

Metoclopramide

Date of monograph revision: 29-Apr-2004; 10-May-2004; 08-Feb-2005; 20-Jul-2006; 21-May-2007; 28-Jul-2008; 12-Oct-2009; 17-Aug-2010; (latest modification: 26-Feb-2011)

Drug Nomenclature (Latest modification: 07-Feb-2011)

Synonyms: Metoclopramida; Métoclopramide; Metoclopramidum; Metoklopramid; Metoklopramidas; Metoklopramidi

BAN: Metoclopramide

INN: Metoclopramide [rINN (en)]

INN: Metoclopramida [rINN (es)]

INN: Métoclopramide [rINN (fr)]

INN: Metoclopramidum [rINN (la)]

INN: Метоклопрамид [rINN (ru)]

INN: مېتوكلوپراميد [rINN (ar)]

INN: 甲氧氯普胺 [rINN (cn)]

Chemical name: 4-Amino-5-chloro-*N*-(2-diethylaminoethyl)-2-methoxybenzamide

Molecular formula: C₁₄H₂₂ClN₃O₂ = 299.8

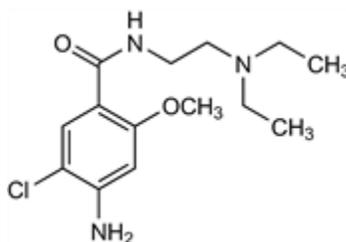
CAS: 364-62-5

ATC code: A03FA01

ATC code (veterinary): QA03FA01

UNII code: L4YEB44I46

Martindale code: 11464-n



Chemical Structure of Metoclopramide

Pharmacopoeias:

In **Chin.**, **Eur.** (see ) , and **Jpn.**

Ph. Eur. 7.1 (Metoclopramide). A white or almost white, fine powder. It exhibits polymorphism. Practically insoluble in water; sparingly soluble to slightly soluble in alcohol; slightly soluble in dichloromethane.

Metoclopramide Hydrochloride

Date of monograph revision: 08-Oct-1996; 04-Mar-1998; 09-Aug-2000; 18-Oct-2001; 10-May-2004; 08-Feb-2005; 20-Jul-2006; 21-May-2007; 28-Jul-2008; 12-Oct-2009; 17-Aug-2010; (latest modification: 26-Feb-2011)

Drug Nomenclature (Latest modification: 07-Feb-2011)

Synonyms: AHR-3070-C; DEL-1267; Metoclopramida, hidrocloruro de; Métoclopramide, chlorhydrate de; Metoclopramidi Hydrochloridum; Metoclopramidi Hydrochloridum Monohydricum; Metoklopramid Hidroklorür; Metoklopramid-hidroklorid; Metoklopramid-hydrochlorid monohydrát; Metoklopramidhydroklorid; Metoklopramidihydrokloridi; Metoklopramido hidrochloridas; Metoklopramidu chlorowodorek; MK-745

BAN: Metoclopramide Hydrochloride [BANM]

USAN: Metoclopramide Hydrochloride

INN: Metoclopramide Hydrochloride [rINNM (en)]

INN: Hidrocloruro de metoclopramida [rINNM (es)]

INN: Métoclopramide, Chlorhydrate de [rINNM (fr)]

INN: Metoclopramidi Hydrochloridum [rINNM (la)]

INN: Метоклопрамида Гидрохлорид [rINNM (ru)]

Molecular formula: C₁₄H₂₂ClN₃O₂·HCl·H₂O = 354.3

CAS: 7232-21-5 (anhydrous metoclopramide hydrochloride); 54143-57-6 (metoclopramide hydrochloride monohydrate); 2576-84-3 (anhydrous metoclopramide dihydrochloride)

ATC code: A03FA01

Martindale: The Complete Drug Reference

ATC code (veterinary): QA03FA01

UNII code: 7B1QZY5SWZ (anhydrous metoclopramide hydrochloride); W1792A2RVD (metoclopramide hydrochloride monohydrate)

Martindale code: 6541-f

Pharmacopoeias:

In **Eur.** (see ) , **Int.** , and **US** .

Ph. Eur. 7.1 (Metoclopramide Hydrochloride). A white or almost white, crystalline powder or crystals. Very soluble in water; freely soluble in alcohol; sparingly soluble in dichloromethane. A 10% solution in water has a pH of 4.5 to 6.0. Protect from light.

USP 33 (Metoclopramide Hydrochloride). A white or practically white, odourless or practically odourless, crystalline powder. Very soluble in water; freely soluble in alcohol; sparingly soluble in chloroform; practically insoluble in ether. Store in airtight containers. Protect from light.

Physicochemical Characteristics (Latest modification: 29-Apr-2004)

Incompatibility

Proprietary preparations of metoclopramide hydrochloride are stated to be incompatible with cephalothin sodium, chloramphenicol sodium, and sodium bicarbonate.

Cisplatin, cyclophosphamide, doxorubicin hydrochloride, morphine hydrochloride, or diamorphine hydrochloride are stated to be compatible with metoclopramide hydrochloride but compatibility is dependent upon factors such as the particular formulation, drug concentration, and temperature.

Adverse Effects (Latest modification: 15-Jul-2008)

Metoclopramide is a dopamine antagonist and may cause extrapyramidal symptoms (usually acute dystonic reactions); these are more common in children and young adults, and at daily doses above 500 micrograms/kg. Parkinsonism and tardive

dyskinesia have occasionally occurred, usually during prolonged treatment in elderly patients, and especially in elderly women.

