

Bemegride (BAN, rINN)

Bemegrida; Bémégride; Bemegridum. 3-Ethyl-3-methylglutarimide; 4-Ethyl-4-methylpiperidine-2,6-dione.

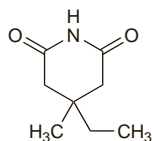
Бемегрид

$C_8H_{13}NO_2 = 155.2$.

CAS — 64-65-3.

ATC — R07AB05.

ATC Vet — QR07AB05.

**Profile**

Bemegride has properties similar to those of doxapram (p.2155). It has been given intravenously as a respiratory stimulant.

Porphyria. Bemegride has been associated with acute attacks of porphyria and is considered unsafe in porphyric patients.

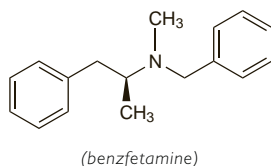
Benzfetamine Hydrochloride (BANM, rINNM) ⊗

Benzfétamine, Chlorhydrate de; Benzfetamini Hydrochloridum; Benzphetamine Hydrochloride; Hidrocloruro de benzfetamina. (+)-N-Benzyl-N,α-dimethylphenethylamine hydrochloride.

Бензфетамина Гидрохлорид

$C_{17}H_{21}N, HCl = 275.8$.

CAS — 156-08-1 (benzfetamine); 5411-22-3 (benzfetamine hydrochloride).



(benzfetamine)

Profile

Benzfetamine hydrochloride is a central stimulant and sympathomimetic with properties similar to those of dexamfetamine (below). It has been used as an anorectic in the treatment of obesity (p.2149), although amfetamines are no longer recommended for this indication. The usual initial oral dose is 25 to 50 mg given once daily, subsequently adjusted, according to requirements, to a dose of 25 to 50 mg up to three times daily.

Preparations

Proprietary Preparations (details are given in Part 3)

USA: Didrex.

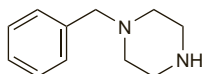
Benzylpiperazine ⊗

N-Benzylpiperazine; 1-Benzylpiperazine. 1-(Phenylmethyl)piperazine.

Бензилпиперазин

$C_{11}H_{16}N_2 = 176.3$.

CAS — 2759-28-6.



NOTE. The following terms have been used as 'street names' (see p.vi) or slang names for various forms of benzylpiperazine: A2; Blast; BZP; Charge; Charlie; Cosmic Kelly; ESP; Euphoria; Exodus; Frenzy; Legal E; Legal X; Nemesis; Pep; Pep Love; Pep Stoned; Pep Twisted; Rapture; The Good Stuff.

Profile

Benzylpiperazine is reported to produce CNS stimulant effects similar to those of the amfetamines (see Dexamfetamine, p.2153) and is subject to abuse.

Other piperazine derivatives subject to abuse include:

- 1-(3,4-methylenedioxybenzyl)piperazine (MDBP)
- 1-(3-trifluoromethylphenyl)piperazine (TFMPP)
- 1-(3-chlorophenyl)piperazine (mCPP)
- 1-(4-methoxyphenyl)piperazine (MeOPP)

◇ References.

1. Gee P, *et al.* Toxic effects of BZP-based herbal party pills in humans: a prospective study in Christchurch, New Zealand. *N Z Med J* 2005; **118**: U1784.
2. Staack RF. Piperazine designer drugs of abuse. *Lancet* 2007; **369**: 1411–13.
3. Johnstone AC, *et al.* Benzylpiperazine: a drug of abuse? *J Psychopharmacol* 2007; **21**: 888–94.
4. Wood DM, *et al.* Collapse, reported seizure—and an unexpected pill. *Lancet* 2007; **369**: 1490.

Catha ⊗

Abyssinian, African, or Arabian Tea; Kat; Kath; Khat; Miraa; Qat; Somali Tea.

Description. Catha consists of the leaves of *Catha edulis* (Celastraceae), and contains cathine, cathinone, celastrin, choline, tannins, and inorganic salts.

The following terms have been used as 'street names' (see p.vi) or slang names for various forms of catha:

Cat; Chat; Feline; Kat; Miraa; Pootie; Qat; Quaadka.

