Dexamfetamine Sulfate *(pINNM)* ⊗

Deksamfetamin Sülfat; Dexamfétamine, Sulfate de; Dexamfetamine Sulphate *(BANM)*; Dexamfetamini Sulfas; Dexamphetamine Sulphate; Dexamphetamini Sulfas; Dextro Amphetamine Sulphate; Dextroamphetamine Sulfate; NSC-73713 (dexamfetamine); Sulfato de dexanfetamina. (*S*)-α-Methylphenethylammonium sulphate; (+)-α-Methylphenethylamine sulphate.

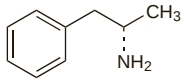
Дексамфетамина Сульфат

$(C_9H_{13}N)_2,H_2SO_4$ = 368.5.

CAS — 51-64-9 (dexamfetamine); 7528-00-9 (dexamfetamine phosphate); 51-63-8 (dexamfetamine sulfate).

ATC — N06BA02.

ATC Vet — QN06BA02.



(dexamfetamine)

NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of dexamfetamine: Dex; Dexies; Dexy; Oranges; Panama; Peaches.

Pharmacopoeias. In *Br.*, *Swiss*, and *US*.

BP 2008 (Dexamfetamine Sulphate). A white or almost white, odourless or almost odourless, crystalline powder. Freely soluble in water; slightly soluble in alcohol; practically insoluble in ether.

USP 31 (Dextroamphetamine Sulfate). A white, odourless, crystalline powder. Soluble 1 in 10 of water and 1 in 800 of alcohol; insoluble in ether. pH of a 5% solution in water is between 5.0 and 6.0.

Adverse Effects

The adverse effects of dexamfetamine are commonly symptoms of overstimulation of the CNS and include insomnia, night terrors, nervousness, restlessness, irritability, and euphoria that may be followed by fatigue and depression. There may be dryness of the mouth, anorexia, abdominal cramps and other gastrointestinal disturbances, headache, dizziness, tremor, sweating, tachycardia, palpitations, myocardial infarction, increased or sometimes decreased blood pressure, altered libido, and impotence. Psychotic reactions, such as hallucinations and delusional thinking, mania, seizures, and stroke have occurred, as has muscle damage with associated rhabdomyolysis and renal complications. Rarely, cardiomyopathy has occurred with chronic use. Sudden death has been reported in patients with structural cardiac abnormalities (see also Effects on the Cardiovascular System, below). In addition in children, growth retardation may occur during prolonged treatment.

In *acute overdosage*, the adverse effects are accentuated and may be accompanied by hyperpyrexia, mydriasis, hyperreflexia, chest pain, cardiac arrhythmias, confusion, panic states, aggressive behaviour, hallucinations, delirium, convulsions, respiratory depression, coma, circulatory collapse, and death. Individual patient response may vary widely and toxic manifestations may occur with quite small overdoses.

Tolerance can develop to some of dexamfetamine's central effects leading to increased doses and habituation. Abrupt cessation after prolonged treatment or abuse of amfetamines has been associated with extreme fatigue, hyperphagia, and depression. However, it is generally accepted that the amfetamines, although widely abused, are not associated with substantial physical dependence.

Abuse of amfetamines for their euphoriant effects has resulted in personality changes, compulsive and stereotyped behaviour, and may induce a toxic psychosis with auditory and visual hallucinations and paranoid delusions.

Abuse. Abuse of amfetamines can lead to toxicity affecting many organs or body systems. There have been reports of *intracerebral haemorrhage*[1-3] and of *cardiomyopathy*.[4-6] *Acute myocardial infarction* has also occurred.[7]

A syndrome characterised by *circulatory collapse*, *fever*, *leukaemoid reaction*, *disseminated intravascular coagulation*, and *rhabdomyolysis* with *diffuse myalgias* and *muscle tenderness* has been described[8] in 5 drug abusers who had used amfetamines or phenmetrazine intravenously. In an earlier study,[9] *necrotising angiitis* was associated with intravenous metamfetamine abuse. A 30-year-old man who had ingested 50 amfetamine sulfate tablets developed *rhabdomyolysis* and *myoglobinuric renal failure*, possibly secondary to a crush syndrome, but in the absence of prolonged coma or other major myotoxic factors.[10] However, *acute interstitial nephritis* and *acute renal failure* have followed oral amfetamine abuse without the associated factors of rhabdomyolysis, hyperpyrexia, or necrotising angiitis.[11]

Chronic use may result in adverse effects such as *hallucinations*, a *delusional disorder* resembling *paranoid schizophrenia*, *stereotyped behaviour*, and *movement disorders*.[12] Although chronic intoxication is the most common precondition for psychosis, individual sensitivities are an important aspect of the drug reaction.

