Preparations

BP 2008: Acebutolol Capsules; Acebutolol Tablets;
USP 31: Acebutolol Hydrochloride Capsules.

Proprietary Preparations (details are given in Part 3)
Belg.: Sectral; ***Canad.:*** Monitan; Rhotral; Sectral; ***Chile:*** Beloc; Grifobutol; ***Cz.:*** Acecor; Apo-Acebutol; Sectral; ***Denm.:*** Diasectral; ***Fin.:*** Diasectral; Espesil; ***Fr.:*** Sectral; ***Ger.:*** Prent; ***Hong Kong:*** Sectral; ***Irl.:*** Sectral†; ***Israel:*** Sectral; ***Ital.:*** Prent; Sectral; ***Malaysia:*** Sectral†; ***Neth.:*** Sectral; ***NZ:*** ACB; ***Pol.:*** Abutol; Sectral; ***Port.:*** Prent; ***S.Afr.:*** Butobloc; Sectral; ***Singapore:*** ACB†; Sectral; ***Spain:*** Sectral†; ***Switz.:*** Sectral†; ***Turk.:*** Prent; ***UK:*** Sectral; ***USA:*** Sectral; ***Venez.:*** Flebutol†.

Multi-ingredient: ***Belg.:*** Sectrazide; ***Ger.:*** Sali-Prent; Tredalat; ***Indon.:*** Sectrazide; ***Neth.:*** Secadrex†; ***Spain:*** Secadrex†; ***UK:*** Secadrex†.

Acenocoumarol *(BAN, rINN)*

Acénocoumarol; Acenocoumarolum; Acenocumarin; Acenocumarol; Acenokumarol; Asenokumarol; G-23350; Nicoumalone; Nikumalon. (*RS*)-4-Hydroxy-3-[1-(4-nitrophenyl)-3-oxobutyl]-coumarin.

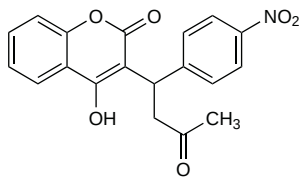
Аценокумарол

$C_{19}H_{15}NO_6$ = 353.3.

CAS — 152-72-7.

ATC — B01AA07.

ATC Vet — QB01AA07.



Pharmacopoeias. In *Br.* and *Pol.*
BP 2008 (Acenocoumarol). An almost white to buff-coloured odourless or almost odourless powder. It exhibits polymorphism. Practically insoluble in water and in ether; slightly soluble in alcohol and in chloroform; dissolves in aqueous solutions of alkali hydroxides.

Adverse Effects, Treatment, and Precautions

As for Warfarin Sodium, p.1425.

Effects on the fetus. In a group of women who received acenocoumarol for anticoagulant prophylaxis of mechanical heart valves during pregnancy,[1] fetal loss occurred in 13 of 61 pregnancies where oral anticoagulation was continued during the first trimester. Apart from 1 case of hydrocephalus no malformations were reported in the remaining neonates.

1. Meschengieser SS, *et al.* Anticoagulation in pregnant women with mechanical heart valve prostheses. *Heart* 1999; **82:** 23–6.

Interactions

The interactions associated with oral anticoagulants are discussed in detail under warfarin (p.1427). Specific references to interactions involving acenocoumarol can be found there under the headings for the following drug groups: analgesics; antiarrhythmics; antibacterials; antidepressants; antifungals; antigout drugs; antihistamines; antineoplastics; antiplatelets; antivirals; diuretics; gastrointestinal drugs; immunosuppressants; lipid regulating drugs; sex hormones; and vaccines.

Pharmacokinetics

Acenocoumarol is readily absorbed from the gastrointestinal tract and is excreted chiefly in the urine mainly as metabolites. It is extensively bound to plasma proteins. Figures reported for elimination half-life vary; UK licensed product information gives a range of 8 to 11 hours. Acenocoumarol crosses the placenta; only small quantities have been detected in breast milk. It is given as a racemic mixture; the *R*-isomer is more potent. The stereo-isomers have different pharmacokinetics. Metabolism of the *S*-isomer is mediated mainly by the cytochrome P450 isoenzyme CYP2C9, which shows genetic polymorphism; other isoenzymes as well are involved in the metabolism of the *R*-isomer.

◊ References.

1. Ufer M. Comparative pharmacokinetics of vitamin K antagonists: warfarin, phenprocoumon and acenocoumarol. *Clin Pharmacokinet* 2005; **44:** 1227–46.

