product information recommends that such patients should be given a test dose of 15 to 20 micrograms/kg with subsequent dosage adjustments being guided by monitoring of the block.

Neuromuscular disorders. Neuromuscular blockade was successfully achieved with mivacurium in an obese elderly patient with myasthenia gravis requiring surgery.[1] Only about half the usual dose was required and even then recovery was delayed. See Atracurium, p.1903 for a discussion of the use of competitive neuromuscular blockers in patients with neuromuscular disorders.

1. Seigne RD, Scott RPF. Mivacurium chloride and myasthenia gravis. *Br J Anaesth* 1994; **72:** 468–9.

Plasma cholinesterase deficiency. There have been reports of prolonged neuromuscular block produced by mivacurium in patients with plasma cholinesterase deficiency.[1-4] Time to full recovery varied; one patient required up to 8 hours.

1. Goudsouzian NG, *et al.* Prolonged neuromuscular block from mivacurium in two patients with cholinesterase deficiency. *Anesth Analg* 1993; **77:** 183–5.
2. Sockalingam I, Green DW. Mivacurium-induced prolonged neuromuscular block. *Br J Anaesth* 1995; **74:** 234–6.
3. Fox MH, Hunt PCW. Prolonged neuromuscular block associated with mivacurium. *Br J Anaesth* 1995; **74:** 237–8.
4. Zimmer S. Mivacurium and prolonged neuromuscular block. *Br J Anaesth* 1995; **75:** 823.

Tourniquets. Mivacurium might be unsuitable for neuromuscular blockade of a limb which has been isolated with a tourniquet in order to provide a bloodless field for surgery.[1] It is largely inactivated by the enzymatic action of plasma cholinesterase and would therefore continue to degrade locally leading to a loss of blockade in the limb, which could not be corrected by further doses unless the tourniquet was deflated. However, as for other competitive neuromuscular blockers, the use of mivacurium to supplement regional anaesthesia has produced prolonged muscle weakness well beyond cuff deflation.[2] This suggests that mivacurium is not broken down in the ischaemic limb and that recovery is not dependent on plasma concentrations of mivacurium.

See also Local Anaesthetics, under Interactions of Atracurium, p.1904, for a report of symptoms suggestive of local anaesthetic toxicity when prilocaine and mivacurium were used together.

1. Shannon PF. Neuromuscular block and tourniquets. *Br J Anaesth* 1994; **73:** 726.
2. Torrance JM, *et al.* Low-dose mivacurium supplementation of prilocaine i.v. regional anaesthesia. *Br J Anaesth* 1997; **78:** 222–3.

Interactions

For interactions associated with competitive neuromuscular blockers, see Atracurium, p.1903.

Metoclopramide. Metoclopramide, an inhibitor of plasma cholinesterase, was found to significantly prolong the duration of action of mivacurium in patients undergoing surgery, although in this study only marginal inhibition of plasma cholinesterase by metoclopramide occurred.[1]

1. Skinner HJ, *et al.* Influence of metoclopramide on plasma cholinesterase and duration of action of mivacurium. *Br J Anaesth* 1999; **82:** 542–5.

Pharmacokinetics

Mivacurium is a mixture of 3 stereoisomers, 2 of which (*cis-trans* and *trans-trans*) are considered to account for most of the neuromuscular blocking effect. All 3 isomers are inactivated by plasma cholinesterase. Renal and hepatic mechanisms are involved in their elimination with excretion in urine and bile.

◊ Reviews.

1. Atherton DPL, Hunter JM. Clinical pharmacokinetics of the newer neuromuscular blocking drugs. *Clin Pharmacokinet* 1999; **36:** 169–89.

Uses and Administration

Mivacurium chloride is a benzylisoquinolinium competitive neuromuscular blocker (see Atracurium, p.1905).

On intravenous injection muscle relaxation occurs within 1.5 to 2.5 minutes, depending on the dose with a duration of action of about 10 to 20 minutes. It is used for endotracheal intubation and to provide muscle relaxation in general anaesthesia for surgical procedures (see Anaesthesia, p.1900) and to aid controlled ventilation (see Intensive Care, p.1901).

