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)

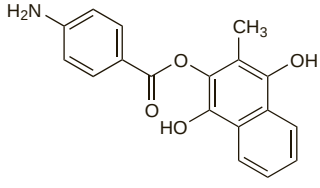
Arg.: Buminate; Zenalb†; ***Austral.:*** Albumex; ***Austria:*** Albuminativ; ***Braz.:*** Albumax†; Albuminar; Beribumin; Blaubimax; Blaubumin†; Plasbumin; Zenalb†; ***Canad.:*** Plasbumin; ***Chile:*** Plasbumin; ***Cz.:*** Flexbumin; ***Denm.:*** Octalbin; ***Fin.:*** Albuminativ; Octalbin; ***Fr.:*** 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)

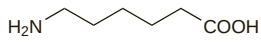
Braz.: Capilarema; ***Ital.:*** Capillarema; ***Port.:*** Capilarema; ***Spain:*** Capilarema†.

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)

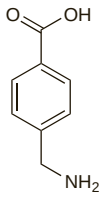
Arg.: Ipsilon; ***Austral.:*** Amicar†; ***Braz.:*** Ipsilon; ***Canad.:*** Amicar†; ***Fr.:*** Hexalense†; ***Hung.:*** Aceprami; ***India:*** Hemocid; ***Ital.:*** Caprolisin; ***Mex.:*** Amicar†; ***NZ:*** Amicar†; ***Port.:*** Epsicaprom; ***Spain:*** Caproamin; ***USA:*** Amicar; ***Venez.:*** Caproamin.

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

Aminomethylbenzoic Acid

Aminometilbenzoico, ácido; PAMBA. 4-Aminomethylbenzoic acid.

$C_8H_9NO_2$ = 151.2.
CAS — 56-91-7.
ATC — B02AA03.
ATC Vet — QB02AA03.



Profile

Aminomethylbenzoic acid is an antifibrinolytic with actions and uses similar to those of tranexamic acid (p.1080). It is given orally in typical doses of 300 mg to 1 g daily, in 3 or 4 divided doses; it is also given by intramuscular injection, or intravenously by slow injection or infusion.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Gumbix†; ***Cz.:*** Gumbix†; Pamba; ***Ger.:*** Gumbix; Pamba.

Ancestim *(USAN, rINN)*

Ancestimum; r-metHuSCF; SCF; Stem Cell Factor. *N*-L-Methionyl-1–165-haematopoietic cell growth factor KL (human clone V19.8:hSCF162), dimer.

Анцестим
CAS — 163545-26-4.
ATC — L03AA12.
ATC Vet — QL03AA12.

Adverse Effects and Precautions

Injection site reactions commonly occur with the use of ancestim. Other skin reactions, including pruritus, rash, and urticaria, are less frequent. Systemic hypersensitivity reactions are also common and may be life-threatening. Premedication with antihistamines (both H_1- and H_2-antagonists) and an inhaled beta$_2$ agonist bronchodilator should be used, and the patient observed for at least an hour after ancestim is given. Tachycardia and respiratory symptoms including pharyngitis, dyspnoea, and cough, have also been reported.

Ancestim should not be given in the period from 24 hours before to 24 hours after a dose of cytotoxic chemotherapy or radiotherapy.

Uses and Administration

Ancestim is a recombinant human stem cell factor. It is used with filgrastim (p.1070) to mobilise peripheral blood progenitor cells that are to be collected by apheresis harvest and used for autologous transplantation. The dose of ancestim is 20 micrograms/kg daily by subcutaneous injection; the injections of ancestim and filgrastim must be given at separate sites.

◊ References.

1. Chin-Yee IH, *et al.* Optimising parameters for peripheral blood leukapheresis after r-metHuG-CSF (filgrastim) and r-metHuSCF (ancestim) in patients with multiple myeloma: a temporal analysis of CD34(+) absolute counts and subsets. *Bone Marrow Transplant* 2002; **30:** 851–60.
2. Prosper F, *et al.* Mobilization of peripheral blood progenitor cells with a combination of cyclophosphamide, r-metHuSCF and filgrastim in patients with breast cancer previously treated with chemotherapy. *Leukemia* 2003; **17:** 437–41.
3. To LB, *et al.* Successful mobilization of peripheral blood stem cells after addition of ancestim (stem cell factor) in patients who had failed a prior mobilization with filgrastim (granulocyte colony-stimulating factor) alone or with chemotherapy plus filgrastim. *Bone Marrow Transplant* 2003; **31:** 371–8.
4. da Silva MG, *et al.* Ancestim (recombinant human stem cell factor, SCF) in association with filgrastim does not enhance chemotherapy and/or growth factor-induced peripheral blood progenitor cell (PBPC) mobilization in patients with a prior insufficient PBPC collection. *Bone Marrow Transplant* 2004; **34:** 683–91.