Uses and Administration

Acenocoumarol is an oral coumarin anticoagulant with actions similar to those of warfarin (p.1432). It is used in the management of thromboembolic disorders (p.1187). The usual dose is 4 to 12 mg on the first day and 4 to 8 mg on the second day; subsequent maintenance doses range from 1 to 8 mg depending on the response. Acenocoumarol is given in a single dose at the same time every day.

Preparations

BP 2008: Acenocoumarol Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Acenotromb; Antitrom; Azecar; Cumarol; Fortonol; Saxion; Sintrom; ***Austria:*** Sintrom; ***Belg.:*** Sintrom; ***Canad.:*** Sintrom; ***Chile:*** Acenox; Coarol; Isquelium; Neo-Sintrom; ***Fr.:*** Mini-sintrom; Sintrom; ***Gr.:*** Sintrom; ***Hung.:*** Syncumar; ***India:*** Acitrom; ***Israel:*** Sintrom; ***Ital.:*** Sintrom; ***Mex.:*** Sintrom; ***Neth.:*** Sintrom Mitis; ***Pol.:*** Sintrom; Syncumar; ***Port.:*** Sintrom; ***Spain:*** Sintrom; ***Switz.:*** Sintrom; ***UK:*** Sinthrome.

Acetyldigoxin

Acetildigoxina; Acetyldigoxin-beta; Acetyldigoxinum; β-Acetyldigoxinum; Acetyldigoxinum Beta; β-Acetylodigoksyna; Asetyylidigoksiini; Desglucolanatoside C. 3β-[(*O*-3-*O*-Acetyl-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-*O*-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-2,6-dideoxy-β-D-*ribo*-hexopyranosyl)oxy]-12β,-14-dihydroxy-5β,14β-card-20(22)-enolide (α-acetyldigoxin); 3β-[(*O*-4-*O*-Acetyl-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-*O*-2,6-dideoxy-β-D-*ribo*-hexopyranosyl-(1→4)-2,6-dideoxy-β-D-*ribo*-hexopyranosyl)oxy]-12β,14-dihydroxy-5β,14β-card-20(22)-enolide (β-acetyldigoxin).

$C_{43}H_{66}O_{15}$ = 823.0.

CAS — 5511-98-8 (α-acetyldigoxin); 5355-48-6 (β-acetyldigoxin).

ATC — C01AA02.

ATC Vet — QC01AA02.

Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (β-Acetyldigoxin). A white or almost white powder. Practically insoluble in water; slightly soluble in alcohol; sparingly soluble in dichloromethane. Protect from light.

Profile

Acetyldigoxin is a cardiac glycoside with positive inotropic activity. It has the general properties of digoxin (p.1259) and has been used similarly in the management of some cardiac arrhythmias (p.1160) and in heart failure (p.1165). Usual oral maintenance doses for the β-isomer are 200 to 400 micrograms daily; the α-isomer has also been used.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Corotal; Lanatilin; Novodigal; ***Ger.:*** Digostada; Digotab; Digox; Digoxin "Didier"†; Novodigal; Stillacor; ***Ital.:*** Cardioreg†.

Multi-ingredient: ***Austria:*** Digi-Aldopur; Gladixol.

Acipimox *(BAN, rINN)*

Acipimoxum; Asipimoks; Asipimoksi; K-9321. 5-Methylpyrazine-2-carboxylic acid 4-oxide.

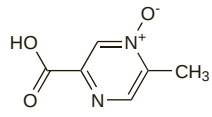
Аципимокс

$C_6H_6N_2O_3$ = 154.1.

CAS — 51037-30-0.

ATC — C10AD06.

ATC Vet — QC10AD06.



Adverse Effects and Precautions

Acipimox may cause peripheral vasodilatation resulting in flushing, itching, and a sensation of heat. Rash and erythema may occur. Gastrointestinal disturbances including heartburn, epigastric pain, nausea, and diarrhoea have been reported, as well as headache, malaise, myalgia, myositis, arthralgia, and dry eye. Urticaria, angioedema, and bronchospasm may occur rarely.

Acipimox is contra-indicated in patients with peptic ulcer disease. It should be used with caution in renal impairment.

Incidence of adverse effects. In a study involving 3009 hyperlipidaemic patients with type 2 diabetes,[1] adverse effects associated with acipimox occurred in 8.8%, resulting in withdrawal in 5.5% of patients. The most frequent adverse effects involved the skin (57.6%), gastrointestinal tract (25.8%), and CNS (9.7%). Labial oedema occurred in 3 cases and an urticarial eruption, collapse, and dyspnoea in another. The incidence of adverse effects was almost twice as high in females as in males, the difference being mainly due to a greater incidence of flushing, pruritus, and skin rashes. The incidence was not affected by age. There was a mean 15.3% reduction in fasting blood-glucose concentrations and an 8.5% reduction in glycosylated haemoglobin during treatment with acipimox.