Doses of neuromuscular blockers need to be carefully titrated for individual patients according to response, and may vary with the procedure, the other drugs given, and the state of the patient; monitoring of the degree of block is recommended in order to reduce the risk of overdosage. Mivacurium is given as the chloride although doses are expressed in terms of mivacurium base. The initial dose by intravenous injection is 70 to 250 micrograms/kg. Doses up to 150 micrograms/kg may be given over 5 to 15 seconds but higher doses should be given over 30 seconds. In patients with asthma or cardiovascular disease, or those who are sensitive to falls in arterial blood pressure, it should be given over 60 seconds. To give a dose of 250 micrograms/kg for tracheal intubation, an injection of 150 micrograms/kg may be followed 30 seconds later by an injection of 100 micrograms/kg. Maintenance doses of 100 micrograms/kg may be given at intervals of 15 minutes. In children aged 2 to 6 months an initial dose of 150 micrograms/kg has been given; in children aged 7 months to 12 years, an initial dose of 200 micrograms/kg has been given. A maintenance dose of 100 micrograms/kg may be given every 6 to 9 minutes for children aged 2 months to 12 years.

Mivacurium chloride may also be given by continuous intravenous infusion for maintenance of block. For adults the initial rate is 8 to 10 micrograms/kg per minute adjusted every 3 minutes if necessary by increments of 1 microgram/kg per minute to a usual rate of 6 to 7 micrograms/kg per minute; in children aged 2 months to 12 years the usual dose is 11 to 14 micrograms/kg per minute.

Reduced doses may be required in the elderly and in patients with hepatic or renal impairment (see below).

◊ Reviews.

1. Mirakhur RK. Newer neuromuscular blocking drugs: an overview of their clinical pharmacology and therapeutic use. *Drugs* 1992; **44:** 182–99.
2. Frampton JE, McTavish D. Mivacurium: a review of its pharmacology and therapeutic potential in general anaesthesia. *Drugs* 1993; **45:** 1066–89.
3. Feldman S. Mivacurium. *Br J Hosp Med* 1997; **57:** 199–201.

Action. Mivacurium has a shorter duration of action than most other competitive neuromuscular blockers. Studies[1-3] suggest that it is a useful alternative to suxamethonium for the production of neuromuscular block of short duration and has the advantage that its block can be reversed with an anticholinesterase. For a discussion of the choice of anticholinesterase for reversal of neuromuscular block produced by short-acting blockers such as mivacurium, see under Neostigmine, p.633. Although its onset of action may be accelerated by giving a priming dose,[4] mivacurium has a slower onset than suxamethonium and so may not be a suitable alternative[5] when rapid intubation is required. For a general review of neuromuscular blockers, see Anaesthesia, p.1900.

1. Brandom BW, *et al.* Comparison of mivacurium and suxamethonium administered by bolus and infusion. *Br J Anaesth* 1989; **62:** 488–93.
2. Caldwell JE, *et al.* Comparison of the neuromuscular block induced by mivacurium, suxamethonium or atracurium during nitrous oxide-fentanyl anaesthesia. *Br J Anaesth* 1989; **63:** 393–9.
3. Goldberg ME, *et al.* Comparison of tracheal intubating conditions and neuromuscular blocking profiles after intubating doses of mivacurium chloride or succinylcholine in surgical outpatients. *Anesth Analg* 1989; **69:** 93–9.
4. Haxby EJ, *et al.* Mivacurium priming intervals. *Br J Anaesth* 1994; **72:** 485P.
5. Anonymous. Mivacurium—a new neuromuscular blocker. *Med Lett Drugs Ther* 1992; **34:** 82.

Administration in the elderly. In a study[1] comparing the effects of mivacurium in elderly and young adults, the duration of neuromuscular effects was prolonged in elderly patients by about 30%. The mean infusion requirement in elderly patients was 3.67 micrograms/kg per minute compared with 5.5 micrograms/kg per minute in young adults.

Licensed product information states that elderly patients may require decreased infusion rates or smaller or less frequent maintenance bolus doses.