Increased serum concentrations of *levothyroxine* have been associated with heavy amfetamine abuse in 4 psychiatric patients.[13]

Abrupt cessation after prolonged treatment or abuse of amfetamines may cause *extreme fatigue*, *hyperphagia*, and *depression*. *Depressive stupor* has been reported in 3 long-term abusers of amfetamine after sudden withdrawal.[14]

1. Delaney P, Estes M. Intracranial hemorrhage with amphetamine abuse. *Neurology* 1980; **30:** 1125–8.
2. Harrington H, *et al*. Intracerebral hemorrhage and oral amphetamine. *Arch Neurol* 1983; **40:** 503–7.
3. Salanova V, Taubner R. Intracerebral haemorrhage and vasculitis secondary to amphetamine use. *Postgrad Med J* 1984; **60:** 429–30.
4. Smith HJ, *et al*. Cardiomyopathy associated with amphetamine administration. *Am Heart J* 1976; **91:** 792–7.
5. Call TD, *et al*. Acute cardiomyopathy secondary to intravenous amphetamine abuse. *Ann Intern Med* 1982; **97:** 559–60.
6. Hong R, *et al*. Cardiomyopathy associated with the smoking of crystal methamphetamine. *JAMA* 1991; **265:** 1152–4.
7. Waksman J, *et al*. Acute myocardial infarction associated with amphetamine use. *Mayo Clin Proc* 2001; **76:** 323–6.
8. Kendrick WC, *et al*. Rhabdomyolysis and shock after intravenous amphetamine administration. *Ann Intern Med* 1977; **86:** 381–7.
9. Citron BP, *et al*. Necrotizing angiitis associated with drug abuse. *N Engl J Med* 1970; **283:** 1003–11.
10. Scandling J, Spital A. Amphetamine-associated myoglobinuric renal failure. *South Med J* 1982; **75:** 237–40.
11. Foley RJ, *et al*. Amphetamine-induced acute renal failure. *South Med J* 1984; **77:** 258–60.
12. Ellinwood EH, Kilbey MM. Fundamental mechanisms underlying altered behavior following chronic administration of psychomotor stimulants. *Biol Psychiatry* 1980; **15:** 749–57.
13. Morley JE, *et al*. Amphetamine-induced hyperthyroxinemia. *Ann Intern Med* 1980; **93:** 707–9.
14. Tuma TA. Depressive stupor following amphetamine withdrawal. *Br J Hosp Med* 1993; **49:** 361–3.

Effects on the cardiovascular system. There have been very rare reports of sudden death in children and adults taking *Adderall* (*Shire*), a combination preparation of amfetamine salts. In February 2005 Health Canada[1] reported that, worldwide, there have been 20 cases of sudden death in patients taking the product at usual recommended doses; 14 of these deaths occurred in children. In some cases there was no history of cardiac disorders nor any structural abnormalities. Health Canada considered the incidence of fatal adverse effects to be greater with this combination preparation than with other stimulants and consequently withdrew the product from the Canadian market. However, a subsequent independent review committee recommended[2] that *Adderall* be allowed back onto the Canadian market although they did warn that it should not be used in patients with structural cardiac abnormalities.[3]

The committee also recommended that all stimulants used in attention deficit hyperactivity disorder undergo enhanced postmarketing surveillance and consequently, in May 2006, Health Canada[4] revised the labelling of amfetamine, atomoxetine, dexamfetamine, dexmethylphenidate, and methylphenidate to include warnings about their effects on the cardiovascular system. However, it was also noted that the incidence or reporting rates of serious cardiovascular adverse effects with these drugs, including fatal cases, were no greater than background rates.

