ally present with fever of sudden onset, sore throat, mouth ulcers, headache, and malaise. This condition is also known as **agranulocytosis**. Other causes of acquired neutropenia include serious bacterial and viral infections, radiotherapy, neoplasms that invade bone marrow, and some auto-immune disorders.

The management of acquired neutropenia includes the treatment of any contributory condition. Drug-induced neutropenia is usually managed by withdrawal of the offending drug. After an idiosyncratic reaction the implicated drug should not be given again, since abrupt neutropenia will usually be precipitated. Colony-stimulating factors can be used to manage drug-induced neutropenia.

In all neutropenic patients onset of fever is indicative of serious infection and is treated immediately with empirical antibacterial therapy as described on p.174.

◊ General references.

1. Zeidler C, *et al.* Congenital neutropenias. *Rev Clin Exp Hematol* 2003; **7:** 72–83.
2. Bhatt V, Saleem A. Drug-induced neutropenia—pathophysiology, clinical features, and management. *Ann Clin Lab Sci* 2004; **34:** 131–7.
3. James RM, Kinsey SE. The investigation and management of chronic neutropenia in children. *Arch Dis Child* 2006; **91:** 852–8.

Albumin ⊗

Albümin; Albúmina; Albumine; Albuminum.

ATC — B05AA01.

ATC Vet — QB05AA01; QV08DA01 (microspheres of human albumin).

Pharmacopoeias. Many pharmacopoeias have monographs, including *Eur.* (see p.vii) and *US.*

Ph. Eur. 6.2 (Human Albumin Solution; Albumini Humani Solutio). An aqueous solution of protein obtained from the plasma of healthy donors; the plasma is tested for the absence of hepatitis B surface antigen and antibodies against HIV-1 and HIV-2 and hepatitis C virus. It is prepared as a concentrated solution containing 15 to 25% of total protein or as an isotonic solution containing 3.5 to 5% of total protein; not less than 95% of the total protein is albumin. A suitable stabiliser, such as sodium octanoate or *N*-acetyltryptophan or a combination of the two, may be added but no antimicrobial preservative is added. It contains not more than 160 mmol of sodium per litre and not more than 200 micrograms of aluminium per litre. The solution is sterilised by filtration and distributed aseptically into containers which are sealed to prevent contamination and maintained at 59° to 61° for not less than 10 hours. Finally, the containers are incubated for not less than 14 days at 30° to 32° or for not less than 4 weeks at 20° to 25° and examined visually for signs of microbial contamination. It should be stored in a colourless glass container and protected from light.

A clear, almost colourless, yellow, amber, or green slightly viscous liquid. A solution in sodium chloride 0.9% containing 1% protein has a pH of 6.7 to 7.3.

The BP 2008 gives Albumin and Human Albumin as approved synonyms.

USP 31 (Albumin Human). A sterile, nonpyrogenic, preparation of serum albumin obtained by fractionating material (blood, plasma, serum, or placentas) from healthy human donors, the source material being tested for the absence of hepatitis B surface antigen. It is made by a process that yields a product that is safe for intravenous use. It contains 4, 5, 20, or 25% of serum albumin and not less than 96% of the total protein is albumin. It may contain sodium acetyltryptophanate with or without sodium caprylate as a stabilising agent; it contains no added antimicrobial agent. It contains 130 to 160 mmol of sodium per litre. It is a practically odourless, moderately viscous, clear, brownish fluid. It should be stored in airtight containers.

Adverse Effects and Precautions

Adverse reactions to albumin infusion occur rarely and include nausea and vomiting, increased salivation, flushing, urticaria, hypotension, tachycardia, and febrile reactions. These effects usually respond to slowing or stopping the infusion. Allergic reactions, including severe anaphylactic shock, are possible. Rapid increases in circulatory volume can cause vascular overload, hypertension, haemodilution, and pulmonary oedema. Solutions containing albumin 20 or 25% are hyperosmotic and draw fluid from the extravascular compartment.

Infusion of albumin solutions is contra-indicated in patients with severe anaemia or heart failure. They should be given with caution to patients with hypertension or low cardiac reserve. Dehydrated patients may require additional fluids. Injured or postoperative patients should be observed carefully when given albumin as the rise in blood pressure may result in bleeding from previously undetected sites.

Human albumin preparations carry a risk of viral transmission. Manufacturing processes, including heating to about 60°, have reduced the risk of transmitting some viral infections.