1. Maddineni VR, *et al.* Neuromuscular and haemodynamic effects of mivacurium in elderly and young adult patients. *Br J Anaesth* 1994; **73:** 608–12.

Administration in hepatic or renal impairment. The pharmacokinetics of mivacurium have been studied in patients with renal[1-3] or hepatic impairment.[1,4,5] The duration of relaxation produced by mivacurium was about 1.5 times greater than normal in patients with end-stage renal disease and up to about 3 times greater than normal in patients with end-stage liver disease. Reduced plasma-cholinesterase activity in the patients with hepatic impairment may have played an important part in this effect. Although an anticholinesterase such as neostigmine hastens recovery by only a few minutes in healthy subjects, its use may be indicated in patients in whom recovery is delayed.[2]

Licensed product information recommends that in patients with end-stage renal or liver disease, the dose should be adjusted according to individual clinical response.

1. Cook DR, *et al.* Pharmacokinetics of mivacurium in normal patients and in those with hepatic or renal failure. *Br J Anaesth* 1992; **69:** 580–5.
2. Phillips BJ, Hunter JM. Use of mivacurium chloride by constant infusion in the anephric patient. *Br J Anaesth* 1992: **68:** 492–8.
3. Head-Rapson AG, *et al.* Pharmacokinetics and pharmacodynamics of the three isomers of mivacurium in health, in end-stage renal failure and in patients with impaired renal function. *Br J Anaesth* 1995; **75:** 31–6.
4. Devlin JC, *et al.* Pharmacodynamics of mivacurium chloride in patients with hepatic cirrhosis. *Br J Anaesth* 1993; **71:** 227–31.
5. Head-Rapson AG, *et al.* Pharmacokinetics of the three isomers of mivacurium and pharmacodynamics of the chiral mixture in hepatic cirrhosis. *Br J Anaesth* 1994; **73:** 613–18.

Preparations

Proprietary Preparations (details are given in Part 3)
Arg.: Mivacron; ***Austral.:*** Mivacron; ***Austria:*** Mivacron; Novacrium; ***Belg.:*** Mivacron; ***Braz.:*** Mivacron†; ***Canad.:*** Mivacron†; ***Chile:*** Mivacron; ***Cz.:*** Mivacron; ***Denm.:*** Mivacron; ***Fin.:*** Mivacron; ***Fr.:*** Mivacron; ***Ger.:*** Mivacron; ***Gr.:*** Mivacron; ***Hong Kong:*** Mivacron; ***Hung.:*** Mivacron; ***Irl.:*** Mivacron; ***Israel:*** Mivacron†; ***Ital.:*** Mivacron; ***Malaysia:*** Mivacron; ***Neth.:*** Mivacron; ***Norw.:*** Mivacron; ***NZ:*** Mivacron; ***Pol.:*** Mivacron; ***Port.:*** Mivacron; ***Rus.:*** Mivacron (Мивакрон); ***S.Afr.:*** Mivacron; ***Singapore:*** Mivacron; ***Spain:*** Mivacron; ***Swed.:*** Mivacron; ***Switz.:*** Mivacron; ***Turk.:*** Mivacron; ***UK:*** Mivacron; ***USA:*** Mivacron†.

Pancuronium Bromide *(BAN, USAN, rINN)*

Bromuro de pancuronio; NA-97; Org-NA-97; Pancuronii bromidum; Pancuronium, bromure de; Pankuronio bromidas; Pankuroniowy bromek; Pankuroniumbromid; Pankuronium-bromid; Pankuroniumbromidi; Pankuronyum Bromür. 1,1′-(3α,17β-Diacetoxy-5α-androstan-2β,16β-ylene)bis(1-methylpiperidinium) dibromide.

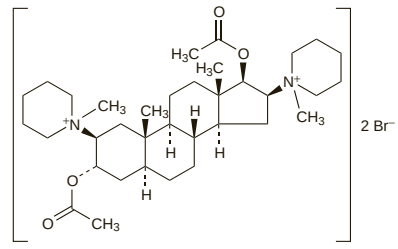
Панкурония Бромид

$C_{35}H_{60}Br_2N_2O_4$ = 732.7.

CAS — 15500-66-0.

