP) the platelet aggregation is extensive and obstructs the vessels of various organs producing ischaemia or even infarction. The CNS, notably the brain, is often the area predominantly affected although some degree of renal involvement may occur. It is an uncommon disorder; adult women, in whom the condition presents as a chronic relapsing illness, are slightly more frequently affected. It may be associated with abnormalities of von Willebrand factor due to deficiency or impaired activity of a protease, ADAMTS-13.[5,6]

In **haemolytic-uraemic syndrome** (HUS) the platelet aggregation is relatively less widespread and less severe and mainly affects the renal microvasculature although extra-renal manifestations may also occur. The primary consequences are hypertension and acute renal insufficiency or ultimately, if untreated, renal failure. Most cases of HUS occur in early childhood and follow a diarrhoeal illness caused by *Shigella dysenteriae* or *Escherichia coli*. However, the condition is becoming increasingly recognised in adults, particularly the elderly. Some cases may be drug induced. With appropriate symptomatic therapy HUS is typically a self-limiting disease with spontaneous recovery although fatalities have been known.

The supportive **management** of both syndromes follows similar lines.[1,3,4] In HUS, or TTP with renal symptoms, special attention needs to be directed towards the prevention of renal failure. Hypovolaemia should be corrected, with careful control of fluid and electrolyte balance and hypertension. Haemodialysis will be needed if renal failure develops. Severe anaemia requires blood transfusion, but platelet transfusion should be avoided.

Plasma exchange (see above) is considered to be the mainstay of therapy for TTP.[1-6] The optimal regimen has not been determined, but it is usually performed daily. There is also some debate about the preferred fluid replacement; plasma exchange using cryosupernatant (the plasma remaining after cryoprecipitate is prepared, and which is depleted of von Willebrand factor) may be more efficacious than fresh frozen plasma.[3] When plasma exchange is not available, infusion of fresh frozen plasma may be used.[1,3] In HUS, there is some debate over the use of plasma exchange or infusion. Some consider that these have no proven benefit in HUS[2,3] but others[1] have challenged this belief.

Antiplatelet therapy and corticosteroids are often given, although neither has been adequately investigated and antiplatelets such as ticlopidine and clopidogrel have been reported to cause TTP (see p.1411). Aspirin and dipyridamole have been used, but are not recommended when profound thrombocytopenia is present because of the potential bleeding risk, without proven benefit. However, low-dose aspirin may be used when platelet counts have recovered after plasma exchange in TTP.[1,3] Some reports have described improved outcome in both syndromes with corticosteroids.[7] They are often used with plasma exchange in TTP.[1,3,4] However, a randomised, double-blind trial[8] in children with HUS failed to show any difference between oral corticosteroids and placebo in terms of haematological or neurological recovery, although renal function appeared to improve more rapidly in those receiving corticosteroids.

Other drugs may also be tried, particularly in refractory TTP. Some treatments that have been reported to be beneficial in case reports or small series include normal immunoglobulin,[1,4] azathioprine,[1] ciclosporin,[1,3] cyclophosphamide,[3] and vincristine.[1-4] The monoclonal antibody, rituximab, is under investigation.[2] The use of a protein-A immuno-adsorption column may be considered in the management of TTP associated with malignancy or bone marrow transplantation.[3] Epoprostenol may be tried in order to inhibit platelet-endothelial interactions but again has not been subject to controlled studies; anecdotal evidence presents both favourable and negative results.[9] Alteplase has been used successfully in one patient with HUS.[10] Splenectomy may also be considered.[1,3,4]