1. Health Canada. Health Canada has suspended market authorization of ADDERALL XR (amphetamine salts), a drug approved for Attention Deficit Hyperactivity Disorder (ADHD) in children (issued 9th February, 2005). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/medeff/adderall_xr_hpc-cps-eng.pdf (accessed 11/08/08)
2. Health Canada. Report of the "Adderall XR New Drug Committee" (issued 26th August, 2005). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/prodpharma/ndca_rep_cnma_rap_2005-08-25-eng.pdf (accessed 11/08/08)
3. Shire, Canada. ADDERALL XR and serious adverse events (issued 31st August, 2005). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/medeff/adderall_xr2_hpc-cps-eng.pdf (accessed 11/08/08)
4. Health Canada. Attention deficit hyperactivity disorder (ADHD) drugs: updated and standardized labelling regarding very rare cardiac-related adverse events (issued May 2006). Available at: http://www.hc-sc.gc.ca/dhp-mps/alt_formats/hpfb-dgpsa/pdf/medeff/adhd-tdah_medic_hpc-cps_e.pdf (accessed 21/09/06)

Effects on growth. The Pediatric Subcommittee of the FDA Psychopharmacologic Drugs Advisory Committee reviewed the growth-suppressing effects of stimulant medication in hyperkinetic children.[1] There was reasonable evidence that stimulant drugs, particularly in higher doses, moderately suppressed growth in weight and might have a minor suppressing effect on growth in stature. There were indications that some growth caught up during drug holidays, and that early growth suppression was not evident in adulthood. Careful monitoring during treatment was recommended.

See also under Methylphenidate Hydrochloride, p.2159.

1. Roche AF, *et al*. The effects of stimulant medication on the growth of hyperkinetic children. *Pediatrics* 1979; **63:** 847–50.

Treatment of Adverse Effects

Activated charcoal may be given to delay absorption if the patient presents within 1 hour; gastric lavage may be considered in recent large ingestions. In general the management of overdosage with amfetamines involves supportive and symptomatic therapy. Sedation is usually sufficient. Forced acid diuresis has been advocated to increase amfetamine excretion but is seldom necessary and should only be considered in severely poisoned patients; it requires close supervision and monitoring.

Precautions

Dexamfetamine is contra-indicated in patients with cardiovascular disease including moderate to severe hypertension and those with structural cardiac abnormalities, cardiomyopathy, or advanced arteriosclerosis. It should also not be used in patients with hyperthyroidism, glaucoma, hyperexcitability, or agitated states. Dexamfetamine should not be given to those with a history of drug or alcohol abuse and it should be avoided in pregnant or breast-feeding women.

It should be given with caution to patients with mild hypertension, renal impairment, bipolar disorder, or unstable personality. Behavioural disturbances and thought disorders may be exacerbated in psychotic patients.

Height and weight in children should be monitored as growth retardation may occur.

Care may be needed in certain patients predisposed to tics or Tourette's syndrome as symptoms may be provoked. Dexamfetamine is likely to reduce the convulsive threshold; caution is therefore advised in patients with epilepsy. However, it appears that in some countries amfetamines have been included in antiepileptic preparations containing phenytoin or phenobarbital in an attempt to increase their antiepileptic action. Amfetamines may impair patients' ability to drive or to operate machinery.

Diabetic control should be monitored when central stimulants are used for the control of obesity.

Prolonged high doses may need gradual withdrawal as abrupt cessation may produce fatigue and mental depression.

Abuse. Dexamfetamine is subject to extensive abuse and for this reason its availability is severely curtailed. For adverse effects associated with abuse, see above.

Porphyria. Amfetamines are considered to be unsafe in patients with porphyria although there is conflicting experimental evidence of porphyrinogenicity. Metamfetamine has been associated with acute attacks of porphyria.

Pregnancy. No difference was found in the incidence of severe congenital anomalies between 1824 children of mothers prescribed amfetamines or phenmetrazine during pregnancy and 8989 children of mothers who had not received these drugs.[1] Though an excess of oral clefts was noted in the offspring of mothers prescribed amfetamines, there was no excess of congenital heart disease.[1] This was contrary to a previous suggestion[2] in which congenital heart disease in 184 infants had been studied and a link to maternal dexamfetamine exposure postulated. There has been a report of a bradycardia followed by death in a fetus due to maternal intravenous self-administration of 500 mg of amfetamine.[3]

1. Milkovich L, van den Berg BJ. Effects of antenatal exposure to anorectic drugs. *Am J Obstet Gynecol* 1977; **129:** 637–42.
2. Nora JJ, *et al*. Dexamphetamine: a possible environmental trigger in cardiovascular malformations. *Lancet* 1970; **i:** 1290–1.
3. Dearlove JC, Betteridge TJ. Stillbirth due to intravenous amphetamine. *BMJ* 1992; **304:** 548.

