correct hypoxaemia in order to prevent development of any associated oxygen toxicity, including increased carbon dioxide retention. High concentrations of oxygen should be used in carbon monoxide poisoning and, in selected patients, treatment with hyperbaric oxygen considered.

In respiratory failure associated with chronic obstructive pulmonary disease (conditions such as chronic bronchitis and emphysema) oxygen is usually given initially at an inspired concentration of up to 28%. High concentrations are to be avoided as they may enhance carbon dioxide retention and narcosis.

Oxygen at a pressure greater than 1 atmosphere absolute, i.e. hyperbaric oxygen therapy (below), is given by enclosing the patient in a special high-pressure chamber. It may be used in carbon monoxide poisoning, as an adjunct in the treatment of severe anaerobic infections, especially gas gangrene, and for the treatment of decompression sickness and gas emboli.

◊ General references.


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2. Gribbin HR. Management of respiratory failure. *Br J Hosp Med* 1993; **49:** 461–77.
3. Tarpy SP, Celli BR. Long-term oxygen therapy. *N Engl J Med* 1995; **333:** 710–14.
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9. Balfour-Lynn IM, *et al.* Home oxygen for children: who, how and when? *Thorax* 2005; **60:** 76–81.


**Cluster headache.** Inhalation of 100% oxygen can provide rapid and effective treatment of cluster headache attacks (p.616) but practical difficulties associated with its use result in other drugs being preferred. It has been noted that the evidence of value is limited.

References.


1. Fogan L. Treatment of cluster headache: a double-blind comparison of oxygen v air inhalation. *Arch Neurol* 1985; **42:** 362–3.
2. Rozen TD. High oxygen flow rates for cluster headache. *Neurology* 2004; **63:** 593.
3. Bennett MH, *et al.* Normobaric and hyperbaric oxygen therapy for migraine and cluster headache. Available in The Cochrane Database of Systematic Reviews; Issue 3. Chichester: John Wiley; 2008 (accessed 21/08/08).


**Hyperbaric oxygen therapy.** The use of hyperbaric oxygen therapy, which involves the intermittent inhalation of 100% oxygen under a pressure of greater than 1 atmosphere in a specialised chamber, has been reviewed.[1-3] In the 1960s hyperbaric therapy was used for disorders such as *myocardial infarction*, *stroke*, *senility*, and *cancer* but clinical studies and experience have shown little benefit and enthusiasm has since waned. Hyperbaric oxygen therapy has also been tried in *multiple sclerosis* but there is little evidence of benefit. There are, however, other disorders for which the evidence supporting the efficacy of hyperbaric oxygen is much stronger.

Hyperbaric oxygen is a safe and effective primary therapy for *decompression sickness* and *air or gas embolism*. The effect is achieved through the mechanical reduction in bubble size in the blood brought about by an increase in ambient pressure; the increased oxygenation of blood due to the additional pressure used for these conditions (often 6 rather than 2 or 3 atmospheres) is also beneficial.

The role of hyperbaric oxygen therapy in *carbon monoxide poisoning* is unclear but it should be considered in selected patients (see p.1688). Its mechanism of action is not fully understood; it increases the rate at which carboxyhaemoglobin concentrations decline, increases intracellular delivery of oxygen, and may also reduce lipid peroxidation and thus spare neuronal cell membranes.

Hyperbaric oxygen is used as adjunctive therapy in *clostridial infections (gas gangrene)* (p.171). Early treatment appears to reduce systemic toxic reactions (probably by inhibiting the production of alpha toxin by the anaerobic bacteria, *Clostridium*) thus enabling patients to tolerate surgery more readily; additionally there is a clearer demarcation of viable and nonviable tissue. *Necrotising fasciitis* (p.180) is another infection in which hyperbaric oxygen therapy may be useful.