Aluminium toxicity. Albumin solutions may contain appreciable amounts of aluminium. Marked increases in plasma-aluminium concentrations have been demonstrated in patients receiving large volumes by infusion and accumulation of aluminium may occur in patients with renal impairment.[1-3] In the UK albumin solutions with an aluminium content of less than 200 micrograms/litre are available for use in premature infants and patients undergoing dialysis.

1. Milliner DS, *et al.* Inadvertent aluminum administration during plasma exchange due to aluminum contamination of albumin-replacement solutions. *N Engl J Med* 1985; **312:** 165–7.
2. Maher ER, *et al.* Accumulation of aluminium in chronic renal failure due to administration of albumin replacement solutions. *BMJ* 1986; **292:** 306.
3. Maharaj D, *et al.* Aluminium bone disease in patients receiving plasma exchange with contaminated albumin. *BMJ* 1987; **295:** 693–6.

Critically ill patients. Volume expansion with albumin (a colloid) has been widely used in critically ill patients, although its use had never been formally tested in large controlled studies. A systematic review based on available studies up to March 1998 (relatively small, old trials that recorded only a small number of deaths) suggested that albumin was of no benefit in critically ill patients with hypovolaemia, burns, or hypoalbuminaemia, and that it might be linked to increased mortality.[1] The authors of the review stressed that these results should be treated with caution but nevertheless called for an urgent reconsideration of the use of albumin in critically ill patients.

The review was severely criticised[2] and while it was recognised that albumin had probably been overused in the past it was considered that more studies were required to define the effect of albumin on mortality.[3-5] Another review[6] found that the use of albumin did not significantly affect mortality; this meta-analysis had broader criteria and included studies that were considered to be relevant but that had been excluded by the other review.

In response to this debate, albumin 4% was compared with sodium chloride 0.9% for resuscitation in a study of 6997 hypovolaemic patients in intensive care (the SAFE study).[7] This large, randomised, double-blind study found equivalent rates of death from any cause during the 28-day study period. Survival-time during the 28 days, length of stay in the intensive care unit and in hospital, time on mechanical inhalation or renal replacement therapy, and development of organ failure were also similar. Although these two fluids seem clinically equivalent in a heterogeneous population of patients in intensive care, further study of selected groups, such as those with trauma or severe sepsis, is required.

An update to the original 1998 review included the results of SAFE. The authors maintained that patients with burns (a group excluded from the large trial) or hypoproteinaemia might still be at risk of increased mortality, and although no longer suggesting a generally increased risk, concluded that there was no evidence that albumin reduced mortality in patients with hypovolaemia. Whether highly selected groups of critically ill patients might benefit is as yet unclear.[8]

Pharmacovigilance data reported to albumin suppliers over 3 years (1998 to 2000) has also been analysed.[9] During this period of heightened awareness about possible adverse effects of albumin, due to the publication of the 1998 review, a total of 1.62×10^7 doses of 40 g had been distributed. Serious adverse effects possibly or probably related to albumin were found to be rare, and no death was classified as probably related to albumin use.

On a broader level, debate continues about the relative merits and risks of such colloid solutions, compared with those of crystalloids such as glucose or sodium chloride solutions, in the management of hypovolaemia and shock (p.1183).

1. Cochrane Injuries Group Albumin Reviewers. Human albumin administration in critically ill patients: systematic review of randomised controlled trials. *BMJ* 1998; **317:** 235–40.
2. Various. Human albumin administration in critically ill patients. *BMJ* 1998; **317:** 882–6. [Letters.]
3. Tomlin M. Albumin usage in the critically ill. *Pharm J* 1998; **261:** 193.
4. McClelland B. Albumin: don't confuse us with the facts. *BMJ* 1998; **317:** 829–30.
5. Committee on Safety of Medicines/Medicines Control Agency. The safety of human albumin. *Current Problems* 1999; **25:** 11. Available at: http://www.mhra.gov.uk/home/idcplg?IdcService=GET_FILE&dDocName=CON2023234&RevisionSelectionMethod=LatestReleased (accessed 08/06/06)
6. Wilkes MM, Navickis RJ. Patient survival after human albumin administration: a meta-analysis of randomized, controlled trials. *Ann Intern Med* 2001; **135:** 149–64.
7. The SAFE Study Investigators. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med* 2004; **350:** 2247–56.
8. The Albumin Reviewers. Human albumin solution for resuscitation and volume expansion in critically ill patients. Available in The Cochrane Database of Systematic Reviews; Issue 4. Chichester: John Wiley; 2004 (accessed 27/10/05).
9. Vincent J-L, *et al.* Safety of human albumin—serious adverse events reported worldwide in 1998–2000. *Br J Anaesth* 2003; **91:** 625–30.