Other adverse effects include restlessness, drowsiness, anxiety, and diarrhoea. Hypotension, hypertension, dizziness, headache, and depression may occur and there are isolated reports of blood disorders, hypersensitivity reactions (rash, bronchospasm), and neuroleptic malignant syndrome. Disorders of cardiac conduction have been reported with intravenous metoclopramide.

Metoclopramide stimulates prolactin secretion and may cause galactorrhoea or related disorders. Transient increases in plasma-aldosterone concentrations have been reported.

Effects on the blood

Agranulocytosis was associated with use of metoclopramide on 2 separate occasions.¹ Both episodes resolved within 2 to 3 weeks of withdrawing metoclopramide. Methaemoglobinaemia²⁻⁴ and sulphaemoglobinaemia^{5,6} have also been reported. Methaemoglobinaemia (⇒📖) may be treated with methylthionium chloride. In one case,³ the patient died despite such treatment; blood samples taken before death were found to be haemolysed, and assays showed deficiency in G6PD activity (for the view that methylthionium chloride should not be used in G6PD-deficient patients, see Uses of Methylthionium Chloride, ⇒📖).

1. Harvey RL, Luzar MJ. [Metoclopramide-induced agranulocytosis](#). *Ann Intern Med* 1988; **108**: 214–15. [PubMed](#)
2. Grant SCD, et al. [Methaemoglobinaemia produced by metoclopramide in an adult](#). *Eur J Clin Pharmacol* 1994; **47**: 89. [PubMed](#)
3. Karadsheh NS, et al. [Metoclopramide-induced methemoglobinemia in a patient with co-existing deficiency of glucose-6-phosphate dehydrogenase and NADH-cytochrome b5 reductase: failure of methylene blue treatment](#). *Haematologica* 2001; **86**: 659–60. [PubMed](#)
4. Mérieau E, et al. [Métoclopramide et méthémoglobinémie néonatale](#). *Arch Pediatr* 2005; **12**: 438–41. [PubMed](#)

5. Van Veldhuizen PJ, Wyatt A. [Metoclopramide-induced sulfhemoglobinemia](#). *Am J Gastroenterol* 1995; **90**: 1010–11. [PubMed](#)
6. Aravindhan N, Chisholm DG. [Sulfhemoglobinemia presenting as pulse oximetry desaturation](#). *Anesthesiology* 2000; **93**: 883–4. [PubMed](#)

Effects on the cardiovascular system

There have been reports of hypotension,¹ hypertension,^{2,3} and supraventricular tachycardia⁴ associated with metoclopramide. Bradycardia followed by total heart block,⁵ and sinus arrest,⁶ have also been reported. It was suggested that metoclopramide may have contributed to a prolonged QT interval and torsade de pointes in a patient with heart failure and renal impairment.⁷

1. Park GR. [Hypotension following metoclopramide administration during hypotensive anaesthesia for intracranial aneurysm](#). *Br J Anaesth* 1978; **50**: 1268–9. [PubMed](#)
2. Sheridan C, et al. [Transient hypertension after high doses of metoclopramide](#). *N Engl J Med* 1982; **307**: 1346. [PubMed](#)
3. Filibeck DJ, et al. [Metoclopramide-induced hypertensive crisis](#). *Clin Pharm* 1984; **3**: 548–9. [PubMed](#)
4. Bevacqua BK. [Supraventricular tachycardia associated with postpartum metoclopramide administration](#). *Anesthesiology* 1988; **68**: 124–5. [PubMed](#)
5. Midttun M, Øberg B. [Total heart block after intravenous metoclopramide](#). *Lancet* 1994; **343**: 182–3. [PubMed](#)
6. Malkoff MD, et al. [Sinus arrest after administration of intravenous metoclopramide](#). *Ann Pharmacother* 1995; **29**: 381–3. [PubMed](#)
7. Siddique SM, et al. [Metoclopramide as a possible cause of prolonged QT syndrome and torsade de pointes in a patient with heart failure and renal insufficiency](#). *Ann Intern Med* 2009; **150**: 502–4. [PubMed](#)

Effects on the endocrine system

Aldosteronism

Metoclopramide has been reported to increase plasma-aldosterone concentrations in healthy individuals¹ and in patients with liver cirrhosis and ascites associated with

secondary hyperaldosteronism.² Increased plasma aldosterone after metoclopramide use has also been associated with the development of oedema in a patient with congestive heart failure.³ The metoclopramide-induced aldosterone response was blunted by giving neostigmine beforehand.¹

1. Sommers DK, *et al.* [Effect of neostigmine on metoclopramide-induced aldosterone secretion in man.](#) *Eur J Clin Pharmacol* 1989; **36**: 411–13. [PubMed](#)
2. Mazzacca G, *et al.* [Metoclopramide and secondary hyperaldosteronism.](#) *Ann Intern Med* 1983; **98**: 1024–5. [PubMed](#)
3. Zumoff B. [Metoclopramide and edema.](#) *Ann Intern Med* 1983; **98**: 557. [PubMed](#)

Hyperprolactinaemia

Hyperprolactinaemia, galactorrhoea, and pituitary adenoma occurred in a 49-year-old woman with reflux oesophagitis who had received metoclopramide for 3 months.¹ Her plasma-prolactin concentrations fell to normal and her symptoms resolved over 4 months after withdrawal of metoclopramide. The pituitary tumour was considered to be incidental to, and not caused by, metoclopramide therapy. A 21-year-old female developed mastodynia, galactorrhoea, and hyperprolactinaemia after 2 days of treatment with metoclopramide;² she had previously taken domperidone intermittently without any problems. Her symptoms resolved rapidly on stopping therapy. Galactorrhoea has also been reported³ in a neonate given metoclopramide.