ATC — M03AC01.

ATC Vet — QM03AC01.



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

Ph. Eur. 6.2 (Pancuronium Bromide). White, yellowish-white, or slightly pink, hygroscopic crystalline powder. Very soluble to freely soluble in water; freely soluble in alcohol; very soluble in dichloromethane. Store in airtight containers. Protect from light.

USP 31 (Pancuronium Bromide). A white, yellowish-white, or slightly pink, crystalline hygroscopic powder. Freely soluble in water, in alcohol, and in dichloromethane. Store in airtight containers at a temperature of 15° to 25°. Protect from light.

Adverse Effects, Treatment, and Precautions

As for competitive neuromuscular blockers in general (see Atracurium, p.1902).

Pancuronium has vagolytic and sympathomimetic action, which may cause tachycardia and hypertension, but does not produce ganglionic blockade. It has little histamine-releasing effect. Hypersensitivity reactions are relatively rare but bradycardia, bronchospasm, hypotension, and cardiovascular collapse have been reported. Pancuronium has been associated with excessive salivation in some patients.

Pancuronium should be used with caution in patients with raised catecholamine concentrations, or in those who are receiving drugs with sympathomimetic effects, as cardiovascular adverse effects are more likely in these patients.

Effects on the ears. A study[1] found that neonates who survived congenital diaphragmatic hernia were more likely to suffer from sensorineural hearing loss after prolonged use of pancuronium bromide during the neonatal period. However, the authors commented that the association is not necessarily causal and that further investigation is required.

1. Cheung P-Y, *et al.* Prolonged use of pancuronium bromide and sensorineural hearing loss in childhood survivors of congenital diaphragmatic hernia. *J Pediatr* 1999; **135:** 233–9.

Hypersensitivity. Reports of anaphylactoid or anaphylactic reactions associated with pancuronium bromide.

See also under Suxamethonium Chloride, p.1910.

1. Brauer FS, Ananthanarayan CR. Histamine release by pancuronium. *Anesthesiology* 1978; **49:** 434–5.
2. Patriarca G, *et al.* Pancuronium allergy: a case report. *Br J Anaesth* 1989; **62:** 210–12.
3. Moneret-Vautrin DA, *et al.* Simultaneous anaphylaxis to thiopentone and a neuromuscular blocker: a study of two cases. *Br J Anaesth* 1990; **64:** 743–5.
4. Sanchez-Guerrero IM, *et al.* Anaphylactoid reaction induced by pancuronium during general anaesthesia. *Eur J Anaesthesiol* 1998; **15:** 613–14.

Postoperative complications. Because of its prolonged duration of action, pancuronium may be more likely than other neu-

romuscular blockers to produce residual neuromuscular block; such residual block is associated with an increased incidence of postoperative respiratory complications.[1,2]

1. Berg H, *et al.* Residual neuromuscular block is a risk factor for postoperative pulmonary complications: a prospective, randomised, and blinded study of postoperative pulmonary complications after atracurium, vecuronium and pancuronium. *Acta Anaesthesiol Scand* 1997; **41:** 1095–1103.
2. Bissinger U, *et al.* Postoperative residual paralysis and respiratory status: a comparative study of pancuronium and vecuronium. *Physiol Res* 2000; **49:** 455–62.

Interactions

For interactions associated with competitive neuromuscular blockers, see Atracurium, p.1903.

Pharmacokinetics

On intravenous injection pancuronium bromide is rapidly distributed into body tissues; about 80% may be bound to plasma proteins. A small proportion is metabolised in the liver to metabolites with weak neuromuscular blocking activity. It is largely excreted in urine as unchanged drug and metabolites; a small amount is excreted in bile. The plasma elimination half-life is about 2 hours. It crosses the placenta in small amounts.

Pregnancy. In 15 patients undergoing caesarean section[1] given pancuronium bromide 100 micrograms/kg intravenously with other agents, mean maternal arterial and umbilical venous serum concentrations of pancuronium bromide and metabolites were 520 and 120 nanograms/mL, respectively at delivery (mean of 13 minutes after injection), giving a fetal to maternal ratio of 0.23.