1. Elliott MA, Nichols WL. Thrombotic thrombocytopenic purpura and hemolytic uremic syndrome. *Mayo Clin Proc* 2001; **76:** 1154–62.
2. Moake JL. Thrombotic microangiopathies. *N Engl J Med* 2002; **347:** 589–600.
3. British Society for Haematology. Guidelines on the diagnosis and management of the thrombotic microangiopathic haemolytic anaemias. *Br J Haematol* 2003; **120:** 556–73. Also available at: http://www.bcshguidelines.com/pdf/BJH556.pdf (accessed 27/10/05)
4. Nabhan C, Kwaan HC. Current concepts in the diagnosis and management of thrombotic thrombocytopenic purpura. *Hematol Oncol Clin North Am* 2003; **17:** 177–99.
5. Mayer SA, Aledort LM. Thrombotic microangiopathy: differential diagnosis, pathophysiology and therapeutic strategies. *Mt Sinai J Med* 2005; **72:** 166–75.
6. George JN. Thrombotic thrombocytopenic purpura. *N Engl J Med* 2006; **354:** 1927–35.
7. Bell WR, *et al.* Improved survival in thrombotic thrombocytopenic purpura-hemolytic uremic syndrome: clinical experience in 108 patients. *N Engl J Med* 1991; **325:** 398–403.
8. Perez N, *et al.* Steroids in the hemolytic uremic syndrome. *Pediatr Nephrol* 1998; **12:** 101–4.
9. Bobbio-Pallavicini E, *et al.* Intravenous prostacyclin (as epoprostenol) infusion in thrombotic thrombocytopenic purpura: four case reports and review of the literature. *Haematologica* 1994; **79:** 429–37.
10. Kruez W, *et al.* Successful treatment of haemolytic-uraemic syndrome with recombinant tissue-type plasminogen activator. *Lancet* 1993; **341:** 1665–6.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Octaplas; ***Cz.:*** Octaplas; ***Fin.:*** Octaplas; ***Ger.:*** Octaplas; ***Ital.:*** Octaplas; Plasmasafe; ***Mex.:*** Octaplas; ***Neth.:*** Octaplas; ***Norw.:*** Octaplas; ***NZ:*** Octaplas; ***Port.:*** Novoplas; Octaplas; ***Swed.:*** Octaplas; ***Switz.:*** Octaplas; ***UK:*** Octaplas.

Multi-ingredient: ***Port.:*** Quixil.

Plasma Protein Fraction ⊗

Fracción proteica del plasma.

Pharmacopoeias. Many pharmacopoeias have monographs, including *US*.

USP 31 (Plasma Protein Fraction). A sterile preparation of serum albumin and globulin obtained by fractionating material (blood, plasma, or serum) from healthy human donors, the source material being tested for the absence of hepatitis B surface antigen. It contains 5% of protein; not less than 83% of the total protein is albumin; not more than 17% is alpha and beta globulins; not more than 1% has the electrophoretic properties of gamma globulin. It contains sodium acetyltryptophanate with or without sodium caprylate as a stabilising agent but no antimicrobial preservative. It contains 130 to 160 mmol/litre of sodium, and not more than 2 mmol/litre of potassium. A solution in 0.15M sodium chloride containing 1% protein has a pH between 6.7 and 7.3. It should be used within 4 hours of opening the container.

Profile

Plasma protein fraction consists mainly of albumin with a small proportion of globulins; it does not contain blood-clotting factors. It has properties and uses similar to those of other albumin solutions (p.1052). It is given intravenously as a solution containing 5% of total protein. The amount of plasma protein fraction given will depend upon the clinical condition of the patient. For hypovolaemic shock an initial infusion of up to 500 mL for adults has been suggested at a rate not normally exceeding 10 mL/minute. A suggested dose in infants and small children for shock with dehydration is up to 33 mL/kg given at a rate of up to 5 to 10 mL/minute. In hypoproteinaemia, 1 to 1.5 litres of a 5% solution will provide 50 to 75 g of protein. Patients with normal blood volume may require slow infusion to prevent excessive volume expansion.

As with other albumin solutions, plasma protein fraction should not be used for parenteral nutrition.

Preparations

USP 31: Plasma Protein Fraction.