1. Cooper BT, *et al.* [Galactorrhoea, hyperprolactinaemia, and pituitary adenoma presenting during metoclopramide therapy.](#) *Postgrad Med J* 1982; **58**: 314–15. [PubMed](#)
2. Bozzolo M, *et al.* [Medikamentös induzierte Hyperprolaktinämie und Galaktorrhöe.](#) *Schweiz Rundsch Med Prax* 1992; **81**: 1511–13. [PubMed](#)
3. Paturi B, *et al.* [Galactorrhea with metoclopramide use in the neonatal unit.](#) *J Perinatol* 2009; **29**: 391–2. [PubMed](#)

Effects on mental state

There are isolated reports of dose-related delirium, depression, and uncontrollable crying in patients treated with metoclopramide in doses of 40 to 80 mg daily.¹⁻³

Symptoms resolved on reducing the dose or withdrawing metoclopramide, and tolerance could be achieved by gradually increasing the dose. In another report,⁴ a patient developed acute akathisia (see Extrapyrarnidal Effects →) shortly after starting metoclopramide 10 mg four times daily; this was accompanied by restlessness, anxiety, and irritability. Despite stopping the drug after 2 days, and treatment with paroxetine and lorazepam, the patient started to have panic attacks, which increased in frequency and severity; he also developed agoraphobia. Two months after taking metoclopramide he was diagnosed with major depressive disorder, which included intermittent suicidal ideation. Following aggressive therapy and hospitalisation, his symptoms gradually resolved.

Insomnia, with or without daytime drowsiness, has also been reported in patients taking metoclopramide 40 mg daily.⁵

Hypersensitivity psychosis has also been reported, shortly after stopping metoclopramide.⁶ Symptoms included hallucinations, delusional experiences, anxiety, restlessness, and sleep disturbances; improvement and gradual resolution were noted after treatment with risperidone.

1. 1. Bottner RK, Tullio CJ. [Metoclopramide and depression](#). *Ann Intern Med* 1985; **103**: 482. [PubMed](#)
2. 2. Adams CD. [Metoclopramide and depression](#). *Ann Intern Med* 1985; **103**: 960. [PubMed](#)
3. 3. Fishbain DA, Rogers A. [Delirium secondary to metoclopramide hydrochloride](#). *J Clin Psychopharmacol* 1987; **7**: 281–2. [PubMed](#)
4. 4. Anfinson TJ. [Akathisia, panic, agoraphobia, and major depression following brief exposure to metoclopramide](#). *Psychopharmacol Bull* 2002; **36**: 82–93. [PubMed](#)
5. 5. Saxe TG. [Metoclopramide side effects](#). *Ann Intern Med* 1983; **98**: 674. [PubMed](#)
6. 6. Lu M-L, et al. [Metoclopramide-induced supersensitivity psychosis](#). *Ann Pharmacother* 2002; **36**: 1387–90. [PubMed](#)

Extrapyrarnidal effects

The Adverse Reactions Register of the UK CSM for the years 1967 to 1982 contained 479 reports of extrapyramidal reactions in which metoclopramide was the suspected drug: 455 were for dystonic-dyskinetic reactions, 20 for parkinsonism, and 4 for tardive dyskinesia.¹

Acute **dystonic-dyskinetic** reactions occur most commonly in children and young adults¹⁻³ and about 70% of reactions are in females;^{1,3} many are associated with doses above those recommended by the manufacturers.^{1,3,4} Symptoms reported include oculogyric crisis,^{4,5} opisthotonus,⁶ torticollis,^{5,7} trismus,^{5,7} a tetanus-like reaction,⁸ and blue coloration of the tongue;⁶ akathisia after the use of metoclopramide alone,⁹ or with droperidol¹⁰ or haloperidol⁹ has been reported (see also Effects on Mental State, ⇨📖).

These effects usually occur within 72 hours of starting treatment¹ but have been reported within 30 minutes of receiving metoclopramide.⁴ When metoclopramide is given intravenously, the rate may be important in determining the incidence of acute extrapyramidal reactions: a study indicated that akathisia was less frequent when the same dose was given as a 15-minute infusion rather than a 2-minute bolus injection.¹¹ Acute effects can occur in patients who have previously taken metoclopramide without complications^{5,8,10} and may be precipitated by other drugs. Although generally self-limiting, deaths have occurred.^{1,5} The reactions are readily reversed by an antihistamine such as diphenhydramine,⁷ or an antimuscarinic such as benztropine;^{4,6} prophylactic use of diphenhydramine has been suggested for patients with a history of extrapyramidal reactions and in those less than 30 years of age.^{7,8}

Metoclopramide-associated **parkinsonism** is thought to occur less commonly than the acute dystonias and is seen mainly in older patients. Symptoms usually appear several months after starting metoclopramide, but may occur within days or not for several years. Withdrawal of metoclopramide usually results in resolution of symptoms, although it may take several months.¹ A study has suggested that metoclopramide-induced parkinsonism (misdiagnosed as idiopathic Parkinson's disease) may be more common in the elderly than generally realised.¹²