Preparations

Proprietary Preparations (details are given in Part 3)
Austral.: Stemgen; ***Canad.:*** Stemgen; ***NZ:*** Stemgen.

Antithrombin III *(BAN, rINN)*

Antithrombin III Human; Antithrombine III; Antithrombinum III; Antitrombiini III; Antitrombin III; Antitrombina III; Antitrombina III humana; Antytrombina III; AT-III; Cofactor I de la heparina; Heparin Cofactor; Heparin Cofactor I; Major Antithrombin.

Антитромбин III
CAS — 52014-67-2.
ATC — B01AB02.
ATC Vet — QB01AB02.

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

Ph. Eur. 6.2 (Human Antithrombin III Concentrate; Antithrombinum III Humanum Densatum). A preparation of a glycoprotein fraction obtained from human plasma that inactivates thrombin in the presence of an excess of heparin. The plasma 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. The method of preparation includes a step or steps that have been shown to remove or to inactivate known agents of infection. The antithrombin III concentrate is passed through a bacteria-retentive filter, distributed into sterile containers, and immediately frozen. The preparation is freeze-dried and the containers sealed under vacuum or in an atmosphere of inert gas. No antimicrobial preservative is added but a suitable stabiliser (such as albumin) is permitted. When reconstituted in the volume of solvent stated on the label, the resulting solution contains not less than 25 international units of antithrombin III per mL.
A white or almost white, hygroscopic, friable solid or powder. Store in airtight containers. Protect from light.

USP 31 (Antithrombin III Human). A glycoprotein, which is the major inhibitor of thrombin and other activated clotting factors, including factors IX, X, XI, and XII, and the cofactor through which heparin exerts its effect. It is obtained from human plasma of healthy donors who must, as far as can be ascertained, be free from detectable agents of infection transmissible by transfusion of blood or blood derivatives. The method of manufacturing includes steps that have been shown to remove or inactivate known agents of infection. The antithrombin III concentrate is passed through a bacteria-retentive filter, filled aseptically into its final, sterile containers, and immediately frozen. It is then freeze-dried, and the containers are closed under vacuum. No antimicrobial preservative is added at any stage of production. When reconstituted in the recommended volume of diluent, the pH is between 6.0 and 7.5, and the potency is not less than 25 USP units of antithrombin III per mL.
Store at a temperature of 2° to 8°, excursions permitted up to 25°. Protect from light.

Antithrombin Alfa *(USAN, rINN)*

Antithrombine Alfa; Antithrombinum Alfa; Antitrombina alfa; Human Antithrombin III from the milk of transgenic goats (glycoform alfa); Recombinant Human Antithrombin.

Антитромбин Альфа
CAS — 84720-88-7.

Units

The potency of antithrombin III is expressed in international units and preparations may be assayed using the second International Standard for antithrombin concentrate (1997); each ampoule contains 4.7 international units of functional activity and 5.1 international units of antigenic activity.

One USP unit is described as the amount of antithrombin III that forms a complex with 1 unit of thrombin at 25° in the presence of heparin at a pH of 8.4. Since assays of antithrombin III are carried out at 37°, it is unclear whether USP units and international units are precisely equivalent, but in practice US preparations, like those elsewhere, appear to have their potency defined in international units.

The potency of antithrombin alfa is also expressed in international units.

Adverse Effects and Precautions

Adverse effects of antithrombin III include flushing, headache, dizziness, chest tightness, nausea, a foul taste in the mouth, chills, and cramps. These can be controlled by slowing or stopping the infusion. Allergic reactions occur rarely.

Human plasma-derived antithrombin III preparations carry a risk of viral transmission. Manufacturing processes, including heating to about 60°, have reduced the risk of transmitting some viral infections. Antithrombin alfa is produced in the milk of transgenic goats, and should not be used in patients who are hypersensitive to goat proteins or goat milk components.

Uses and Administration

Antithrombin III is a protein in plasma; it is the major endogenous inhibitor of thrombin and other activated clotting factors including factors IX, X, XI, and XII (p.1045), and is the cofactor through which heparin (p.1303) exerts its effect. Genetic and acquired deficiency of antithrombin III occurs and is associated with susceptibility to thromboembolic disorders.

Human plasma-derived antithrombin III is given intravenously to patients with antithrombin III deficiency in the treatment of thromboembolism and for prophylaxis associated with surgical and obstetric procedures. The aim of therapy is to restore plasma-antithrombin III concentrations to at least 80% of normal. The dose, frequency, and duration of therapy are individualised for each patient taking into account the patient's pretreatment concentration and presence of active coagulation. A usual initial dose is about 30 to 50 international units/kg.