Dilution. If concentrated albumin solutions are to be diluted before use, a suitable solution such as sodium chloride 0.9% or glucose 5% must be used. Albumin 25% that was erroneously diluted with water to produce a hypo-osmolar albumin 5% solution has produced severe haemolysis and renal failure in patients undergoing plasmapheresis,[1,2] including a fatality in one patient.[3]

1. Steinmuller DR. A dangerous error in the dilution of 25 percent albumin. *N Engl J Med* 1998; **338:** 1226.
2. Pierce LR, *et al.* Hemolysis and renal failure associated with use of sterile water for injection to dilute 25% human albumin solution. *Am J Health-Syst Pharm* 1998; **55:** 1057,1062, 1070.
3. Anonymous. Hemolysis associated with 25% human albumin diluted with sterile water—United States, 1994–1998. *MMWR* 1999; **48:** 157–9.

Transmission of infections. There has been concern that albumin preparations may carry a potential risk of transmission of viral and subviral particles, notably Creutzfeldt-Jakob disease. In 1993, *Pasteur-Mérieux* (one of the largest producers of blood products) withdrew all products containing albumin derived from placental blood[1] due to uncertainty regarding the adequacy of screening procedures for placentas as a source. It was considered that the agent responsible for Creutzfeldt-Jakob disease might be contained in placentas from women who have been treated with growth hormone derived from cadaver pituitaries. More recently, the production of blood products (including albumin) using plasma from UK donors has been phased out due to the possible risk of transmission of new variant Creutzfeldt-Jakob disease.

1. Anonymous. Placental-derived albumin preparations withdrawn. *WHO Drug Inf* 1994; **8:** 29–30.

Uses and Administration

Albumin is the major protein involved in maintaining colloid osmotic pressure in the blood. It also binds a number of endogenous and exogenous substances including bilirubin, steroid hormones, and many, mainly acidic, drugs.

Albumin solutions are used for plasma volume replacement and to restore colloid osmotic pressure. They have been used in conditions such as burns, severe acute albumin loss, and acute hypovolaemic shock (p.1183). They are also used as an exchange fluid in therapeutic plasmapheresis. Concentrated albumin solutions are used in neonatal hyperbilirubinaemia associated with haemolytic disease of the newborn (p.2204). They have also been suggested for short-term management of hypoproteinaemia in hepatic disease and in diuretic-resistant patients with nephrotic syndrome but are of little value in chronic hypoproteinaemias.

Albumin may be included in diagnostic preparations such as those labelled with technetium-99m (p.2055) for use as radiopharmaceuticals in scanning of the heart, lung, liver, spleen, bone marrow, veins, and lymphatic system. Albumin labelled with iodine-125 (p.2054) is used to measure blood and plasma volumes, blood circulation, and cardiac output. A suspension of albumin microspheres with perflutren (p.1488) is available for enhancing cardiac ultrasound imaging.

Recombinant forms of human albumin have been developed as excipients for vaccines and other drug products, and for the treatment of hypoalbuminaemia and hypovolaemic shock.

Albumin solutions are usually available as 4.5% or 5% solutions, which are iso-osmotic with plasma, and as 20% or 25% solutions which are hyperosmotic with respect to plasma, and cause a movement of fluid from the extravascular to the intravascular compartment. These concentrated solutions may be used undiluted or may be diluted with a suitable solution, commonly sodium chloride 0.9% or glucose 5%. Adequate hydration should be maintained and electrolytes monitored in patients receiving hyperosmotic solutions of albumin.

The amount of albumin solution given will depend upon the clinical condition of the patient and the response to treatment. The following doses have been suggested:

- acute hypovolaemic shock: an initial dose of 25 g for adults (for example, 500 mL of a 5% solution or 100 mL of a 25% solution) and up to about 1 g/kg for children
- hypoproteinaemia: a maximum of 2 g/kg daily
- neonatal hyperbilirubinaemia: 1 g/kg before exchange transfusion

The rate of infusion should be adjusted according to the indication and patient response, but in general, suggested rates of infusion are up to 5 mL/minute (5% solution) or 1 to 2 mL/minute (20% solution). In plasmapheresis the albumin infusion rate should be adjusted according to the rate of removal.