There is some evidence that hyperbaric oxygen may be useful in other types of *wounds*. In an *acute crush injury* therapy may reduce oedema via vasoconstriction and reverse ischaemia by increased oxygen delivery. In *problem wounds*, including venous ulcers, therapy may increase the tissue oxygen tension and stimulate angioneogenesis but it is emphasised that it is adjunctive therapy and not a replacement for meticulous local care. Hyperbaric oxygen therapy might also reduce the risk of major amputation in patients with chronic *diabetic foot ulcer*.[4,5] Improved healing of leg ulcers has been reported[6] with use of hyperbaric oxygen therapy in patients with livedoid vasculopathy. Other wounds in which therapy may be beneficial include thermal burns and compromised skin grafts and flaps. The management of burns and wounds is described on p.1578 and p.1585, respectively.

*Radiation therapy* can damage normal adjacent tissue resulting in tissue hypoxia and eventual cell death. Hyperbaric oxygen therapy appears to aid in salvaging such tissue by stimulating angioneogenesis in marginally viable tissue and has been demonstrated to be beneficial in osteoradionecrosis, radiation-induced haemorrhagic cystitis (p.2178), and other radiation-damaged soft tissue.[7]

There has been interest in the use of hyperbaric oxygen in children with *cerebral palsy*, although a randomised study[8] found that it was no better than pressurised air. However, improved neurological outcomes have been reported with hyperbaric oxygen therapy in neonates with hypoxic-ischaemic encephalopathy.[9]


1. Grim PS, *et al.* Hyperbaric oxygen therapy. *JAMA* 1990; **263:** 2216–20.
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**Respiratory failure.** Respiratory failure occurs when the arterial plasma partial pressure of oxygen ($P_aO_2$) and of carbon dioxide ($P_aCO_2$) cannot be maintained within normal physiological limits.[1] Respiratory failure can be classified into 2 types, both of which are characterised by a low $P_aO_2$ (hypoxaemia). However, in type I the $P_aCO_2$ is normal or low whereas in type II, referred to as ventilatory failure, $P_aCO_2$ is raised (hypercapnia). Some conditions, for example asthma, can produce either type of respiratory failure.

Management of respiratory failure mainly involves giving oxygen to reverse hypoxaemia, and specific therapy for any underlying condition. Respiratory stimulants may be considered in some situations.

In type I respiratory failure oxygen is used in high concentrations. Nasal prongs and certain face masks can provide concentrations of up to 60% but if concentrations higher than this are needed then tight-fitting anaesthetic-type masks or methods of delivery such as endotracheal intubation have to be used.

In type II respiratory failure both high and low concentrations are used according to need.

Patients with *acute severe asthma* (p.1108) should usually be given oxygen at high concentrations of 40 to 60%. In patients with exacerbations of chronic respiratory disorders such as *chronic obstructive pulmonary disease* (COPD, p.1112) the aim is to improve hypoxaemia without increasing hypercapnia and respiratory acidosis.[2] The initial concentration of oxygen to give in COPD exacerbations is controversial. During the transfer to hospital, UK guidelines[3] recommend starting at 40% and titrating upwards if the oxygen saturation falls below 90% and downwards if the patient becomes drowsy or if the saturation exceeds 93 to 94%. Special care is needed for patients with known type II respiratory failure, especially if they require a long ambulance journey or receive oxygen at home for a prolonged period before an ambulance arrives, as uncontrolled oxygen therapy can result in suppression of respiratory drive, carbon dioxide narcosis, and respiratory arrest. In hospital, arterial blood gases should be used to guide treatment. Other guidelines[4] consider that lower initial oxygen concentrations of 24 to 28% are usually sufficient. Patients with exacerbations of chronic ventilatory failure already have an increased central drive to the respiratory muscles and therefore respiratory stimulants such as doxapram have a limited role but may be indicated for short-term use if hypercapnia worsens as a result of oxygen. For most patients with chronic obstructive pulmonary disease, non-invasive ventilation is the initial treatment of choice for hypercapnic ventilatory failure during exacerbations.[3] Respiratory stimulants may be considered in the management of *postanaesthetic hypoventilation*. Although naloxone can reverse respiratory depression caused by opioid analgesics careful dosage adjustment is required as it can also abolish analgesia. Specific antagonists such as naloxone and flumazenil are also used to treat hypoventilation associated with opioid and benzodiazepine overdosage, respectively. If oxygen therapy fails to raise $P_aO_2$ in respiratory failure and there is worsening hypercapnia and respiratory acidosis the use of artificial ventilation should be considered.