1. Lavezzari M, *et al.* Results of a phase IV study carried out with acipimox in type II diabetic patients with concomitant hyperlipoproteinaemia. *J Int Med Res* 1989; **17:** 373–80.

Pharmacokinetics

Acipimox is rapidly and completely absorbed from the gastrointestinal tract and peak plasma concentrations occur within 2 hours. It does not bind to plasma proteins and the plasma half-life is about 2 hours. It is not significantly metabolised and is excreted in the urine, largely unchanged.

Uses and Administration

Acipimox is a lipid regulating drug related to nicotinic acid (p.1957). It is used to reduce cholesterol and triglycerides in the management of hyperlipidaemias (see Action, below), including type IIa, IIb, or IV hyperlipoproteinaemias.

Acipimox is given orally in a usual dose of 250 mg two or three times daily, taken with meals. Doses of up to 1200 mg daily have been used. The dose should be adjusted in renal impairment (see below).

Action. Acipimox is used in the management of hyperlipidaemias (p.1169); it is a derivative of nicotinic acid and has similar effects on plasma lipoproteins but is better tolerated.[1] Its primary action is inhibition of lipolysis, leading to a reduction in circulating free fatty acids and consequently a reduction in very-low-density lipoprotein (VLDL) production in the liver. This results in a reduction of triglycerides, particularly in patients with hypertriglyceridaemia;[2] there may also be a decrease in low-density lipoprotein (LDL)-cholesterol and total cholesterol, and an increase in high-density lipoprotein (HDL)-cholesterol. Similar effects have been reported in patients with mixed hyperlipoproteinaemias, although the reduction of triglycerides and LDL-cholesterol was not significant.[3]

Reduction of free fatty acids by acipimox has a number of other physiological effects that have been utilised. Insulin secretion and sensitivity may be modified, and acipimox has been tried in type 2 diabetes mellitus; it improves plasma lipids and may also reduce blood-glucose concentrations,[4] and has been of benefit in patients with type A insulin resistance.[5] Beneficial effects have also been reported[6] in patients with HIV-associated lipodystrophy and insulin resistance. Growth hormone secretion is stimulated in obese subjects, and acipimox has been used in the investigation of growth hormone disorders.[7] There is also an increase in glucose uptake by the heart, and acipimox has been used to enhance myocardial imaging in ^{18}F-fluorodeoxyglucose positron-emission tomography.[8]

1. Tornvall P, Walldius G. A comparison between nicotinic acid and acipimox in hypertriglyceridaemia—effects on serum lipids, lipoproteins, glucose tolerance and tolerability. *J Intern Med* 1991; **230:** 415–21.
2. Ball MJ, *et al.* Acipimox in the treatment of patients with hyperlipidaemia: a double blind trial. *Eur J Clin Pharmacol* 1986; **31:** 201–4.
3. Otto C, *et al.* Effects of acipimox on haemorheology and plasma lipoproteins in patients with mixed hyperlipoproteinaemia. *Br J Clin Pharmacol* 1998; **46:** 473–8.
4. Lavezzari M, *et al.* Results of a phase IV study carried out with acipimox in type II diabetic patients with concomitant hyperlipoproteinaemia. *J Int Med Res* 1989; **17:** 373–80.
5. Kumar S, *et al.* Suppression of non-esterified fatty acids to treat type A insulin resistance syndrome. *Lancet* 1994; **343:** 1073–4.
6. Hadigan C, *et al.* Inhibition of lipolysis improves insulin sensitivity in protease inhibitor-treated HIV-infected men with fat redistribution. *Am J Clin Nutr* 2003; **77:** 490–4.
7. Cordido F, *et al.* Effect of acute pharmacological reduction of plasma free fatty acids on growth hormone (GH) releasing hormone-induced GH secretion in obese adults with and without hypopituitarism. *J Clin Endocrinol Metab* 1998; **83:** 4350–4.
8. Knuuti MJ, *et al.* Enhancement of myocardial [fluorine-18]fluorodeoxyglucose uptake by a nicotinic acid derivative. *J Nucl Med* 1994; **35:** 989–98.