1. Wingard LB, *et al.* Modified fluorometric quantitation of pancuronium bromide and metabolites in human maternal and umbilical serums. *J Pharm Sci* 1979; **68:** 914–15.

Uses and Administration

Pancuronium bromide is an aminosteroidal competitive neuromuscular blocker (see Atracurium, p.1905). Muscle relaxation occurs within about 1.5 to 2 minutes of intravenous injection and lasts for about 45 to 60 minutes.

Pancuronium bromide is used for endotracheal intubation and to provide muscle relaxation in general anaesthesia for surgical procedures (see Anaesthesia, p.1900) and to aid controlled ventilation (see Intensive Care, p.1901).

Doses of neuromuscular blockers need to be carefully titrated for individual patients according to response, and may vary with the procedure, the other drugs given, and the state of the patient; monitoring of the degree of block is recommended in order to reduce the risk of overdosage. The initial dose for intubation is usually 50 to 100 micrograms/kg by intravenous injection, with maintenance doses of 10 to 20 micrograms/kg. Children may be given similar doses. Some manufacturers recommend a reduction in the initial dose to 20 to 60 micrograms/kg when pancuronium is given following suxamethonium. Doses of 30 to 40 micrograms/kg initially have been suggested in neonates, with maintenance doses of 10 to 20 micrograms/kg as necessary; in the UK, the *BNFC* suggests that higher doses may be used for neonates in some cases. In the USA, dosage based on an initial test dose of 20 micrograms/kg has been advocated for the neonate.

Adult patients under intensive care who require assisted ventilation for conditions such as intractable status asthmaticus or tetanus may be given 60 micrograms/kg intravenously every 1 to 1½ hours or less frequently.

Care should be taken when giving pancuronium to patients with hepatic or renal impairment, see below.

Administration in hepatic impairment. Prolonged neuromuscular blockade may occur in patients with liver disease given pancuronium bromide since increased elimination half-life with increased volume of distribution and reduced clearance has been reported.[1] However, the expanded distribution volume may necessitate an increase in the dose of pancuronium in these patients[1,2] and may be interpreted as resistance to the neuromuscular blocking effects of pancuronium.

1. Duvaldestin P, *et al.* Pancuronium pharmacokinetics in patients with liver cirrhosis. *Br J Anaesth* 1978; **50:** 1131–6.
2. Ward ME, *et al.* Althesin and pancuronium in chronic liver disease. *Br J Anaesth* 1975; **47:** 1199–1204.

Administration in renal impairment. Prolonged neuromuscular blockade may occur when pancuronium is given to patients with severe renal impairment. Pancuronium distributes rapidly into extracellular fluid after intravenous injection and the initial neuromuscular blockade produced will depend upon the peak drug concentration in this fluid. Since extracellular fluid volume is increased in chronic renal failure such patients may require a larger initial dose of pancuronium and a 45% increase in dose requirement has been reported[1] in patients with end-stage renal failure. Renal excretion is the main route of elimination and prolonged elimination half-life with reduced clearance may be expected in renal failure; total dose requirements may be reduced. The main infusion rate of pancuronium to maintain 90% blockade in patients with end-stage renal failure was reported to be 61.5% less than for patients with normal renal function.

1. Gramstad L. Atracurium, vecuronium and pancuronium in end-stage renal failure. *Br J Anaesth* 1987; **59:** 995–1003.

Fetal paralysis. Pancuronium bromide 100 micrograms/kg of the estimated fetal-weight, given into the umbilical vein, produced fetal paralysis for about 40 minutes during intravascular exchange transfusion.[1] A dose of 200 to 300 micrograms/kg produced fetal paralysis for about 1 to 8 hours for more complicated transfusion procedures.[2] No adverse effects were reported.

1. Copel JA, *et al.* The use of intravenous pancuronium bromide to produce fetal paralysis during intravascular transfusion. *Am J Obstet Gynecol* 1988; **158:** 170–1.
2. Moise KJ, *et al.* Intravenous pancuronium bromide for fetal neuromuscular blockade during intrauterine transfusion for red-cell alloimmunization. *Obstet Gynecol* 1989; **74:** 905–8.