Severe respiratory failure in *neonates* may result from various disorders. Use of surfactant or inhaled nitric oxide may be of benefit in some cases but extracorporeal membrane oxygenation (ECMO), where blood is removed from the neonate, oxygenated, and re-injected in a continuous circuit that also removes carbon dioxide, may be required.[5] ECMO has also been used in older children and in adults,[6] but is less well established.


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3. National Collaborating Centre for Chronic Conditions. Chronic obstructive pulmonary disease: national clinical guideline on management of chronic obstructive pulmonary disease in adults in primary and secondary care. *Thorax* 2004; **59** (suppl 1): 1–232. Also available at: http://thorax.bmj.com/content/vol59/suppl_1/ (accessed 18/12/07)
4. McKenzie DK, *et al.* The COPDX plan: Australian and New Zealand guidelines for the management of chronic obstructive pulmonary disease 2007. Available at: http://www.copdx.org.au/guidelines/documents/COPDX_Sep28_2007.pdf (accessed 18/12/07)
5. Barrington KJ, Finer NN. Care of near term infants with respiratory failure. *BMJ* 1997; **315:** 1215–18.
6. Peek GJ, *et al.* Extracorporeal membrane oxygenation: potential for adults and children? *Hosp Med* 1998; **59:** 304–8.


**Wounds.** Hyperbaric oxygen therapy may have a role in the management of infected and problem wounds (see above). Supplemental normobaric oxygen has been tried in the prevention of postoperative wound infections, but results of controlled studies[1-3] have been contradictory and its role is not established.

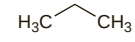

1. Greif R, *et al.* Supplemental perioperative oxygen to reduce the incidence of surgical-wound infection. *N Engl J Med* 2000; **342:** 161–7.
2. Pryor KO, *et al.* Surgical site infection and the routine use of perioperative hyperoxia in a general surgical population: a randomized controlled trial. *JAMA* 2004; **291:** 79–87.
3. Belda FJ, *et al.* Supplemental perioperative oxygen and the risk of surgical wound infection: a randomized controlled trial. *JAMA* 2005; **294:** 2035–42.


### Preparations

**Proprietary Preparations** (details are given in Part 3)
**Multi-ingredient:** ***Fr.:*** Kalinox; Medimix; ***S.Afr.:*** Entonox; ***UK:*** Entonox; Equanox.

## Propane

Dimethylmethane; E944; Propano; Propyl Hydride.
$C_3H_8$ = 44.10.
*CAS — 74-98-6.*



**Pharmacopoeias.** In *USNF*.
**USNF 26** (Propane). A colourless gas. It is highly flammable and explosive. Store in airtight cylinders at a temperature not exceeding 40°.

### Profile

Propane is used as a refrigerant and as an aerosol propellant (p.1688). It is also widely used as a fuel.

◊ Reports of toxicity associated with the abuse or misuse of propane.[1-6]


1. James NK, Moss ALH. Cold injury from liquid propane. *BMJ* 1989; **299:** 950–1.
2. Siegel E, Wason S. Sudden death caused by inhalation of butane and propane. *N Engl J Med* 1990; **323:** 1638.
3. Tsoukali H, *et al.* Death during deliberate propane inhalation. *Forensic Sci Int* 1998; **93:** 1–4.
4. McLennan JJ, *et al.* Propane-associated autoerotic fatalities. *Am J Forensic Med Pathol* 1998; **19:** 381–6.
5. Grosse K, Grosse J. Propanmissbrauch: extreme Dosissteigerung durch Toleranzentwicklung. *Nervenarzt* 2000; **71:** 50–3.
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### Preparations