Administration in renal impairment. Acipimox is contra-indicated in patients with a creatinine clearance below 30 mL/minute. In patients with creatinine clearance between 30 and 60 mL/minute, the interval between doses should be increased.

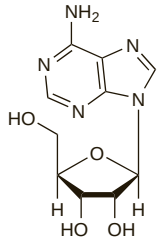
Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Olbetam; ***Belg.:*** Olbetam; ***Braz.:*** Olbetam†; ***Chile:*** Olbetam; ***Denm.:*** Olbetam; ***Ger.:*** Olbemox†; ***Gr.:*** Olbetam; ***Hong Kong:*** Olbetam; ***Hung.:*** Olbetam; ***Irl.:*** Olbetam†; ***Israel:*** Olbetam; ***Ital.:*** Olbetam; ***Mex.:*** Olbetam†; ***Neth.:*** Nedios; Olbetam; ***NZ:*** Olbetam; ***S.Afr.:*** Olbetam; ***Singapore:*** Olbetam; ***Switz.:*** Olbetam; ***Thai.:*** Olbetam; ***UK:*** Olbetam.

Adenosine *(BAN, USAN)*

Adenocin; Adenosiini; Adenosin; Adenosina; Adénosine; Adenosinum; Adenozin; Adenozinas; Adenozyna; SR-96225; SUNY-4001. 6-Amino-9-β-D-ribofuranosyl-9*H*-purine.

Аденозин
$C_{10}H_{13}N_5O_4$ = 267.2.
CAS — 58-61-7.
ATC — C01EB10.
ATC Vet — QC01EB10.



Pharmacopoeias. In *Eur.* (see p.vii) and *US*.
Ph. Eur. 6.2 (Adenosine). A white, or almost white, crystalline powder. Slightly soluble in water; soluble in hot water; practically insoluble in alcohol and in dichloromethane; dissolves in dilute mineral acids.
USP 31 (Adenosine). A white, odourless crystalline powder. Slightly soluble in water; practically insoluble in alcohol. Store in airtight containers. Protect from light.

Stability. Adenosine was found to be stable[1] when it was mixed with glucose 5%, lactated Ringer's, sodium chloride 0.9%, or a mixture of glucose 5% and lactated Ringer's and stored in polypropylene syringes or PVC bags.

1. Ketkar VA, *et al.* Stability of undiluted and diluted adenosine at three temperatures in syringes and bags. *Am J Health-Syst Pharm* 1998; **55:** 466–70.

Adverse Effects, Treatment, and Precautions

Adverse effects of adenosine are usually transient, lasting less than a minute, due to its very short plasma half-life. They include nausea, lightheadedness, flushing, headache, angina-like chest pain, apprehension, and dyspnoea. Bronchospasm has been reported. Like other antiarrhythmics, adenosine may worsen arrhythmias. Bradycardia and heart block have been reported. Adenosine is a vasodilator and reduces blood pressure; the larger doses given by intravenous infusion may rarely produce significant hypotension and reflex tachycardia. Infusion may also be associated with abdominal, throat, neck, and jaw discomfort. Treatment is rarely needed for adverse effects but in persistent cases aminophylline or theophylline may be given.

Adenosine is contra-indicated in patients with second- or third-degree AV block or in those with sick sinus syndrome (unless they have a pacemaker) and should be avoided or used with caution in patients with QT prolongation since torsade de pointes has occurred very rarely. It is also contra-indicated in asthmatic subjects and should be used with caution in patients with obstructive pulmonary disease. Intravenous infusion of adenosine should be used with caution in patients who may develop hypotensive complications such as those with autonomic dysfunction, pericarditis, or stenotic valvular heart disease. Patients with recent heart transplantation may have increased sensitivity to the cardiac effects of adenosine.

◊ Use of the *University of Wisconsin solution* [UW Solution; Belzer UW Solution (commercially available as *Viaspan*)] for the hypothermic storage of kidneys before transplantation has been associated with bradycardia, prolonged PR intervals, and heart block.[1,2] The solution contains hetastarch, allopurinol, glutathione, and adenosine. The adenosine was considered to be the arrhythmogenic factor. Some centres had used the solution to flush kidneys before implantation,[2] a use for which it was never intended.[3] When used properly the adenosine in solution is catabolised to hypoxanthine and inosine, which do not cause cardiac problems, but this takes some time in hypothermic conditions.[3]

1. Prien T, *et al.* Bradyarrhythmia with University of Wisconsin preservation solution. *Lancet* 1989; **i:** 1319–20.
2. Vanrenterghem Y, *et al.* University of Wisconsin preservation solute and bradyarrhythmia. *Lancet* 1989; **ii:** 745.
3. Belzer FO. Correct use of University of Wisconsin preservation solution. *Lancet* 1990; **335:** 362.