Albumin solutions should not be used for parenteral nutrition.

◊ References.

1. Nicholson JP, *et al.* The role of albumin in critical illness. *Br J Anaesth* 2000; **85:** 599–610.
2. Matejtschuk P, *et al.* Production of human albumin solution: a continually developing colloid. *Br J Anaesth* 2000; **85:** 887–95.
3. Haynes GR, *et al.* Albumin administration–what is the evidence of clinical benefit? A systematic review of randomized controlled trials. *Eur J Anaesthesiol* 2003; **20:** 771–93.
4. Mendez CM, *et al.* Albumin therapy in clinical practice. *Nutr Clin Pract* 2005; **20:** 314–20.
5. 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.
6. Kobayashi K. Summary of recombinant human serum albumin development. *Biologicals* 2006; **34:** 55–9.

Preparations

Ph. Eur.: Human Albumin Solution;
USP 31: Albumin Human.

Proprietary Preparations (details are given in Part 3)

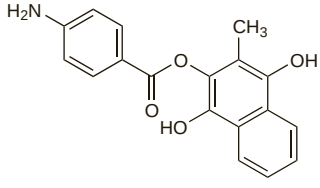
Arg.: Buminate; Zenalb†; ***Austral.:*** Albumex; ***Austria:*** Albuminativ; ***Braz.:*** Albumax†; Albuminar; Beribumin; Blaubimax; Blaubumin†; Plasbumin; Zenalb†; ***Canad.:*** Plasbumin; ***Chile:*** Plasbumin; ***Cz.:*** Flexbumin; ***Denm.:*** Octalbin; ***Fin.:*** Albuminativ; Octalbin; ***Fr.:*** Octalbine; Vialebex; ***Ger.:*** Humanalbin; ***Gr.:*** Zenalb; ***Hong Kong:*** Albuminar; Albutein; Biseko; Buminate; Kamapharm; Plasbumin; ***Indon.:*** Albapure; Alburaas; Albutein; Farmin; Fimalbumin; Octalbin; Plasbumin; ***Israel:*** Albuminar; Egg Plus; Plasbumin†; ***Ital.:*** Albital; Albuman†; Alburex; Albutein; Plasbumin; ***Jpn:*** Medway; ***Malaysia:*** Albutein; Buminate; Plasbumin†; Zenalb; ***Mex.:*** Albital†; Albumar†; Albumyn†; Biomina†; Buminate†; Hi-Bumin†; Octalbin; Probialbumin†; Seralbumin†; Vanderbumin; ***Neth.:*** Albuminativ†; Cealb; Octalbine; ***NZ:*** Albumex; ***Philipp.:*** Albumax; Albuminar; Albutein; Plasbumin; ***Pol.:*** Biseko; ***Port.:*** Flexbumin; ***Rus.:*** Plasbumin (Плазбумин); ***S.Afr.:*** Albusol; ***Singapore:*** Albutein; Plasbumin†; Zenalb; ***Spain:*** Octalbin; Plasbumin; ***Swed.:*** Albuminativ; ***Switz.:*** Albuman; ***Thai.:*** Alburaas; Albutein; Buminate; Zenalb; ***Turk.:*** Alba; Albuman; Albuminar; Cealb; Plasbumin; Zenalb; ***UK:*** Alba†; Albutein; Zenalb; ***USA:*** Albumarc; Albuminar; Albutein; Buminate; Plasbumin.

Multi-ingredient: ***Denm.:*** Pharmalgen Albumin; ***Swed.:*** Tisseel Duo Quick.

Aminaphthone

Aminaftona; Aminaftone; Aminaphtone; Aminonaphthone. 2-Hydroxy-3-methylnaphtho-1,4-hydroquinone 2-(4-aminobenzoate); 3-Methylnaphthalene-1,2,4-triol 2-(4-aminobenzoate).

$C_{18}H_{15}NO_4$ = 309.3.
CAS — 14748-94-8.



Profile

Aminaphthone is a haemostatic. Daily doses of 150 to 225 mg orally have been used.

Preparations

Proprietary Preparations (details are given in Part 3)

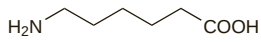
Braz.: Capilarema; ***Ital.:*** Capillarema; ***Port.:*** Capilarema; ***Spain:*** Capilarema†.