Tardive dyskinesia may rarely be associated with use of metoclopramide. Development of this condition is directly related to the length of treatment with metoclopramide and the number of doses taken.¹³ The reaction is usually confined to elderly patients after prolonged oral use,^{14,15} but it has been reported with short-term high-dose parenteral use as an antiemetic in cancer chemotherapy,¹⁶ and a case has been reported in an 8-year-old child treated with metoclopramide for gastro-oesophageal reflux disease in usual doses.¹⁷ The average duration of treatment before the onset of symptoms was 14 months (range 4 to 44 months) in a report of 11 cases¹⁵ and 26 months (range 8 to 60 months) in a report of 12 cases;¹⁸ some patients did not have symptoms until after withdrawal of metoclopramide. Tardive dyskinesia is potentially irreversible and its management is difficult.¹⁸ Some patients improve after withdrawal of metoclopramide but symptoms persisting during follow-up periods of up to 3 years have been reported.^{14,15} The emphasis must be on prevention, hence the recommendation that metoclopramide should not be prescribed for the long-term treatment of minor symptoms, especially in elderly patients.¹⁸ After analyses suggested that metoclopramide was the most common cause of drug-induced movement disorders and that about 20% of patients who used metoclopramide used it for longer than 3 months, the FDA recommended that chronic use of metoclopramide should be avoided in all patients unless the benefit of treatment is believed to outweigh the risk.¹³

1. Bateman DN, et al. [Extrapyramidal reactions with metoclopramide](#). *BMJ* 1985; **291**: 930–2. [PubMed](#)
2. Anonymous. [Measuring therapeutic risk](#). *Lancet* 1989; **ii**: 139–40. [PubMed](#)
3. Adverse Drug Reactions Advisory Committee. [Metoclopramide—choose the dose carefully](#). *Aust Adverse Drug React Bull* 1990; Feb.
4. Tait P, et al. [Metoclopramide side effects in children](#). *Med J Aust* 1990; **152**: 387. [PubMed](#)
5. Pollera CF, et al. [Sudden death after acute dystonic reaction to high-dose metoclopramide](#). *Lancet* 1984; **ii**: 460–1. [PubMed](#)
6. Alroe C, Bowen P. [Metoclopramide and prochlorperazine: "the blue-tongue sign"](#). *Med J Aust* 1989; **150**: 724–5. [PubMed](#)
7. Kris MG, et al. [Extrapyramidal reactions with high-dose metoclopramide](#). *N Engl J Med* 1983; **309**: 433–4. [PubMed](#)

8. 8. Della Valle R, et al. [Metoclopramide-induced tetanus-like dystonic reaction](#). *Clin Pharm* 1985; **4**: 102–3. [PubMed](#)
9. 9. Akagi H, Kumar TM. [Akathisia: overlooked at a cost](#). *BMJ* 2002; **324**: 1506–7. [PubMed](#)
- 10.10. Barnes TRE, et al. [Acute akathisia after oral droperidol and metoclopramide preoperative medication](#). *Lancet* 1982; **ii**: 48–9. [PubMed](#)
- 11.11. Regan LA, et al. [Slower infusion of metoclopramide decreases the rate of akathisia](#). *Am J Emerg Med* 2009; **27**: 475–80. [PubMed](#)
- 12.12. Avorn J, et al. [Increased incidence of levodopa therapy following metoclopramide use](#). *JAMA* 1995; **274**: 1780–2. [PubMed](#)
- 13.13. FDA. FDA requires boxed warning and risk mitigation strategy for metoclopramide-containing drugs (issued 26th February, 2009). Available at: [online](#) (accessed 15/07/10)
- 14.14. Grimes JD, et al. [Long-term follow-up of tardive dyskinesia due to metoclopramide](#). *Lancet* 1982; **ii**: 563. [PubMed](#)
- 15.15. Wiholm B-E, et al. [Tardive dyskinesia associated with metoclopramide](#). *BMJ* 1984; **288**: 545–7. [PubMed](#)
- 16.16. Breitbart W. [Tardive dyskinesia associated with high-dose intravenous metoclopramide](#). *N Engl J Med* 1986; **315**: 518. [PubMed](#)
- 17.17. Putnam PE, et al. [Tardive dyskinesia associated with use of metoclopramide in a child](#). *J Pediatr* 1992; **121**: 983–5. [PubMed](#)
- 18.18. Orme ML'E, Tallis RC. [Metoclopramide and tardive dyskinesia in the elderly](#). *BMJ* 1984; **289**: 397–8. [PubMed](#)

Hypersensitivity

A patient given intravenous metoclopramide and butylhyoscine for gastrointestinal disturbances developed pruritus, generalised urticaria, hypotension, and loss of consciousness. She was treated with intravenous fluid, antihistamines, and corticosteroids. Skin prick tests revealed a response to metoclopramide, suggesting an IgE-mediated allergic reaction.¹

1. 1. Kerstan A, et al. [Anaphylaxis during treatment of nausea and vomiting: IgE-mediated metoclopramide allergy](#). *Ann Pharmacother* 2006; **40**: 1889–90. [PubMed](#)

Neuroleptic malignant syndrome

Neuroleptic malignant syndrome (NMS, ) has occurred very rarely with metoclopramide. In a report of a case with a fatal outcome, it was noted that 17 additional cases had been published in the years 1978 to 1998, three of which resulted in death.¹ A case report in a 6-month-old female with Freeman-Sheldon syndrome² given metoclopramide suggested that pre-existing fever and myopathy may be predisposing factors. In another case,³ a burns patient, there was some suggestion that the patient may have been susceptible to neuroleptic hyperthermic syndromes such as NMS and malignant hyperthermia. Metoclopramide should be stopped immediately if NMS occurs, and the patient treated urgently with bromocriptine.

