Adverse Effects, Treatment, and Precautions
As for ACE inhibitors, p.1193.

Interactions
As for ACE inhibitors, p.1196.

Pharmacokinetics
Imidapril acts as a prodrug of the diacid imidaprilat, its active metabolite. After oral doses, imidapril is rapidly but incompletely absorbed; absorption is about 70% and is reduced in the presence of food. Imidapril is metabolised in the liver to imidaprilat. The bioavailability of imidaprilat is about 42% after oral doses of imidapril, and peak plasma concentrations of imidaprilat are reached in about 7 hours. Both imidapril and imidaprilat are moderately bound to plasma proteins. About 40% of an oral dose is excreted in the urine, the rest in the faeces. The terminal half-life of imidaprilat is more than 24 hours. Imidapril and imidaprilat are removed by haemodialysis.

◊ References.

1. Hoogkamer JFW, *et al*. Pharmacokinetics of imidapril and its active metabolite imidaprilat following single dose and during steady state in patients with impaired liver function. *Eur J Clin Pharmacol* 1997; **51:** 489–91.
2. Hoogkamer JFW, *et al*. Pharmacokinetics of imidapril and its active metabolite imidaprilat following single dose and during steady state in patients with chronic renal failure. *Eur J Clin Pharmacol* 1998; **54:** 59–61.
3. Harder S, *et al*. Single dose and steady state pharmacokinetics and pharmacodynamics of the ACE-inhibitor imidapril in hypertensive patients. *Br J Clin Pharmacol* 1998; **45:** 377–80.
4. Tsuruoka S, *et al*. Clearance of imidapril, an angiotensin-converting enzyme inhibitor, during hemodialysis in hypertensive renal failure patients: comparison with quinapril and enalapril. *J Clin Pharmacol* 2007; **47:** 259–63.

Uses and Administration
Imidapril is an ACE inhibitor (p.1193). It is used in the treatment of hypertension (p.1171). Imidapril owes its activity to imidaprilat, to which it is converted after oral doses. The maximum haemodynamic effect occurs 6 to 8 hours after a dose, although the full effect may not develop for several weeks during chronic dosing. Imidapril is given orally as the hydrochloride.

In the treatment of hypertension, the usual initial dose of imidapril hydrochloride is 5 mg once daily, before food. Since there may be a precipitous fall in blood pressure in some patients when starting therapy with an ACE inhibitor, the first dose should preferably be given at bedtime. An initial dose of 2.5 mg daily should be used in the elderly, in patients with renal or hepatic impairment, or in those receiving a *diuretic*; if possible, the diuretic should be withdrawn 2 or 3 days before imidapril is started and resumed later if necessary. The usual maintenance dose is 10 mg daily, although up to 20 mg daily may be given if required. The maximum dose for elderly patients is 10 mg daily.

◊ Reviews.

1. Robinson DM, *et al*. Imidapril: a review of its use in essential hypertension, type 1 diabetic nephropathy and chronic heart failure. *Drugs* 2007; **67:** 1359–78.

Preparations
Proprietary Preparations (details are given in Part 3)

Arg.: Tanatril; ***Austria:*** Tanatril; ***Cz.:*** Tanatril; ***Fin.:*** Tanatril; ***Fr.:*** Tanatril; ***Ger.:*** Tanatril; ***Gr.:*** Tanatril; ***Hong Kong:*** Tanatril; ***India:*** Tanatril; ***Indon.:*** Tanapress; ***Jpn:*** Novarok†; Tanatril; ***Malaysia:*** Tanatril; ***Philipp.:*** Norten; Vascor; ***Pol.:*** Tanatril; ***Port.:*** Cardipril; Tanatril†; ***Singapore:*** Tanatril; ***Spain:*** Hipertene; ***Thai.:*** Tanatril; ***UK:*** Tanatril.

Multi-ingredient: ***Philipp.:*** Norplus; Vascoride.

Indapamide *(BAN, USAN, rINN)* ⊗

Indapamid; Indapamida; Indapamidi; Indapamidum; SE-1520. 4-Chloro-*N*-(2-methylindolin-1-yl)-3-sulphamoylbenzamide.

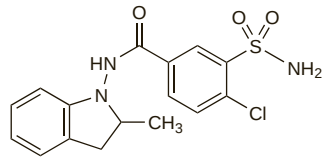
Индапамид

$C_{16}H_{16}ClN_3O_3S$ = 365.8.

CAS — 26807-65-8 (anhydrous indapamide).