**Proprietary Preparations** (details are given in Part 3)
**Multi-ingredient:** ***Austral.:*** Histofreezer†; ***Fr.:*** Cliptol Sport†; Freeze; Histofreezer†; ***Ger.:*** Olbas; ***Irl.:*** Wartner; ***Israel:*** Wartner; ***NZ:*** Wartner; ***UK:*** Histofreezer; Wartner; ***USA:*** Compound W Freeze Off.

14. Anonymous. H. pylori eradication in NSAID-associated ulcers. *Drug Ther Bull* 2005; **43:** 37–40.
15. Tryba M, Cook D. Current guidelines on stress ulcer prophylaxis. *Drugs* 1997; **54:** 581–96.
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17. Sesler JM. Stress-related mucosal disease in the intensive care unit: an update on prophylaxis. *AACN Adv Crit Care* 2007; **18:** 119–26.
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20. Cook D, *et al.* A comparison of sucralfate and ranitidine for the prevention of upper gastrointestinal bleeding in patients requiring mechanical ventilation. *N Engl J Med* 1998; **338:** 791–7.
21. Messori A, *et al.* Bleeding and pneumonia in intensive care patients given ranitidine and sucralfate for prevention of stress ulcer: meta-analysis of randomised controlled trials. *BMJ* 2000; **321:** 1103–6.
22. Barkun A, *et al.* Nonvariceal Upper GI Bleeding Consensus Conference Group. Consensus recommendations for managing patients with nonvariceal upper gastrointestinal bleeding. *Ann Intern Med* 2003; **139:** 843–57. Also available at: http://www.annals.org/cgi/reprint/139/10/843.pdf (accessed 01/07/08)
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25. Andriulli A, *et al.* Proton-pump inhibitors and outcome of endoscopic hemostasis in bleeding peptic ulcers: a series of meta-analyses. *Am J Gastroenterol* 2005; **100:** 207–19.
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### Zollinger-Ellison syndrome

Zollinger-Ellison syndrome is a rare disorder characterised by the presence of a gastrin-producing tumour (gastrinoma), which leads to hypersecretion of gastric acid and consequent peptic ulcer disease (often with complications such as perforation or bleeding), diarrhoea, or malabsorption. Gastrinomas usually occur in the non-beta islet cells of the pancreas or in the duodenal wall. Up to two-thirds are malignant. About 20 to 25% of cases are seen in patients with multiple endocrine neoplasia type 1 (MEN-1) syndrome.

Initial treatment is aimed at controlling the hypersecretion of gastric acid with an antisecretory drug. Giving enough medication just to control symptoms is not considered adequate, and it is important that acid secretion is reduced below 10 mmol/hour. Intravenous $H_2$-antagonists or proton pump inhibitors may be required initially. Once the symptoms have been controlled the tumour can be investigated for surgical removal. When complete removal is not possible then antisecretory therapy is continued indefinitely. A proton pump inhibitor is the drug of choice; it profoundly reduces acid secretion with once- or twice-daily use, although relatively high doses are required compared with those used in other conditions. An $H_2$-antagonist such as cimetidine or ranitidine may be used as an alternative to omeprazole, and, as with omeprazole, daily doses are higher than those used for other conditions; they are given in 3 or 4 divided doses. The somatostatin analogue octreotide can be used to reduce serum gastrin, but has to be given subcutaneously, and is not well tolerated.

Parietal cell vagotomy may be performed to reduce acid secretion if the tumour is not found, to allow lower doses of antisecretory drugs to be used.

References.


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2. Qureshi W, Rashid S. Zollinger-Ellison syndrome: improved treatment options for this complex disorder. *Postgrad Med* 1998; **104:** 155–164.
3. Tomassetti P, *et al.* Treatment of Zollinger-Ellison syndrome. *World J Gastroenterol* 2005; **11:** 5423–32.