1. Nonino F, Campomori A. [Neuroleptic malignant syndrome associated with metoclopramide](#). *Ann Pharmacother* 1999; **33**: 644–5. [PubMed](#)
2. Stein MH, *et al.* [Neuroleptic malignant syndrome induced by metoclopramide in an infant with Freeman-Sheldon syndrome](#). *Anesth Analg* 2006; **103**: 786–7. [PubMed](#)
3. Nachreiner R, *et al.* [Neuroleptic malignant syndrome associated with metoclopramide in a burn patient](#). *J Burn Care Res* 2006; **27**: 237–41. [PubMed](#)

Precautions (Latest modification: 31-Jul-2008)

Metoclopramide should not be used when stimulation of muscular contractions might adversely affect gastrointestinal conditions, as in gastrointestinal haemorrhage, obstruction, perforation, or for a few days after surgery. There have been reports of hypertensive crises in patients with pheochromocytoma given metoclopramide, thus its use is not recommended in such patients; caution is advised in patients with hypertension.

Children, young adults, and the elderly should be treated with care as they are at increased risk of extrapyramidal reactions; in the UK, use of metoclopramide is restricted in patients under 20 years (see Administration in Children, ). Patients on prolonged therapy should be reviewed regularly. Care should also be taken when

metoclopramide is given to patients with renal or hepatic impairment, epilepsy, Parkinson's disease, or a history of depression, atopy (including asthma), or porphyria.

Metoclopramide may cause drowsiness or impaired reactions; patients so affected should not drive or operate machinery.

Breast feeding

Metoclopramide is excreted into breast milk. The American Academy of Pediatrics¹ considers that the use of metoclopramide by mothers during breast feeding may be of concern, owing to its dopamine-receptor blocking activity. UK licensed product information states that problems in humans have not been reported.

1. American Academy of Pediatrics. [The transfer of drugs and other chemicals into human milk](#). *Pediatrics* 2001; **108**: 776–89. [PubMed](#) [Retired May 2010] Correction. *ibid.*; 1029. Also available at: [online](#) (accessed 07/05/04)

Porphyria

Metoclopramide has been associated with acute attacks of porphyria and is considered to be unsafe in porphyric patients, although there is conflicting evidence of porphyrinogenicity, and indeed some have used it successfully in the management of acute attacks.¹

1. Elder GH, *et al.* [Metoclopramide and acute porphyria](#). *Lancet* 1997; **350**: 1104.

Pregnancy

Metoclopramide is often used to treat nausea and vomiting in pregnancy. A large retrospective cohort study confirmed that exposure to metoclopramide in the first trimester was not associated with adverse effects on the fetus including congenital malformations, perinatal death, and low birth-weight.¹

1. Matok I, *et al.* [The safety of metoclopramide use in the first trimester of pregnancy](#). *N Engl J Med* 2009; **360**: 2528–35. [PubMed](#)

Interactions (Latest modification: 05-Sep-2009)

Metoclopramide should be used with caution in patients taking other drugs that can also cause extrapyramidal reactions, such as the phenothiazines. Increased toxicity may occur if metoclopramide is given to patients receiving lithium, and caution is advisable with other centrally active drugs such as antiepileptics. Metoclopramide should be used with care with other drugs acting at central dopamine receptors, such as pergolide. Combining metoclopramide with CNS depressant drugs can lead to increased sedative effects. Antimuscarinics and opioid analgesics antagonise the gastrointestinal effects of metoclopramide.

The absorption of other drugs may be affected by metoclopramide; it may either diminish absorption from the stomach (as with digoxin) or enhance absorption from the small intestine (for example, with alcohol, ciclosporin, levodopa, aspirin, or paracetamol). It inhibits serum cholinesterase and may prolong neuromuscular blockade produced by suxamethonium (see [↔](#)) and mivacurium ([↔](#)). Metoclopramide may also increase prolactin blood concentrations and therefore interfere with drugs which have a hypoprolactinaemic effect such as bromocriptine (but see [↔](#)). It has been suggested that it should not be given to patients receiving MAOIs.

Antidepressants

For reference to extrapyramidal effects and other adverse effects suggestive of serotonin syndrome in patients taking metoclopramide with SSRIs or venlafaxine, see Gastrointestinal Drugs under Interactions for Fluoxetine ([↔](#)) and Venlafaxine ([↔](#)) respectively.

Carbamazepine

For a report of neurotoxicity associated with the use of metoclopramide with carbamazepine, see [↔](#).

Hydroxyzine

Acute anxiety, rigidity, generalised tremor, opisthotonus, and hypertension developed in a 20-year-old man given metoclopramide 10 mg intravenously and hydroxyzine 100 mg intramuscularly.¹ The adverse effects occurred 30 minutes after giving hydroxyzine and it was suggested that hydroxyzine potentiated the onset of the reaction.

1. Fouilladieu JL, *et al.* [Possible potentiation by hydroxyzine of metoclopramide's undesirable side effects.](#) *Anesth Analg* 1985; **64**: 1227–8. [PubMed](#)

Pharmacokinetics (Latest modification: 28-Mar-2007)

Metoclopramide is rapidly and almost completely absorbed from the gastrointestinal tract after oral doses, although conditions such as vomiting or impaired gastric motility may reduce absorption. However, it undergoes hepatic first-pass metabolism, which varies considerably between subjects, and hence absolute bioavailability and plasma concentrations are subject to wide interindividual variation. On average, the bioavailability of oral metoclopramide is about 80%, but it varies between about 30 and 100%. Peak plasma concentrations of metoclopramide occur about 1 to 2 hours after an oral dose. Bioavailability is equally variable after rectal or intranasal doses, although it may be somewhat better if the drug is given intramuscularly. Metoclopramide is weakly bound to plasma proteins (13 to 30%).