Antithrombin alfa is used similarly in the prophylaxis of venous thromboembolism in surgical patients with congenital antithrombin III deficiency. The dose is individualised, but a usual initial dose is about 20 to 25 international units/kg given as an intravenous infusion over 15 minutes, followed by a maintenance infusion of about 4 to 5 international units/kg per hour.

◊ References.

1. Bucur SZ, *et al.* Uses of antithrombin III concentrate in congenital and acquired deficiency states. *Transfusion* 1998; **38:** 481–98.
2. Roemisch J, *et al.* Antithrombin: a new look at the actions of a serine protease inhibitor. *Blood Coag Fibrinol* 2002; **13:** 657–70.
3. Konkle BA, *et al.* Use of recombinant human antithrombin in patients with congenital antithrombin deficiency undergoing surgical procedures. *Transfusion* 2003; **43:** 390–4.

Septicaemia. Antithrombin III has been used in septicaemia (p.190) in an attempt to manage the pro-coagulant state that occurs. Initial small studies reported a reduction in mortality[1] but a large controlled study[2] (KyberSept) found that treatment with antithrombin III had no effect on 28-day mortality. A further small observational study and meta-analysis also found no benefit from the use of antithrombin III in septicaemia.[3] These studies had used antithrombin III for less than 7 days, and a small study[4] in surgical patients with septicaemia found that 14 days of treatment with antithrombin III did improve measures of coagulation and fibrinolysis, the changes being most evident in the second week of therapy. However, the study was not large enough to test effects on mortality. Subsequent analysis of data from the KyberSept study appeared to show that 28-day mortality was in fact reduced in patients who had not been given heparin as well as antithrombin III;[5] combined use increased the risk of bleeding and apparently decreased the benefits of treatment with antithrombin III.

1. Eisele B, *et al.* Antithrombin III in patients with severe sepsis: a randomized, placebo-controlled, double-blind multicenter trial plus a meta-analysis on all randomized, placebo-controlled, double-blind trials with antithrombin III in severe sepsis. *Intensive Care Med* 1998; **24:** 663–72.
2. Warren BL, *et al.* KyberSept Trial Study Group. High-dose antithrombin III in severe sepsis: a randomized controlled trial. *JAMA* 2001; **286:** 1869–78. Correction. *ibid.* 2002; **287:** 192.
3. Messori A, *et al.* Antithrombin III in patients admitted to intensive care units: a multicenter observational study. *Crit Care* 2002; **6:** 447–51.
4. Hoffmann JN, *et al.* Effect of long-term and high-dose antithrombin supplementation on coagulation and fibrinolysis in patients with severe sepsis. *Crit Care Med* 2004; **32:** 1851–9.
5. Hoffmann JN, *et al.* The KyberSept Investigators. Benefit/risk profile of high-dose antithrombin in patients with severe sepsis treated with and without concomitant heparin. *Thromb Haemost* 2006; **95:** 850–6.

Veno-occlusive disease. There is some evidence[1] from case reports and small studies that antithrombin III may have a beneficial effect on veno-occlusive disease associated with haematopoietic stem cell transplantation (p.1811).

1. Ibrahim RB, *et al.* Anti-thrombin III in the management of hematopoietic stem-cell transplantation–associated toxicity. *Ann Pharmacother* 2004; **38:** 1053–9.

Preparations

Ph. Eur.: Human Antithrombin III Concentrate;
USP 31: Antithrombin III Human.

Proprietary Preparations (details are given in Part 3)
Arg.: Kybernin P; ***Austral.:*** Thrombotrol-VF; ***Austria:*** Atenativ; Kybernin P; Thrombhibin; ***Braz.:*** Kybernin P; ***Canad.:*** Thrombate; ***Cz.:*** Anbinex; ATryn; Kybernin P; ***Denm.:*** Atenativ; ***Fin.:*** Atenativ; ***Fr.:*** Aclotine; ***Ger.:*** Anbinex; AT III; Atenativ; Kybernin; ***Gr.:*** Atenativ; Kybernin P; ***Hung.:*** Atenativ; Kybernin P; ***Indon.:*** Kybernin P; ***Ital.:*** Anbin; Atenativ; Kybernin P; ***Jpn:*** Neuart; ***Mex.:*** Atend; Octatit; ***Neth.:*** Atenativ; ***Norw.:*** Atenativ; ***NZ:*** Thrombotrol-VF; ***Port.:*** Atenativ; ATryn; ***Spain:*** Anbinex; Atenativ; Kybernin P; ***Swed.:*** Atenativ; ***Switz.:*** Atenativ; Kybernin; ***Turk.:*** Kybernin P; ***UK:*** ATryn; ***USA:*** Thrombate III.