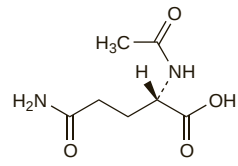

## Aceglutamide Aluminium *(USAN, rINNM)*

Aceglutamida de aluminio; Acéglutamide d'Aluminium; Aluminii Aceglutamidum; KW-1110. Pentakis ($N^2$-acetyl-L-glutaminato)tetrahydroxytrialuminium.

Алюминий Ацеглутамид

$C_{35}H_{59}Al_3N_{10}O_{24}$ = 1084.8.

*CAS — 12607-92-0.*



*(aceglutamide)*

**Pharmacopoeias.** In *Jpn.*

### Profile

Aceglutamide aluminium, a complex of aceglutamide with aluminium hydroxide, is an antacid with general properties similar to those of aluminium hydroxide (p.1706). It is given orally in a usual dose of 700 mg three times daily.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Jpn:*** Glumal.

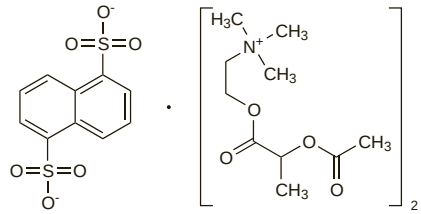
## Aclatonium Napadisilate *(BAN, rINN)*

Aclatonii Napadisilas; Aclatonium Napadisylate; Celatonium Napadisilate; Choline Naphthalene-1,5-Disulphonate (2:1) Dilactate Diacetate; Napadisilate d'Aclatonium; Napadisilato de aclatonio; SKF-100916-J; TM-723. 2-(2-Acetoxypropionyloxy)ethyltrimethylammonium naphthalene-1,5-disulphonate (2:1).

Аклатония Нападизилат

$2C_{10}H_{20}NO_4.C_{10}H_6O_6S_2$ = 722.8.

*CAS — 55077-30-0.*



### Profile

Aclatonium napadisilate is a cholinergic agonist given orally for its prokinetic properties in the management of decreased gastrointestinal motility (p.1694) after gastrointestinal surgery, and to relieve symptoms of biliary dyskinesia and chronic gastritis.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Jpn:*** Abovis.

## Albumin Tannate

Albumiinitannaatti; Albúmina, tanato de; Albumini Tannas; Albumintannat; Albutannin; Tannin Albuminate.

Альбумин Таннат

*CAS — 9006-52-4.*
*ATC — A07XA01.*
*ATC Vet — QA07XA01.*

**Pharmacopoeias.** In *Jpn.*

### Profile

Albumin tannate, a compound of tannin with albumin, is given orally for its astringent properties in the treatment of diarrhoea (p.1694). It is stated to liberate tannic acid (p.2394) in the gastrointestinal tract.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Austria:*** Tannalbin; ***Ger.:*** Tannalbin; ***Neth.:*** Entosorbine-N; Tannalbin†; ***Pol.:*** Taninal.

**Multi-ingredient:** ***Austria:*** Neoplex; ***Belg.:*** Tanalone; ***Cz.:*** Tannacomp†; ***Fin.:*** Tannopon; ***Ger.:*** Tannacomp; ***Hung.:*** Bolus Adstringens; ***Pol.:*** Salotannal; ***Spain:*** Demusin; Salitanol Estreptomicina.

## Alexitol Sodium *(BAN, rINN)*

Alexitol sódico; Alexitol Sodique; Alexitolum Natricum. Sodium poly(hydroxyaluminium) carbonate-hexitol complex.