Effects on the heart. Like most antiarrhythmics, adenosine can worsen arrhythmias, and both bradyarrhythmias and tachyarrhythmias have been reported.[1] Atrial fibrillation may develop in patients given adenosine for paroxysmal supraventricular tachycardia, and in a prospective study[2] occurred in 12% of 200 patients. Although most arrhythmias are of minor importance, ventricular arrhythmias and haemodynamic compromise have been reported[3,4] in patients given adenosine for presumed supraventricular tachycardia who were later discovered to have Wolff-Parkinson-White syndrome. Fatal cardiac arrest has also occurred[5] after the use of adenosine for arrhythmias in 2 patients with underlying cardiopulmonary disorders.

There have also been reports[6,7] of myocardial infarction in patients with ischaemic heart disease given adenosine during stress imaging.

For arrhythmias associated with the use of adenosine in organ preservation solutions see above.

1. Mallet ML. Proarrhythmic effects of adenosine: a review of the literature. *Emerg Med J* 2004; **21:** 408–10.
2. Strickberger SA, *et al.* Adenosine-induced atrial arrhythmia: a prospective analysis. *Ann Intern Med* 1997; **127:** 417–22.
3. Exner DV, *et al.* Proarrhythmia in patients with the Wolff-Parkinson-White syndrome after standard doses of intravenous adenosine. *Ann Intern Med* 1995; **122:** 351–2.
4. Nagappan R, *et al.* Potential dangers of the Valsalva maneuver and adenosine in paroxysmal supraventricular tachycardia—beware preexcitation. *Crit Care Resusc* 2002; **4:** 107–11.
5. Haynes BE. Two deaths after prehospital use of adenosine. *J Emerg Med* 2001; **21:** 151–4.
6. Polad JE, Wilson LM. Myocardial infarction during adenosine stress test. Abstract: *Heart* 2002; **87:** 106. Full version: http://heart.bmj.com/cgi/reprint/87/2/e2.pdf (accessed 10/07/07)
7. Reyes E, *et al.* Acute myocardial infarction during adenosine myocardial perfusion imaging. *J Nucl Cardiol* 2004; **11:** 97–9.

Effects on the respiratory system. Acute exacerbation of asthma can be provoked by inhalation of adenosine. Bronchospasm has also been reported in patients with asthma[1,2] or a history of asthma[3] given adenosine intravenously and bronchospasm followed by respiratory failure in a patient with obstructive pulmonary disease.[4] Respiratory arrest has also been reported in an asthmatic patient.[5]

1. DeGroff CG, Silka MJ. Bronchospasm after intravenous administration of adenosine in a patient with asthma. *J Pediatr* 1994; **125:** 822–3.
2. Drake I, *et al.* Bronchospasm induced by intravenous adenosine. *Hum Exp Toxicol* 1994; **13:** 263–5.
3. Hintringer F, *et al.* Supraventricular tachycardia. *N Engl J Med* 1995; **333:** 323.
4. Burkhart KK. Respiratory failure following adenosine administration. *Am J Emerg Med* 1993; **11:** 249–50.
5. Patton JW, Sharma GK. Adenosine-induced respiratory arrest in an asthmatic patient. *South Med J* 2008; **101:** 328–9.

Migraine. A 35-year-old man with a history of migraine developed symptoms identical to those of his usual episodes of migraine immediately after 2 intravenous bolus doses of adenosine.[1]

1. Brown SGA, Waterer GW. Migraine precipitated by adenosine. *Med J Aust* 1995; **162:** 389–91.

Interactions

Dipyridamole inhibits adenosine uptake and therefore may potentiate the action of adenosine; if use of the two drugs is essential the dosage of adenosine should be reduced. Theophylline and other xanthines are competitive antagonists of adenosine. The risk of AV block may be increased if adenosine is used with other drugs that slow AV conduction.

Pharmacokinetics

Intravenous adenosine is rapidly taken up by an active transport system into erythrocytes and vascular endothelial cells where it is metabolised to inosine and adenosine monophosphate. The plasma half-life is less than 10 seconds.

Uses and Administration

Adenosine is an endogenous adenine nucleoside that is one of the components of nucleic acids (p.2355) and many coenzymes; as such, it is involved in many biological processes. It acts as an antiarrhythmic by stimulating adenosine A_1-receptors and slowing conduction through the AV node. It does not fit into the usual classification of antiarrhythmics (p.1153). It also produces peripheral and coronary vasodilatation by stimulating adenosine A_2-receptors.