Metoclopramide is widely distributed in the body, and readily crosses the blood-brain barrier into the CNS. It also freely crosses the placenta, and has been reported to attain concentrations in fetal plasma about 60 to 70% of those in maternal plasma. Concentrations in breast milk may be higher than those in maternal plasma, particularly in the early puerperium, although concentrations decrease somewhat in the late puerperium.

Elimination of metoclopramide is biphasic, with a terminal elimination half-life of about 4 to 6 hours, although this may be prolonged in renal impairment, with consequent elevation of plasma concentrations. It is excreted in the urine, about 85% of a dose being eliminated in 72 hours, 20% as unchanged metoclopramide and the remainder as

sulfate or glucuronide conjugates, or as metabolites. About 5% of a dose is excreted in faeces via the bile.

Uses and Administration (Latest modification: 05-Aug-2010)

Metoclopramide hydrochloride is a substituted benzamide used for its prokinetic and antiemetic properties. It stimulates the motility of the upper gastrointestinal tract without affecting gastric, biliary, or pancreatic secretion and increases gastric peristalsis, leading to accelerated gastric emptying. Duodenal peristalsis is also increased which decreases intestinal transit time. The resting tone of the gastro-oesophageal sphincter is increased and the pyloric sphincter is relaxed. Metoclopramide possesses parasympathomimetic activity as well as being a dopamine-receptor antagonist with a direct effect on the chemoreceptor trigger zone. It may have serotonin-receptor (5-HT₃) antagonist properties.

Metoclopramide is used in disorders of decreased gastrointestinal motility (↔📖) such as gastroparesis or ileus; in gastro-oesophageal reflux disease (↔📖) and dyspepsia (↔📖); and in nausea and vomiting (↔📖) associated with various gastrointestinal disorders, with migraine, after surgery, and with cancer therapy. Metoclopramide is of no value in the prevention or treatment of motion sickness. It may be used to stimulate gastric emptying during radiographic examinations, to facilitate intubation of the small bowel, and in the management of aspiration syndromes (↔📖).

It is usually given as the hydrochloride monohydrate with doses expressed as the anhydrous hydrochloride. In the USA, the strength of preparations of the hydrochloride monohydrate is usually expressed in terms of the base. Metoclopramide hydrochloride 10.5 mg is equivalent to about 10.0 mg of the anhydrous substance, which is equivalent to about 8.9 mg of the anhydrous base.

For most purposes the total daily dose should not exceed 500 micrograms/kg; dosage reduction is recommended in renal and perhaps hepatic impairment (see Administration in Hepatic or Renal Impairment, ↔📖).

In the UK, the recommended dose, given orally, intramuscularly, or by slow intravenous injection, is 10 mg (expressed as anhydrous metoclopramide hydrochloride) three times daily.

In the USA, the recommended oral dose is 10 to 15 mg (expressed as the base) up to four times daily.

Single doses of 10 to 20 mg (expressed as anhydrous base or anhydrous hydrochloride) may be given orally, intravenously, or intramuscularly when required, such as before a diagnostic procedure.

Modified-release preparations of metoclopramide are available; 15 mg (expressed as anhydrous metoclopramide hydrochloride) is given twice daily.

In the UK, the use of metoclopramide is restricted in patients under 20 years of age (see Administration in Children, )

An intranasal formulation of metoclopramide is available in some countries.

The base, and salts including the dihydrochloride and the glycyrrhizate have also been used.

High-dose therapy. High doses of metoclopramide have been used, often with other drugs such as dexamethasone, in the treatment of the nausea and vomiting associated with cancer chemotherapy. In the UK, the loading dose of metoclopramide before cancer therapy is 2 to 4 mg/kg given as a continuous intravenous infusion over 15 to 20 minutes; it is followed by a maintenance dose of 3 to 5 mg/kg, again as a continuous intravenous infusion, given over 8 to 12 hours. Alternatively, initial doses of up to 2 mg/kg by intravenous infusion over at least 15 minutes may be given before cancer therapy and repeated every 2 hours. The total dosage by either continuous or intermittent infusion should not normally exceed 10 mg/kg in 24 hours. In the USA, dosing is started about 30 minutes before cancer chemotherapy; an initial dose of 2 mg/kg for highly emetogenic chemotherapy, or 1 mg/kg for less emetogenic

chemotherapy, is given by intravenous infusion over at least 15 minutes. This dose is repeated every 2 hours for 2 doses, then every 3 hours for 3 doses.

Administration

Intranasal

References.

1. Ormrod D, Goa KL. [Intranasal metoclopramide](#). *Drugs* 1999; **58**: 315–22. [PubMed](#) Commentaries. *ibid.*; 323–4.

Administration in children

In the UK, the use of metoclopramide is restricted in patients under 20 years of age to severe intractable vomiting of known cause, chemotherapy- or radiotherapy-induced vomiting, as an aid to gastrointestinal intubation, and in premedication. Licensed oral and parenteral (intramuscular or intravenous) doses for all indications, except premedication, are:

for those aged 15 to 19 years and weighing 60 kg and over: 10 mg three times daily

15 to 19 years (30 to 59 kg): 5 mg three times daily

9 to 14 years (30 kg and over): 5 mg three times daily

5 to 9 years (20 to 29 kg): 2.5 mg three times daily

3 to 5 years (15 to 19 kg): 2 mg two or three times daily

1 to 3 years (10 to 14 kg): 1 mg two or three times daily

under 1 year (up to 10 kg): 1 mg twice daily

Where body-weight is below that specified for a given age group, the dose should reflect the weight rather than the age, so that a lower dose is chosen.