Tourette's syndrome. A review[1] of clinical reports concluded that there was virtually no evidence that central stimulants caused or provoked Tourette's syndrome and weak or inadequate evidence that clinically appropriate doses of central stimulants caused tics in previously asymptomatic patients or exacerbated pre-existing symptoms. However, the authors suggested that there was evidence that high or toxic doses might exacerbate or provoke tics in predisposed patients. Long-term methylphenidate therapy did not appear to exacerbate motor or vocal tics in a study[2] of 34 children with ADHD and chronic multiple tic disorder who were followed up for 2 years. However, the authors did point out that careful clinical monitoring is essential to eliminate the possibility of drug-induced exacerbation in individual patients. In contrast, a report[3] on 15 children who developed Tourette's syndrome while receiving stimulant medication for hyperactivity considered that such therapy was contra-indicated in children with motor tics or diagnosed Tourette's syndrome and should be used with caution in children with a family history of these symptoms. In addition, it suggested that the development of motor tic symptoms in any child given stimulants should be a clear indication for stopping immediately to minimise the possibility of eliciting a full-blown Tourette's syndrome.

1. Shapiro AK, Shapiro E. Do stimulants provoke, cause, or exacerbate tics and Tourette syndrome? *Compr Psychiatry* 1981; **22:** 265–73.
2. Gadow KD, *et al*. Long-term methylphenidate therapy in children with comorbid attention-deficit hyperactivity disorder and chronic multiple tic disorder. *Arch Gen Psychiatry* 1999; **56:** 330–6.
3. Lowe TL, *et al*. Stimulant medications precipitate Tourette's syndrome. *JAMA* 1982; **247:** 1729–31.

Interactions

Dexamfetamine is an indirect-acting sympathomimetic and may interact with a number of other drugs. To avoid precipitating a hypertensive crisis, it should not be given to patients being treated with an MAOI or within 14 days of stopping such treatment. Use of beta blockers with amfetamines may produce severe hypertension. Dexamfetamine may also diminish the effects of other antihypertensives, including guanethidine and similar drugs, and concurrent use should be avoided. Patients receiving amfetamines and tricyclic antidepressants require careful monitoring as the risk of cardiovascular effects including arrhythmias may be increased. The urinary excretion of amfetamines is

reduced by urinary alkalinisers, which may enhance or prolong their effects; excretion is increased by urinary acidifiers.

Amfetamines may delay the absorption of ethosuximide, phenobarbital, and phenytoin. The stimulant effects of amfetamines are inhibited by chlorpromazine, haloperidol, and lithium. Disulfiram may inhibit the metabolism and excretion of amfetamines.

Use of sympathomimetics with volatile liquid anaesthetics such as halothane is associated with an increased risk of cardiac arrhythmias.

Pharmacokinetics

Amfetamines are readily absorbed from the gastrointestinal tract and are distributed into most body tissues with high concentrations in the brain and CSF. They are partially metabolised in the liver but a considerable fraction may be excreted in the urine unchanged. Urinary elimination is pH-dependent and enhanced in acid urine. Amfetamines are distributed into breast milk.

◊ References.

1. Steiner E, *et al*. Amphetamine secretion in breast milk. *Eur J Clin Pharmacol* 1984; **27:** 123–4.
2. de la Torre R, *et al*. Clinical pharmacokinetics of amfetamine and related substances: monitoring in conventional and non-conventional matrices. *Clin Pharmacokinet* 2004; **43:** 157–85.
3. Ilett KF, *et al*. Transfer of dexamphetamine into breast milk during treatment for attention deficit hyperactivity disorder. *Br J Clin Pharmacol* 2007; **63:** 371–5.

Uses and Administration

Dexamfetamine, the dextrorotatory isomer of amfetamine, is an indirect-acting sympathomimetic with alpha- and beta-adrenergic agonist activity. It has a marked stimulant effect on the CNS, particularly the cerebral cortex.

Dexamfetamine is used in the treatment of narcolepsy (p.2148). It is also used in the treatment of attention deficit hyperactivity disorder (p.2148); in the UK, this use is limited to refractory hyperactivity disorders in children. Dexamfetamine has been given in the treatment of obesity (p.2149), although amfetamines are no longer recommended for this indication. Amfetamines have also been used to overcome fatigue but, again, such use is considered undesirable. In some countries dexamfetamine has been tried for motion sickness (p.1700), but safer drugs are available.

Dexamfetamine is generally used as the sulfate and is given by mouth.