Adenosine is used to restore sinus rhythm in the treatment of paroxysmal supraventricular tachycardia, including that associated with the Wolff-Parkinson-White syndrome (but see Effects on the Heart, above). It is also used for the differential diagnosis of broad or narrow complex supraventricular tachycardias and in myocardial imaging.

In the treatment of **paroxysmal supraventricular tachycardia**, adenosine may be given in an initial dose of 3 mg by rapid intravenous injection. If this dose is not effective within 1 to 2 minutes, 6 mg may be given and if necessary, 12 mg after a further 1 to 2 minutes. Alternatively, an initial dose of 6 mg followed if necessary by two further doses of 12 mg at 1 to 2 minute intervals may be used, but this higher initial dose should not be given to heart transplant patients as they have an increased sensitivity to adenosine. For differential **diagnosis of supraventricular tachycardias** a similar dosage regimen is used, beginning with a dose of 3 mg followed by 6 mg and then 12 mg at 1 to 2 minute intervals if required. Doses for children with paroxysmal supraventricular tachycardia are discussed below.

In **myocardial imaging** adenosine is given by intravenous infusion in a dose of 140 micrograms/kg per minute for 6 minutes. The radionuclide is injected after 3 minutes of the infusion.

Adenosine and its derivatives, such as adenosine phosphate (p.2247) and adenosine triphosphate (p.2247), have been used in various metabolic disorders because of their role in biological processes. Adenosine triphosphate, as the disodium salt, has been used as an antiarrhythmic.

Administration in children. Adenosine may be used for the management of paroxysmal supraventricular tachycardia in children. Dosage recommendations vary. Licensed product information in the USA states that children weighing less than 50 kg, including neonates and infants, may be given an initial dose of 50 to 100 micrograms/kg; if this is not effective the dose may be increased by 50 to 100 micrograms/kg increments at 1 to 2 minute intervals until the arrhythmia is controlled or a single dose of 300 micrograms/kg is reached. Paediatric advanced cardiac life support guidelines[1] in the USA recommend an initial dose of 100 micrograms/kg (maximum 6 mg) followed by a second dose of 200 micrograms/kg (maximum 12 mg) if required, and are applicable to infants and children. In the UK, the BNFC recommends an initial dose of 100 micrograms/kg for children aged 1 to 12 years, or 150 micrograms/kg for neonates and infants up to 1 year; the dose may be increased by increments of 50 to 100 micrograms/kg at 2 minute intervals, to a maximum single dose of 300 micrograms/kg for neonates and 500 micrograms/kg for infants and children.

1. The American Heart Association. 2005 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 12: pediatric advanced life support. *Circulation* 2005; **112:** (suppl 1): IV167–IV187. Also available at: http://circ.ahajournals.org/cgi/reprint/112/24_suppl/IV-167 (accessed 10/07/07)

Cardiac arrhythmias. Adenosine is used for the termination of paroxysmal supraventricular tachycardia[1-4] (p.1160) and may often be the drug of choice. Bolus intravenous injection of adenosine produces a rapid response and the extremely short plasma half-life (less than 10 seconds) allows dosage titration every 1 to 2 minutes so that most episodes can be controlled within 5 minutes without the danger of drug accumulation.

Adenosine has been used successfully in pregnant women with paroxysmal supraventricular tachycardia[5-8] and cardioversion of fetal supraventricular tachycardia by direct fetal therapy with adenosine has been reported.[9,10]

Adenosine can be used for the differential **diagnosis** of broad complex tachycardia where the mechanism is not known.[1] If the cause is supraventricular, adenosine will terminate the arrhythmia or produce AV block to reveal the underlying atrial rhythm. If the cause is ventricular, adenosine will have no effect on the tachycardia, whereas if an alternative treatment such as verapamil is given to these patients severe hypotension and cardiac arrest can occur.

1. Faulds D, *et al.* Adenosine: an evaluation of its use in cardiac diagnostic procedures, and in the treatment of paroxysmal supraventricular tachycardia. *Drugs* 1991; **41:** 596–624.
2. Garratt CJ, *et al.* Adenosine and cardiac arrhythmias. *BMJ* 1992; **305:** 3–4.
3. Rankin AC, *et al.* Adenosine and the treatment of supraventricular tachycardia. *Am J Med* 1992; **92:** 655–64.