The [BNFC 2009](#) gives similar doses, but also specifies:

1 month to 1 year (up to 10 kg): 100 micrograms/kg (maximum 1 mg) twice daily
neonate: 100 micrograms/kg every 6 to 8 hours (given orally or by intravenous injection only)

The latter dose has also been suggested by the [BNFC 2009](#) for use as a prokinetic in neonates, although metoclopramide is unlicensed in the UK for this indication.

For **premedication** in diagnostic procedures, licensed doses (for use as a single dose orally or parenterally 5 to 10 minutes before the examination and subject to the same body-weight considerations as above) are:

for those aged 15 to 19 years: 10 mg

9 to 14 years: 5 mg

5 to 9 years: 2.5 mg

3 to 5 years: 2 mg

under 3 years: 1 mg

The [BNFC 2009](#) gives similar oral doses, but also specifies:

1 month to 3 years (up to 14 kg): 100 micrograms/kg (maximum 1 mg)

In the USA metoclopramide is licensed for use in children only to facilitate small bowel intubation in those resistant to conventional methods. A single intravenous dose, based on age, is recommended as follows:

over 14 years: 10 mg

6 to 14 years: 2.5 to 5 mg

under 6 years: 100 micrograms/kg

Administration in hepatic or renal impairment

Total clearance of metoclopramide is significantly reduced in patients with **renal impairment**¹⁻³ and the elimination half-life is prolonged to up to 19 hours.² This may be due to impaired metabolism^{1,2} or to an alteration in enterohepatic circulation of metoclopramide.¹ Accumulation of metoclopramide could therefore occur in renal impairment with a possible increased risk of adverse effects. Dosage reductions of at least 50% have therefore been recommended in patients with moderate to severe renal impairment.^{1,2} US licensed product information recommends this dose reduction in those patients whose creatinine clearance is below 40 mL/minute; UK information advises dose reduction in significant renal impairment, but gives no guidance on appropriate doses.

Patients undergoing haemodialysis do not require dosage supplements since relatively little metoclopramide is cleared by this process.^{2,3}

UK licensed product information also recommends that the dose of metoclopramide should be reduced in patients with clinically significant **hepatic impairment**, although again no recommendations on the size of the reduction are given; caution is advised in liver disease, due to loss of conjugation. The US product information notes that metoclopramide undergoes minimal hepatic metabolism and has been used safely in some patients with advanced liver disease (whose renal function was normal), and it makes no such recommendation for reduction of dose. Decreases in clearance and increases in half-life and area under the plasma concentration-time curve have been reported in patients with cirrhosis given metoclopramide.^{4,5}

1. Bateman DN, et al. [The pharmacokinetics of single doses of metoclopramide in renal failure.](#) *Eur J Clin Pharmacol* 1981; **19**: 437–41. [PubMed](#)
2. Lehmann CR, et al. [Metoclopramide kinetics in patients with impaired renal function and clearance by haemodialysis.](#) *Clin Pharmacol Ther* 1985; **37**: 284–9. [PubMed](#)
3. Wright MR, et al. [Effect of haemodialysis on metoclopramide kinetics in patients with severe renal failure.](#) *Br J Clin Pharmacol* 1988; **26**: 474–7. [PubMed](#)

4. Hellstern A, *et al.* [Absolute bioavailability of metoclopramide given orally or by enema in patients with normal liver function or with cirrhosis of the liver.](#) *Arzneimittelforschung* 1987; **37**: 733–6. [PubMed](#)
5. Magueur E, *et al.* [Pharmacokinetics of metoclopramide in patients with liver cirrhosis.](#) *Br J Clin Pharmacol* 1991; **31**: 185–7. [PubMed](#)

Blood disorders

Responses to treatment with metoclopramide have been reported in patients with Diamond-Blackfan anaemia, probably through induction of prolactin release, although the mechanism by which prolactin affects erythropoiesis is unclear.^{1,2} In a pilot study, 3 out of 9 evaluable patients responded after 12 to 15 weeks of therapy; high serum ferritin, pituitary dysfunction, male sex, and younger age may have contributed to the poor response to metoclopramide in other patients.¹ In another case, response was seen by the fourth week of treatment; at the time of the report, the patient had remained asymptomatic and transfusion independent for 8 months.²

1. Abkowitz JL, *et al.* [Response of Diamond-Blackfan anemia to metoclopramide: evidence for a role for prolactin in erythropoiesis.](#) *Blood* 2002; **100**: 2687–91. [PubMed](#)
2. Akiyama M, *et al.* [Successful treatment of Diamond-Blackfan anemia with metoclopramide.](#) *Am J Hematol* 2005; **78**: 295–8. [PubMed](#)

Gastroenteritis

For mention of the use of antiemetics such as metoclopramide to reduce vomiting in acute gastroenteritis, see Ondansetron, .

Hiccup

Metoclopramide has been used in the management of intractable hiccup. For a discussion of hiccup and its management see .