In the treatment of **narcolepsy**, the usual initial dose is 5 to 10 mg daily in divided doses, increased if necessary by 5 to 10 mg at weekly intervals to a maximum of 60 mg daily. The lower initial dose of 5 mg daily is recommended for the elderly and any weekly increments should also be restricted to 5 mg in such patients.

In children with **hyperactivity** individualisation of treatment is especially important. Children aged 6 years and over usually start with a dose of 5 mg once or twice daily; the dose may be increased if necessary by 5 mg at weekly intervals to an upper limit of 20 mg daily, although older children might require up to 40 mg or more daily. Although dexamfetamine is licensed for the treatment of children younger than 6 years of age in some countries, including the UK and the USA, many authorities consider that stimulants should not be used in young children.

In the USA, an immediate-release, combination preparation containing dexamfetamine sulfate and saccharate, with amfetamine sulfate and amfetamine aspartate monohydrate (*Adderall*, *Shire*), is licensed for the treatment of narcolepsy and attention deficit hyperactivity disorder. This formulation is given by mouth in doses similar to those for dexamfetamine (see above). A modified-release formulation is also available for the treatment of attention deficit hyperactivity disorder in adults and children. The initial dose in adults is 20 mg of total amfetamine salts once daily. In children, it is given as for dexamfetamine in initial doses of 10 mg of total amfetamine salts once daily increased gradually up to a maximum of 30 mg daily; in older children aged 13 to 17 years the dose may be increased to a maximum of 20 mg once daily after 1 week if necessary.

Preparations

BP 2008: Dexamfetamine Tablets.

Proprietary Preparations (details are given in Part 3)
Canad.: Dexedrine; ***Switz.:*** Dexamin†; ***UK:*** Dexedrine; ***USA:*** Dexedrine; Dextrostat.

Multi-ingredient: ***Canad.:*** Adderall; ***USA:*** Adderall.

Dexfenfluramine Hydrochloride ⊗

(BANM, USAN, rINNM) ⊗

Deksfenfluramiinihydrokloridi; Dexfenfluramine, Chlorhydrate de; Dexfenfluraminhydroklorid; Dexfenfluramini Hydrochloridum; Hidrocloruro de dexfenfluramina; S-5614 (dexfenfluramine). (*S*)-*N*-Ethyl-α-methyl-3-trifluoromethylphenethylamine hydrochloride.

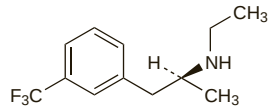
Дексфенфлурамина Гидрохлорид

$C_{12}H_{16}F_3N$,HCl = 267.7.

CAS — 3239-44-9 (dexfenfluramine); 3239-45-0 (dexfenfluramine hydrochloride).

ATC — A08AA04.

ATC Vet — QA08AA04.



(dexfenfluramine)

Profile

Dexfenfluramine is the *S*-isomer of fenfluramine (p.2156). It stimulates the release of serotonin and selectively inhibits its reuptake, but differs from fenfluramine in not possessing any catecholamine agonist activity.

Dexfenfluramine was formerly given orally as the hydrochloride in the treatment of obesity but, like fenfluramine, was withdrawn worldwide after reports of valvular heart defects.

Porphyria. Dexfenfluramine is considered to be unsafe in patients with porphyria because it has been shown to be porphyrinogenic in *in-vitro* systems.

Preparations

Proprietary Preparations (details are given in Part 3)
Cz.: Isolipan†; ***Hung.:*** Isolipan†.

Dexmethylphenidate Hydrochloride

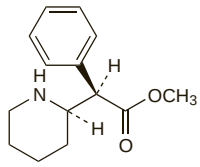
(USAN, rINNM) ⊗

Dexméthylphénidate, Chlorhydrate de; Dexmethylphenidati Hydrochloridum; d-MPH; Hidrocloruro de dexmetilfenidato; *d-threo*-Methylphenidate; *d*-Methylphenidate Hydrochloride. Methyl (2*R*)-phenyl[(2*R*)-piperidin-2-yl]acetate hydrochloride.

Дексметилфенидата Гидрохлорид

$C_{14}H_{19}NO_2$,HCl = 269.8.

CAS — 40431-64-9 (dexmethylphenidate); 19262-68-1 (dexmethylphenidate hydrochloride).



(dexmethylphenidate)

Profile

Dexmethylphenidate hydrochloride is the *d-threo*-enantiomer of racemic methylphenidate hydrochloride (p.2159). It is used as a central stimulant in the treatment of attention deficit hyperactivity disorders.