Cathine (pINN) ⊗

Cathinum; Catina; (+)-Norpseudoephedrine. *threo*-2-Amino-1-phenylpropan-1-ol.

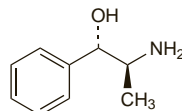
Катин

$C_9H_{13}NO = 151.2$.

CAS — 492-39-7; 36393-56-3.

ATC — A08AA07.

ATC Vet — QA08AA07.

**Cathinone** (pINN) ⊗

Cathinonum; Catinona. (S)-2-Aminopropiophenone.

Катинон

$C_9H_{11}NO = 149.2$.

CAS — 71031-15-7.

Profile

Catha, the leaves of *Catha edulis* (Celastraceae), is used for its stimulant properties among some cultures of Africa and the Middle East, usually by chewing the leaves. Its effects are reported to resemble those of the amfetamines (see Dexamfetamine Sulfate, below), and are thought to be largely due to the content of cathinone. Dependence and psychotic reactions have been reported. Cathine, another constituent, has been used as the hydrochloride as an anorectic.

◇ References to the pharmacology and pharmacokinetics of catha and its constituents¹⁻⁸ and reports of adverse effects.⁹⁻¹⁵

1. Brenneisen R, *et al.* Metabolism of cathinone to (–)-norephedrine and (–)-norpseudoephedrine. *J Pharm Pharmacol* 1986; **38**: 298–300.
2. Brenneisen R, *et al.* Amphetamine-like effects in humans of the khat alkaloid cathinone. *Br J Clin Pharmacol* 1990; **30**: 825–8.
3. Kalix P. Pharmacological properties of the stimulant khat. *Pharmacol Ther* 1990; **48**: 397–416.
4. Kalix P. Chewing khat, an old drug habit that is new in Europe. *Int J Risk Safety Med* 1992; **3**: 143–56.
5. Kalix P. Cathinone, a natural amphetamine. *Pharmacol Toxicol* 1992; **70**: 77–86.
6. Widler P, *et al.* Pharmacodynamics and pharmacokinetics of khat: a controlled study. *Clin Pharmacol Ther* 1994; **55**: 556–62.
7. Kalix P. Catha edulis, a plant that has amphetamine effects. *Pharm World Sci* 1996; **18**: 69–73.
8. Toennes SW, *et al.* Pharmacokinetics of cathinone, cathine and norephedrine after the chewing of khat leaves. *Br J Clin Pharmacol* 2003; **56**: 125–30.
9. Rumpf KW, *et al.* Rhabdomyolysis after ingestion of an appetite suppressant. *JAMA* 1983; **250**: 2112.
10. Gough SP, Cookson IB. Khat-induced schizophreniform psychosis in UK. *Lancet* 1984; **i**: 455.
11. Roper JP. The presumed neurotoxic effects of Catha edulis—an exotic plant now available in the United Kingdom. *Br J Ophthalmol* 1986; **70**: 779–81.
12. Zureikat N, *et al.* Chewing khat slows the oro-caecal transit time. *Gut* 1992; **33** (suppl): S23.
13. Yousef G, *et al.* Khat chewing as a cause of psychosis. *Br J Hosp Med* 1995; **54**: 322–6.
14. Al-Motarreb A, *et al.* Khat chewing and acute myocardial infarction. *Heart* 2002; **87**: 279–280.
15. Al-Motarreb A, *et al.* Khat chewing is a risk factor for acute myocardial infarction: a case-control study. *Br J Clin Pharmacol* 2005; **59**: 574–81.

Preparations

Proprietary Preparations (details are given in Part 3)

Ger.: Antiadiposum X-112 T; **S.Afr.:** Dietene; Eetless; Leanor; Nobese No. 1; Slim 'n Trim; Thinz; **Switz.:** Antiadiposum X-112; Belloform†; Limit-X†; Miniscap†; **Thai:** Mirapront N†.

Multi-ingredient. Mex.: Redotex; Redotex NF.

Clobenzorex Hydrochloride (rINN) ⊗

Clobenzorex, Chlorhydrate de; Clobenzorexi Hydrochloridum; Hidrocloruro de clobenzorex; SD-271-12. (+)-N-(2-Chlorobenzyl)-α-methylphenethylamine hydrochloride.