Neuroleptic malignant syndrome. Pancuronium is one of several drugs for which there have been isolated reports[1] of success in the management of neuroleptic malignant syndrome (p.972).

1. Sangal R, Dimitrijevic R. Neuroleptic malignant syndrome: successful treatment with pancuronium. *JAMA* 1985; **254:** 2795–6.

Preparations

BP 2008: Pancuronium Injection.

Proprietary Preparations (details are given in Part 3)
Arg.: Bemicin; Pancuron; Pavulon; Plumger; ***Austral.:*** Pavulon; ***Braz.:*** Pancuron; Pavulon; ***Chile:*** Pavulon; ***Cz.:*** Pavulon; ***Denm.:*** Pavulon; ***Fin.:*** Pavulon; ***Fr.:*** Pavulon; ***Gr.:*** Pavulon; ***Hong Kong:*** Pavulon; ***Hung.:*** Pavulon†; ***India:*** Panconium; ***Indon.:*** Pavulon; ***Irl.:*** Pavulon†; ***Israel:*** Pavulon; ***Ital.:*** Pavulon; ***Jpn:*** Mioblock; ***Malaysia:*** Pavulon; ***Mex.:*** Bromurex; Panlem; ***Neth.:*** Pavulon; ***Norw.:*** Pavulon; ***Philipp.:*** Pavulon; ***Port.:*** Pancurox; Pavulon†; ***S.Afr.:*** Curon-B; Pavulon; ***Singapore:*** Pavulon; ***Spain:*** Pavulon†; ***Swed.:*** Pavulon; ***Switz.:*** Pavulon; ***Thai.:*** Pavulon; ***Turk.:*** Pavulon; ***USA:*** Pavulon†; ***Venez.:*** Panuron; Pavulon; Pesium.

Pipecuronium Bromide *(BAN, USAN, rINN)*

Bromuro de pipecuronio; Pipecurium Bromide; Pipecuronii Bromidum; Pipécuronium, Bromure de; Pipekuroniumbromid; Pipekuroniumbromidi; RGH-1106. 1,1,1′,1′-Tetramethyl-4,4′-(3α,17β-diacetoxy-5α-androstan-2β,16β-diyl)dipiperazinium dibromide.

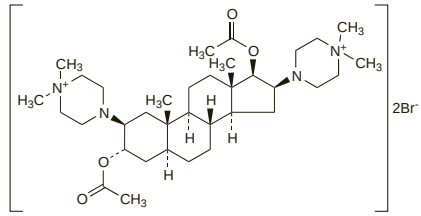
Пипекурония Бромид

$C_{35}H_{62}Br_2N_4O_4$ = 762.7.

CAS — 52212-02-9 (anhydrous pipecuronium bromide); 68399-57-5 (pipecuronium bromide dihydrate).

ATC — M03AC06.

ATC Vet — QM03AC06.



Profile

Pipecuronium bromide is an aminosteroidal competitive neuromuscular blocker (see Atracurium, p.1902). Pipecuronium is reported to have no significant cardiovascular adverse effects or histamine-related effects. On intravenous injection muscle relaxation occurs within 2.5 to 3 minutes with a duration of action of about 30 minutes to 2 hours, depending on the dose.

Pipecuronium bromide has been used for endotracheal intubation and to provide muscle relaxation in general anaesthesia for surgical procedures (see Anaesthesia, p.1900) and to aid mechanical ventilation (see Intensive Care, p.1901).

Doses of neuromuscular blockers need to be carefully titrated for individual patients according to response, and may vary with the procedure, the other drugs given, and the state of the patient; monitoring of the degree of block is recommended in order to reduce the risk of overdosage. Initial doses of 80 to 100 micrograms/kg intravenously have been recommended, with subsequent doses of 10 to 20 micrograms/kg. Lower initial doses are given following suxamethonium or in patients at high risk: 50 to 60 micrograms/kg has been recommended, or 35 micrograms/kg for caesarean section.

◊ Reviews and studies.