Dexmethylphenidate hydrochloride is licensed for use in children aged 6 years and older. For patients new to methylphenidate the starting dose of dexmethylphenidate hydrochloride is 2.5 mg twice daily. Each dose should be given at least four hours apart. Dosage may be adjusted in 2.5 to 5 mg increments weekly to a maximum of 10 mg twice daily.

For patients currently using methylphenidate the starting dose of dexmethylphenidate hydrochloride is half the dose of the racemic substance. The maximum recommended dose is 10 mg twice daily.

A modified-release formulation is also available for once-daily dosing.

Dexmethylphenidate should be stopped if there is no improvement in symptoms after appropriate adjustments in dosage over one month. It also needs to be stopped from time to time in those who do respond to assess the patient's condition.

◊ References.

1. Robinson DM, Keating GM. Dexmethylphenidate extended release: in attention-deficit hyperactivity disorder. *Drugs* 2006; **66:** 661–8.

Preparations

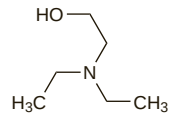
Proprietary Preparations (details are given in Part 3)
USA: Focalin.

Diethylaminoethanol ⊗

Dietilaminoetanol. 2-Diethylaminoethanol.

$C_6H_{15}NO$ = 117.2.

CAS — 100-37-8.



Profile

Diethylaminoethanol is an analogue of deanol (p.2152) and has been used similarly as the malate. The hydrochloride has also been used.

Preparations

Proprietary Preparations (details are given in Part 3)
Gr.: Durobion.

Multi-ingredient: ***Austria:*** Barokaton.

Diethylpropion Hydrochloride *(BANM)* ⊗

Amfepramone Hydrochloride *(pINNM)*; Amfépramone, Chlorhydrate d'; Amfepramoni Hydrochloridum; Hidrocloruro de anfepramona. *N*-(1-Benzoylethyl)-*NN*-diethylammonium chloride; 2-Diethylaminopropiophenone hydrochloride; (*RS*)-α-Diethylaminopropiophenone hydrochloride.

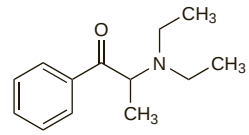
Амфепрамона Гидрохлорид

$C_{13}H_{19}NO$,HCl = 241.8.

CAS — 90-84-6 (diethylpropion); 134-80-5 (diethylpropion hydrochloride).

ATC — A08AA03.

ATC Vet — QA08AA03.



(diethylpropion)

NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of diethylpropion: Blue; Blues.

Pharmacopoeias. In *US*.
USP 31 (Diethylpropion Hydrochloride). A white to off-white, fine crystalline powder. Is odourless or has a slight characteristic odour. It may contain tartaric acid as a stabilising agent. Soluble 1 in 0.5 of water, 1 in 3 of alcohol, and 1 in 3 of chloroform; practically insoluble in ether. Protect from light.

Adverse Effects, Treatment, and Precautions

As for Dexamfetamine Sulfate, p.2153. In addition gynaecomastia has been reported rarely. The incidence of central adverse effects may be lower with diethylpropion than with dexamfetamine. Diethylpropion should not be given to patients with emotional instability or a history of psychiatric illness. It should be avoided in children and the elderly. Diethylpropion hydrochloride is subject to abuse.

Porphyria. Diethylpropion is considered to be unsafe in patients with porphyria because it has been shown to be porphyrinogenic in *animals*.

Interactions

Diethylpropion is an indirect-acting sympathomimetic and, similarly to dexamfetamine (p.2153), may interact with a number of other drugs.

Pharmacokinetics

Diethylpropion is readily absorbed from the gastrointestinal tract. It is extensively metabolised in the liver and possibly the gastrointestinal tract and is excreted in the urine. Diethylpropion crosses the blood-brain barrier and the placenta. Diethylpropion and its metabolites are distributed into breast milk.

Uses and Administration

Diethylpropion hydrochloride is a central stimulant and indirect-acting sympathomimetic with the actions of dexamfetamine (p.2154). It is used as an oral anorectic in the short-term treatment of obesity (p.2149), although stimulants are not generally recommended for this indication.

Doses of 25 mg three times daily 1 hour before meals or 75 mg once daily in mid-morning as a modified-release preparation, are given. To reduce the risk of dependence, diethylpropion should not be given for more than a few weeks at a time.

Regulatory authorities in the EU have called for the withdrawal of diethylpropion from the market (see under Effects on the Cardiovascular System in Fenfluramine, p.2156).