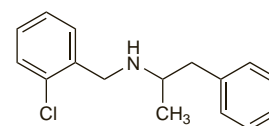
Клобензорекса Гидрохлорид

$C_{16}H_{18}ClN, HCl = 296.2$.

CAS — 13364-32-4 (clobenzorex); 5843-53-8 (clobenzorex hydrochloride).

ATC — A08AA08.

ATC Vet — QA08AA08.



(clobenzorex)

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

Profile

Clobenzorex hydrochloride is a central stimulant and sympathomimetic with properties similar to those of dexamfetamine (below). It has been used as an anorectic in the treatment of obesity (p.2149) but regulatory authorities in the EU have called for the withdrawal of all anorectics from the market (see under Effects on the Cardiovascular System in Fenfluramine, p.2156).

Preparations

Proprietary Preparations (details are given in Part 3)

Mex.: Asenlix; Itravil; Obeclox; Redicres.

Deanol (BAN) ⊗

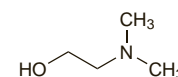
Démanol. 2-Dimethylaminoethanol.

$C_4H_{11}NO = 89.14$.

CAS — 108-01-0 (deanol); 3342-61-8 (deanol aceglumate); 3635-74-3 (deanol acetamidobenzoate); 968-46-7 (deanol benzilate); 71-79-4 (deanol benzilate hydrochloride); 15585-86-1 (deanol cyclohexylpropionate); 5988-51-2 (deanol tartrate).

ATC — N06BX04.

ATC Vet — QN06BX04.



NOTE. Deanol Aceglumate is pINN.

Profile

Deanol, a precursor of choline, may enhance central acetylcholine formation. It has been used as a central stimulant in the treatment of hyperactivity in children but its efficacy is not proven. It has been included in preparations used as tonics and for the management of impaired mental function.

It has been used as a variety of salts and esters including deanol aceglumate, deanol acetamidobenzoate, deanol bisorcate, deanol cyclohexylpropionate (cyprodenate; cyprodemanol), deanol hemisuccinate, deanol pidolate, and deanol tartrate. Deanol benzilate (deanol diphenylglycolate; benzacine) has been used as the hydrochloride in antispasmodic preparations.

Preparations

Proprietary Preparations (details are given in Part 3)

Arg.: DM Active; **Belg.:** Actebra†; **Fr.:** Astyl†; **Ger.:** Risatarun; **Ital.:** Rischiani†; **Pol.:** Bimanol; **Port.:** Tonibral†; **Rus.:** Nooderin (Нюоклерин).

Multi-ingredient. Fr.: Acti 5; Debrumyl; **Ger.:** Rowachol comp†; **Port.:** Actilam; Debrumyl; Forticol; Tonice; **Spain:** Anti Anorex Triple; Denubil; **Switz.:** Vigoran†.

Dexamfetamine Sulfate (pINNM) ⊗

Deksamfetamin Sülfat; Dexamfetamine, Sulfate de; Dexamfetamine Sulphate (BANM); Dexamfetamini Sulfas; Dexamphetamine Sulphate; Dexamphetamini Sulfas; Dextro Amphetamine Sulphate; Dextroamphetamine Sulfate; NSC-73713 (dexamfetamine); Sulfato de dexamfetamina. (5)- α -Methylphenethylammonium sulphate; (+)- α -Methylphenethylamine sulphate.

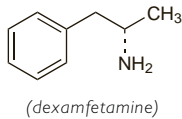
Дексамфетамин Сульфат

(C₉H₁₃N)₂·H₂SO₄ = 368.5.

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

ATC — N06BA02.

ATC Vet — QN06BA02.



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 described⁸ in 5 drug abusers who had used amfetamines or phenmetrazine intravenously. In an earlier study,⁹ *necrotising angitis* 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 angitis.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 hemorrhage 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 angitis 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 Canada¹ 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 recommended² 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 Canada⁴ revised the labelling of amfetamine, atomoxetine, dexamfetamine, dextmethylphenidate, 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 overdose 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 suggestion² 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; **1**: 1290-1.
3. Dearlove JC, Betteridge TJ. Stillbirth due to intravenous amphetamine. *BMJ* 1992; **304**: 548.

Tourette's syndrome. A review¹ 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 study² 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 report³ 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