Lactation induction

Metoclopramide has been used^{1,2} in doses of 10 mg three times daily for its dopamine antagonist properties to stimulate lactation in women who wish to breast feed and in whom mechanical stimulation of the nipple alone is inadequate, including mothers of adopted babies or babies born to surrogates.^{3,4} However, pharmacological lactation induction should be viewed as adjunctive to mechanical methods and the duration of therapy should probably be limited to 7 to 14 days.^{1,2} In addition, the efficacy of metoclopramide in stimulating lactation in women with preterm deliveries has been questioned by a controlled study.⁵ Young women are at increased risk of extrapyramidal effects from metoclopramide—see under Adverse Effects, [↗📄](#). There has also been concern about the presence of the drug in breast milk. For a discussion of lactation inhibition and induction, see [↗📄](#).

1. Anderson PO, Valdés V. [Increasing breast milk supply](#). *Clin Pharm* 1993; **12**: 479–80. [PubMed](#)
2. Gabay MP. [Galactogogues: medications that induce lactation](#). *J Hum Lact* 2002; **18**: 274–9. [PubMed](#)
3. Cheales-Siebenaler NJ. [Induced lactation in an adoptive mother](#). *J Hum Lact* 1999; **15**: 41–3. [PubMed](#)
4. Biervliet FP, et al. [Induction of lactation in the intended mother of a surrogate pregnancy: case report](#). *Hum Reprod* 2001; **16**: 581–3. [PubMed](#)
5. Hansen WF, et al. [Metoclopramide effect on breastfeeding the preterm infant: a randomized trial](#). *Obstet Gynecol* 2005; **105**: 383–9. [PubMed](#)

Migraine

Metoclopramide is used in the treatment of migraine ([↗📄](#)) to alleviate nausea and vomiting and gastric stasis, which commonly develop as a migraine attack progresses and can lead to poor absorption of oral antimigraine preparations. It may also be given to counteract nausea and vomiting from the use of ergotamine. Metoclopramide is included in some combination analgesic preparations for the treatment of acute attacks of migraine. In one study, oral lysine aspirin with metoclopramide was as effective as oral sumatriptan in the treatment of migraine.¹ However, a review of combinations of oral metoclopramide plus an analgesic for migraine suggested that they were likely to be

less effective than triptan monotherapy, although they might be an option for patients in whom triptans are contra-indicated or who could not tolerate triptans.² Metoclopramide with sumatriptan may be effective in patients unresponsive to a triptan alone.³

Parenteral metoclopramide has also been shown to be an effective treatment for acute migraine; it reduces pain, and to some extent nausea (although other antiemetics may be more effective), and a systematic review concluded that it should be considered a first-line treatment for migraine in the emergency department.⁴ A later study found intravenous metoclopramide (with intermittent doses of diphenhydramine) to be comparable to subcutaneous sumatriptan in terms of pain relief at both 2 and 24 hours after treatment.⁵

1. Tfelt-Hansen P, et al. [The effectiveness of combined oral lysine acetylsalicylate and metoclopramide compared with oral sumatriptan for migraine.](#) *Lancet* 1995; **346**: 923–6. [PubMed](#)
2. Azzopardi TD, Brooks NA. [Oral metoclopramide as an adjunct to analgesics for the outpatient treatment of acute migraine.](#) *Ann Pharmacother* 2008; **42**: 397–402. [PubMed](#)
3. Schulman EA, Dermott KF. [Sumatriptan plus metoclopramide in triptan-nonresponsive migraineurs.](#) *Headache* 2003; **43**: 729–33. [PubMed](#)
4. Colman I, et al. [Parenteral metoclopramide for acute migraine: meta-analysis of randomised controlled trials.](#) *BMJ* 2004; **329**: 1369–72. [PubMed](#)
5. Friedman BW, et al. [A trial of metoclopramide vs sumatriptan for the emergency department treatment of migraines.](#) *Neurology* 2005; **64**: 463–8. [PubMed](#)

Orthostatic hypotension

Metoclopramide has been tried in the management of some patients with orthostatic hypotension, as mentioned on [⇨](#).

Tourette's syndrome

A small, short-term study in children and adolescents with Tourette's syndrome (see Tics, [⇨](#)) or chronic tic disorders found that treatment with oral metoclopramide (up to 40 mg daily) significantly reduced tic score and severity compared with placebo.¹

1. 1. Nicolson R, *et al.* [A randomized, double-blind, placebo-controlled trial of metoclopramide for the treatment of Tourette's disorder.](#) *J Am Acad Child Adolesc Psychiatry* 2005; **44**: 640–6. [PubMed](#)

Variceal haemorrhage

Metoclopramide 20 mg intravenously controlled bleeding from oesophageal varices within 15 minutes in 10 of 11 patients compared with 4 of 11 patients given placebo; all patients were treated by sclerotherapy.¹ Lower oesophageal sphincter pressure is increased by metoclopramide, thus reducing blood flow to varices and achieving haemostasis; another study² found use of metoclopramide with intravenous glyceryl trinitrate to be more effective than glyceryl trinitrate alone in reducing intravariceal pressure.

For a discussion of variceal haemorrhage and its management, see [↔](#).

1. 1. Hosking SW, *et al.* [Pharmacological constriction of the lower oesophageal sphincter: a simple method of arresting variceal haemorrhage.](#) *Gut* 1988; **29**: 1098–1102. [PubMed](#)
2. 2. Sarin SK, Saraya A. [Effects of intravenous nitroglycerin and nitroglycerin and metoclopramide on intravariceal pressure: a double blind, randomized study.](#) *Am J Gastroenterol* 1995; **90**: 48–53. [PubMed](#)

Preparations (Latest modification: 07-Feb-2011)

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The symbol ✕ denotes a preparation which is discontinued or no longer actively marketed.

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