Aminocaproic Acid *(BAN, USAN, rINN)*

Acide aminocaproïque; Ácido aminocapróico; Ácido aminocaproico; Acidum aminocaproicum; Aminokaproninhappo; Aminokaprono rūgštis; Aminokapronsav; Aminokapronsyra; CL-10304; CY-116; EACA; Epsilon Aminocaproic Acid; JD-177; Kwas ε-aminokapronowy; Kyselina aminokapronová; NSC-26154. 6-Aminohexanoic acid.

Аминокапроновая Кислота

$C_6H_{13}NO_2$ = 131.2.
CAS — 60-32-2.
ATC — B02AA01.
ATC Vet — QB02AA01.



Pharmacopoeias. In *Eur.* (see p.vii) and *US.*

Ph. Eur. 6.2 (Aminocaproic Acid). A white or almost white, crystalline powder or colourless crystals. Freely soluble in water; slightly soluble in alcohol. A 20% solution in water has a pH of 7.5 to 8.0.

USP 31 (Aminocaproic Acid). A fine, white, odourless or practically odourless, crystalline powder. Soluble 1 in 3 of water and 1 in 450 of methyl alcohol; slightly soluble in alcohol; practically insoluble in chloroform and in ether; freely soluble in acids and in alkalis. Its solutions are neutral to litmus. Store in airtight containers.

Adverse Effects

Adverse effects associated with aminocaproic acid include dose-related gastrointestinal disturbances, dizziness, tinnitus, headache, nasal and conjunctival congestion, and skin rashes. Aminocaproic acid may cause muscle damage. This has usually occurred with high doses given for prolonged periods; renal failure may develop. Thrombotic complications have been reported, although they are usually a consequence of inappropriate use. If aminocaproic acid is given by rapid intravenous injection it can produce hypotension, bradycardia, and arrhythmias. There have been reports of a few patients suffering from convulsions, dry ejaculation, or cardiac and hepatic damage.

Effects on the blood. Very high doses of aminocaproic acid (36 g or more daily) have been given intravenously in the management of subarachnoid haemorrhage (see Stroke, p.1185). One study[1] reported rebleeding and excessive intra-operative bleeding and suggested that this was due to an antiplatelet effect of the aminocaproic acid. However, a comment on this report[2] pointed out that any antiplatelet effect was independent of its antifibrinolytic action and that this effect could only aggravate rebleeding, if it occurs, rather than causing it. However, early surgical intervention is now used to manage subarachnoid haemorrhage, and in a series of 307 patients treated with high-dose short-term aminocaproic acid before early surgery it was found that, compared with older reports in the literature, there was a low rate of rebleeding without an apparent increase in adverse effects.[3]

1. Glick R, *et al.* High dose ε-aminocaproic acid prolongs the bleeding time and increases rebleeding and intraoperative hemorrhage in patients with subarachnoid hemorrhage. *Neurosurgery* 1981; **9:** 398–401.
2. Kassell NF. Comment. *Neurosurgery* 1981; **9:** 401.
3. Leipzig TJ, *et al.* Reducing the risk of rebleeding before early aneurysm surgery: a possible role for antifibrinolytic therapy. *J Neurosurg* 1997; **86:** 220–5.

Effects on the kidneys. Adverse renal effects of aminocaproic acid are rare but have included renal arterial thrombosis, glomerular capillary thrombosis, and renal pelvic or ureteral obstruction caused by upper urinary tract thrombosis.[1] Cases of acute renal failure associated with myopathy are described under Effects on the Muscles, below.

1. Manjunath G, *et al.* Epsilon-aminocaproic acid and renal complications: case report and review of the literature. *Clin Nephrol* 2002; **58:** 63–7.

Effects on the muscles. There have been cases of reversible myopathy,[1-4] associated with daily doses of aminocaproic acid ranging from 10 to 49 g and treatment durations of about 1 to 3 months. In some patients myoglobinuria or acute tubular necrosis also occurred. Suggested mechanisms for the reaction have included a direct dose-related effect on the muscle fibre[2] or a defect in aerobic energy provision induced by aminocaproic acid.[3]

1. Brown JA, *et al.* Myopathy induced by epsilon-aminocaproic acid. *J Neurosurg* 1982; **57:** 130–4.
2. Vanneste JAL, van Wijngaarden GK. Epsilon-aminocaproic acid myopathy. *Eur Neurol* 1982; **21:** 242–8.
3. Van Renterghem D, *et al.* Epsilon amino caproic acid myopathy: additional features. *Clin Neurol Neurosurg* 1984; **86:** 153–7.
4. Seymour BD, Rubinger M. Rhabdomyolysis induced by epsilon-aminocaproic acid. *Ann Pharmacother* 1997; **31:** 56–8.