Proprietary Preparations (details are given in Part 3)
Austria: Biseko; ***Cz.:*** Biseko†; ***Ger.:*** Biseko; ***Gr.:*** Alburex; ***Hung.:*** Biseko; ***Indon.:*** Plasmanate; ***Israel:*** Plasmanate; ***Ital.:*** Haimaserum†; PPS; Uman-Serum; ***Malaysia:*** Plasmanate†; ***Philipp.:*** Plasmanate; ***S.Afr.:*** Bioplasma FDP; ***Thai.:*** Biseko; ***USA:*** Plasma-Plex; Plasmanate; Protenate.

Multi-ingredient: ***Fin.:*** Tisseel Duo Quick; ***Ger.:*** Tissucol Duo S; Tissucol-Kit; ***Hung.:*** Tissucol-Kit; ***Ital.:*** Tissucol; ***Swed.:*** Tisseel Duo Quick; ***Switz.:*** Tissucol Duo S.

Platelets

Plaquetas.

Pharmacopoeias. Many pharmacopoeias have monographs, including *US*.

USP 31 (Platelets). The portion of blood that contains platelet cells derived from human whole blood from which red blood cells and a portion of the plasma are removed by centrifugation, sedimentation, or apheresis. Platelets derived from whole blood may be pooled from multiple donors to form one dose of platelets. The source blood for platelets must be tested for syphilis, hepatitis B, human T-cell lymphotropic virus (HTLV) Type I and Type II, hepatitis C, and HIV Type 1 and Type 2.

Platelets derived from whole blood should have a minimum of 5.5×10^{10} platelet cells suspended in a volume of 40 to 70 mL of original plasma. Platelets produced by apheresis should have a minimum of 3.0×10^{11} platelet cells suspended in 100 to 500 mL of original plasma or in an approved additive solution.

Platelets derived from whole blood or by apheresis may be further processed by filtration for removal of leucocytes, or by irradiation to inactivate lymphocytes.

The names of the different platelet preparations are:

- Platelets—prepared from a single unit of whole human blood within 8 hours of collection
- Platelets, Pooled—individual platelet units derived from whole human blood and pooled by aseptic techniques, labelled with a unique identifying number related to the number of individual units pooled, and with an expiry date of 4 hours after pooling of the individual units
- Platelets, Pheresis—prepared by apheresis from a single donor
- Platelets, Leukocyte Reduced—prepared from whole blood, either by centrifugation or by sedimentation, and filtered to yield less than 8.3×10^5 white blood cells in the final container
- Platelets, Pheresis, Leukocyte Reduced—contains less than 5×10^6 white blood cells, prepared by apheresis, with or without a filter

Platelets may be stored in plasma or in an approved additive solution at 20° to 24° with continuous gentle agitation for no more than 5 days after date of preparation. The pH must be greater than 6.2 throughout the storage period.

USP 31 (Platelet Concentrate). It contains the platelets taken from plasma obtained, in a single procedure, by whole blood collection, plasmapheresis, or plateletpheresis from a single suitable human donor. The platelets are suspended in a specified volume (20 to 30 mL, or 30 to 50 mL) of the original plasma. The suspension contains not less than 5.5×10^{10} platelets per unit in not less than 75% of the units tested. It should be stored in hermetically-sealed sterile containers at 20° to 24° (30 to 50 mL volume), or at 1° to 6° (20 to 30 mL volume) except during transport when the temperature may be 1° to 10°. The expiration time is not more than 72 hours from the time of collection of the source material. Continuous gentle agitation must be maintained if stored at 20° to 24°. The suspension must be used within 4 hours of opening the container and should be administered with equipment that contains a filter.

Adverse Effects and Precautions

Transmission of infection has been associated with the transfusion of blood products including platelets (p.1056). Since platelets are stored at room temperature there is increased risk of bacterial infection after transfusion. Transfusion reactions including fever and urticaria are not uncommon. Recipients of multiple transfusions of platelet concentrates from random donors may develop antibodies to HLA which result in impaired responsiveness to subsequent transfusions.