1. Mirakhur RK. Newer neuromuscular blocking drugs: an overview of their clinical pharmacology and therapeutic use. *Drugs* 1992; **44:** 182–99.
2. Tassonyi E, *et al.* Pharmacokinetics of pipecuronium in infants, children and adults. *Eur J Drug Metab Pharmacokinet* 1995; **20:** 203–8.
3. Melloni C. Farmacologia clinica del pipecuronium; studio comparativo della sua durata clinica in anestesia bilanciata (propofol/fentanyl) vs isoflurano. *Minerva Anestesiol* 1995; **61:** 491–500.
4. Meretoja OA, Erkola O. Pipecuronium revisited: dose-response and maintenance requirement in infants, children, and adults. *J Clin Anesth* 1997; **9:** 125–9.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Arpilon†; ***Cz.:*** Arduan; ***Hung.:*** Arduan; ***Pol.:*** Arduan; ***Rus.:*** Aperomid (Аеромид)†; Arduan (Ардуан).

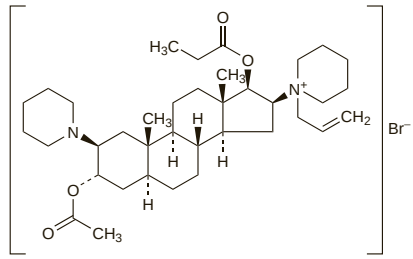
Rapacuronium Bromide *(BAN, USAN, rINN)*

Bromuro de rapacuronio; Org-9487; Rapacuronii Bromidum; Rapacuronium, Bromure de. 1-(3α-Acetoxy-2β-piperidino-17β-propionyloxy-5α-androstan-16β-yl)-1-allylpiperidinium bromide; 1-Allyl-1-(3α,17β-dihydroxy-2β-piperidino-5α-androstan-16β-yl)piperidinium bromide, 3-acetate 17-propionate.

Рапакурония Бромид

$C_{37}H_{61}BrN_2O_4$ = 677.8.

CAS — 156137-99-4.



Profile

Rapacuronium bromide, an analogue of vecuronium (p.1913), is an aminosteroidal competitive neuromuscular blocker (see Atracurium, p.1905). It was used to provide muscle relaxation in general anaesthesia for surgical procedures and for endotracheal intubation, but was withdrawn from the market after reports of severe bronchospasm, including fatalities.

◊ Reviews.

1. Wight WJ, Wright PMC. Pharmacokinetics and pharmacodynamics of rapacuronium bromide. *Clin Pharmacokinet* 2002; **41:** 1059–76.

Rocuronium Bromide *(BAN, USAN, rINN)*

Bromuro de rocuronio; Org-9426; Rocuronii bromidum; Rocuronium, bromure de; Rokuroniowy bromek; Rokuroniumbromid; Rokuronium-bromid; Rokuroniumbromidi; Rokuronyum Bromür. 1-Allyl-1-(3α,17β-dihydroxy-2β-morpholino-5α-androstan-16β-yl)pyrrolidinium bromide 17-acetate; 1-(17β-Acetoxy-3α-hydroxy-2β-morpholino-5α-androstan-16β-yl)-1-allylpyrrolidinium bromide.

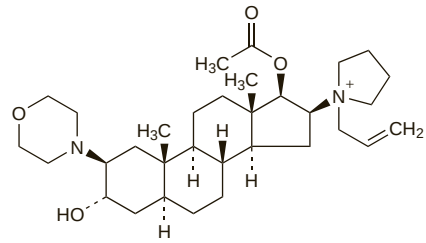
Рокурония Бромид

$C_{32}H_{53}BrN_2O_4$ = 609.7.

CAS — 119302-91-9.

ATC — M03AC09.

ATC Vet — QM03AC09.



(rocuronium)

Pharmacopoeias. In *Eur.* (see p.vii).
Ph. Eur. 6.2 (Rocuronium Bromide). An almost white or pale yellow, slightly hygroscopic, powder. Freely soluble in water and in dehydrated alcohol. A 1.0% solution in water has a pH of 8.9 to 9.5. Store in airtight containers. Protect from light.

Adverse Effects, Treatment, and Precautions

As for competitive neuromuscular blockers in general (see Atracurium, p.1902). Rocuronium is reported to