Precautions

As for Tranexamic Acid, p.1081.

The range of adverse effects that have been noted with aminocaproic acid indicates that caution is required in patients with renal or cardiac disorders. Should treatment be prolonged, it is advisable to monitor creatine phosphokinase values for signs of muscle damage.

Renal impairment. High anion gap metabolic acidosis developed in a 65-year-old woman with sepsis and acute renal failure who received aminocaproic acid for a haemorrhagic coagulopathy.[1] The acidosis improved temporarily after haemodialysis and resolved on withdrawal of aminocaproic acid and systemic alkalinisation. Although the dose of aminocaproic acid had been reduced because of renal impairment, it was suggested that more conservative dosing and close monitoring may be indicated in such patients. Hyperkalaemia has been attributed to the use of aminocaproic acid in a few patients with chronic renal failure.[2]

1. Budris WA, *et al.* High anion gap metabolic acidosis associated with aminocaproic acid. *Ann Pharmacother* 1999; **33:** 308–11.
2. Nzerue CM, Falana B. Refractory hyperkalaemia associated with use of epsilon-aminocaproic acid during coronary bypass in a dialysis patient. *Nephrol Dial Transplant* 2002; **17:** 1150–1.

Interactions

Retinoids. Aminocaproic acid should be used with caution in patients receiving oral *tretinoin* (see Antifibrinolytics, p.1619).

Pharmacokinetics

Aminocaproic acid is readily absorbed when given orally and peak plasma concentrations are reached within 2 hours. It is widely distributed and is rapidly excreted in the urine, mainly unchanged, with a terminal elimination half-life of about 2 hours.

Uses and Administration

Aminocaproic acid is an antifibrinolytic used similarly to tranexamic acid (p.1081) in the treatment and prophylaxis of haemorrhage associated with excessive fibrinolysis. It has also been used in the prophylaxis of hereditary angioedema (below).

A plasma concentration of about 130 micrograms/mL is considered to be necessary for effective inhibition of fibrinolysis and the recommended dosage schedules are aimed at producing and maintaining this concentration for as long as is necessary. For the treatment and prophylaxis of **haemorrhage**, aminocaproic acid may be given orally in an initial dose of 4 to 5 g, followed by 1 to 1.25 g every hour. Alternatively, the same dose may be given intravenously as a 2% solution; the initial dose (4 to 5 g) should be given over one hour followed by a continuous infusion of 1 g/hour. Up to 8 hours of treatment is often sufficient. Should treatment need to be extended, then the maximum dose over 24 hours should not normally exceed 24 g.

In patients with **haemophilia** (p.1048) who undergo dental extraction, aminocaproic acid has been given in an initial dose of 6 g orally immediately after the procedure, followed by 6 g orally every 6 hours for up to 10 days.

Care is required when aminocaproic acid is used in patients with renal impairment and dosage should be reduced.

Hereditary angioedema. In the management of hereditary angioedema (p.1081), antifibrinolytic drugs may be used as an alternative to androgens for the prophylaxis of attacks. The usual oral dose of aminocaproic acid in such patients is 1 g three or four times daily. It has also been used intravenously for acute attacks, and anecdotal reports suggest it may be modestly helpful, but there is no published evidence suggesting significant benefit.[1]

1. Zuraw BL. Current and future therapy for hereditary angioedema. *Clin Immunol* 2005; **114:** 10–16.

Preparations

USP 31: Aminocaproic Acid Injection; Aminocaproic Acid Syrup; Aminocaproic Acid Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Ipsilon; ***Austral.:*** Amicar†; ***Braz.:*** Ipsilon; ***Canad.:*** Amicar†; ***Fr.:*** Hexalense†; ***Hung.:*** Aceprami; ***India:*** Hemocid; ***Ital.:*** Caprolisin; ***Mex.:*** Amicar†; ***NZ:*** Amicar†; ***Port.:*** Epsicaprom; ***Spain:*** Caproamin; ***USA:*** Amicar; ***Venez.:*** Caproamin.

Multi-ingredient: ***Braz.:*** Eaca Balsamico; Expectovac†; Ginurovac†; ***Spain:*** Caprofides Hemostatico.