Алекситол Натрий

*CAS — 66813-51-2.*

### Profile

Alexitol sodium is an antacid with general properties similar to those of aluminium hydroxide (p.1706). It is given orally in doses of 360 to 720 mg when required, up to a maximum of sixteen 360-mg tablets in 24 hours.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Hong Kong:*** Actal; ***Malaysia:*** Actal; ***Singapore:*** Actal; ***Thai.:*** Actal; ***UK:*** Actal.

**Multi-ingredient:** ***Malaysia:*** Actal Plus.

## Alicaforsen *(rINN)*

Alicaforsén; Alicaforsenum. 2′-Deoxy-(*R*)-*P*-thioguanylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioadenylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioadenylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioguanylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiothymidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioguanylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioguanylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioadenylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiothymidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thioguanylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiothymidylyl-(3′→5′)-2′-deoxy-(*R*)-*P*-thiocytidylyl-(3′→5′)-2′-deoxyadenosine.

Аликафорсен

$C_{192}H_{244}N_{75}O_{98}P_{19}S_{19}$ = 6368.2.

*CAS — 185229-68-9.*

## Alicaforsen Sodium *(USAN, rINNM)*

Alicaforsen Nonadecasodium; Alicaforsén sódico; Alicaforsen Sodique; Alicaforsenum Natricum; Isis-2302.

Аликафорсен Натрий

$C_{192}H_{225}N_{75}Na_{19}O_{98}P_{19}S_{19}$ = 6785.8.

*CAS — 331257-52-4.*

### Profile

Alicaforsen is an antisense oligonucleotide that inhibits the production of the cellular adhesion molecule ICAM-1, which plays a role in the inflammatory response involved in inflammatory bowel disease. It has been tried in ulcerative colitis and pouchitis, and with less success in Crohn's disease.

◊ References.


1. Barish CF. Alicaforsen therapy in inflammatory bowel disease. *Expert Opin Biol Ther* 2005; **5:** 1387–91.
2. Miner PB, *et al.* Safety and efficacy of two dose formulations of alicaforsen enema compared with mesalazine enema for treatment of mild to moderate left-sided ulcerative colitis: a randomized, double-blind, active-controlled trial. *Aliment Pharmacol Ther* 2006; **23:** 1403–13. Correction. *ibid.*; **24:** 1268.
3. van Deventer SJ, *et al.* A phase II dose ranging, double-blind, placebo-controlled study of alicaforsen enema in subjects with acute exacerbation of mild to moderate left-sided ulcerative colitis. *Aliment Pharmacol Ther* 2006; **23:** 1415–25.
4. Miner PB, *et al.* Bioavailability and therapeutic activity of alicaforsen (ISIS 2302) administered as a rectal retention enema to subjects with active ulcerative colitis. *Aliment Pharmacol Ther* 2006; **23:** 1427–34.
5. Yacyshyn B, *et al.* A randomized, double-masked, placebo-controlled study of alicaforsen, an antisense inhibitor of intercellular adhesion molecule 1, for the treatment of subjects with active Crohn's disease. *Clin Gastroenterol Hepatol* 2007; **5:** 215–20.

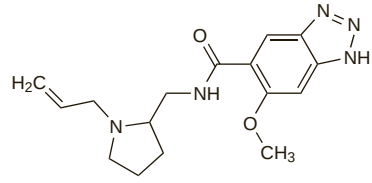

## Alizapride Hydrochloride *(rINNM)*

Alizapride, Chlorhydrate d'; Alizapridi Hydrochloridum; Hidrocloruro de alizaprida. *N*-(1-Allyl-2-pyrrolidinylmethyl)-6-methoxy-1*H*-benzotriazole-5-carboxamide hydrochloride.

Ализаприда Гидрохлорид

$C_{16}H_{21}N_5O_2$,HCl = 351.8.

*CAS — 59338-93-1 (alizapride); 59338-87-3 (alizapride hydrochloride).*
*ATC — A03FA05.*
*ATC Vet — QA03FA05.*



*(alizapride)*

### Adverse Effects and Precautions

As for Metoclopramide, (see p.1748).