ATC — C03BA11.

ATC Vet — QC03BA11.



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

Ph. Eur. 6.2 (Indapamide). A white or almost white powder. Practically insoluble in water; soluble in alcohol. Protect from light.

USP 31 (Indapamide). A white to off-white crystalline powder. Practically insoluble in water; soluble in alcohol, in glacial acetic acid, in acetonitrile, in ethyl acetate, and in methyl alcohol; very slightly soluble in chloroform and in ether.

Adverse Effects, Treatment, and Precautions
As for Hydrochlorothiazide, p.1307.

Effects on the blood. A 58-year-old woman[1] had bleeding from the mucous membrane of the tongue 18 months after starting treatment with a modified-release form of indapamide; she was found to have mild thrombocytopenia, and petechiae were observed. After withdrawal of the drug, bleeding stopped immediately; the platelet count returned to normal within 10 days and the skin lesions faded quickly.

1. Hasanova EA, Agasiyeva NE. Bleeding associated with indapamide SR therapy. *Ann Pharmacother* 2005; **39:** 199–200.

Effects on carbohydrate and lipid metabolism. Several studies have reported no changes in blood-glucose concentrations during indapamide treatment,[1-3] although elevated concentrations have been reported in individual patients.[4,5] There have been reports of increases in total cholesterol[2] and of no change.[3] No adverse biochemical changes were found in studies[6] of a modified-release preparation.

1. Velussi M, *et al*. Treatment of mild-to-moderate hypertension with indapamide in type II diabetics: midterm (six months) evaluation. *Curr Ther Res* 1988; **44:** 1076–86.
2. Prisant LM, *et al*. Biochemical, endocrine, and mineral effects of indapamide in black women. *J Clin Pharmacol* 1990; **30:** 121–6.
3. Leonetti G, *et al*. Long-term effects of indapamide: final results of a two-year Italian multicenter study in systemic hypertension. *Am J Cardiol* 1990; **65:** 674–714.
4. Slotkoff L. Clinical efficacy and safety of indapamide in the treatment of edema. *Am Heart J* 1983; **106:** 233–7.
5. Beling S, *et al*. Long term experience with indapamide. *Am Heart J* 1983; **106:** 258–62.
6. Weidmann P. Metabolic profile of indapamide sustained-release in patients with hypertension: data from three randomised double-blind studies. *Drug Safety* 2001; **24:** 1155–65.

Effects on electrolyte balance. It has been claimed that indapamide produces few adverse biochemical effects at the usual dose of 2.5 mg daily. However, by 2002, 164 cases of hyponatraemia had been reported to the Australian Adverse Drug Reactions Advisory Committee (ADRAC)[1], of which 68 also described hypokalaemia. Most patients were elderly women. A review[2] of some of these cases suggested that hyponatraemia was more commonly reported with indapamide than with chlorothiazide, although it was pointed out[3] that the true incidence cannot be determined from spontaneous reports. ADRAC recommends that indapamide should be used cautiously. It may be that indapamide has no clinical advantage over low-dose thiazide diuretics.

1. Australian Adverse Drug Reactions Advisory Committee (ADRAC). Indapamide and hyponatraemia. *Aust Adverse Drug React Bull* 2002; **21:** 11. Also available at: http://www.tga.health.gov.au/adr/aadrb/aadr0208.htm (accessed 06/07/04)
2. Chapman MD, *et al*. Hyponatraemia and hypokalaemia due to indapamide. *Med J Aust* 2002; **176:** 219–21.
3. Howes LG. Hyponatraemia and hypokalaemia caused by indapamide. *Med J Aust* 2002; **177:** 53–4.

Effects on the kidneys. Acute interstitial nephritis was associated with indapamide treatment in a 74-year-old patient.[1]

1. Newstead CG, *et al*. Interstitial nephritis associated with indapamide. *BMJ* 1990; **300:** 1344.

Effects on the skin. Sixteen cases of skin rash attributed to indapamide had been reported to the Netherlands Centre for Monitoring of Adverse Reactions to Drugs.[1] All patients had taken indapamide 2.5 mg daily for hypertension. The skin rash was accompanied by fever in 5 cases. In all cases the rash subsided within 14 days of withdrawal, and 11 patients subsequently took thiazides, furosemide, or clopamide without recurrence. Among 188 cases of skin rash attributed to indapamide reported to the WHO Collaborating Centre for International Drug Monitoring were 4 cases of erythema multiforme and 2 of epidermal necrolysis. A further case of toxic epidermal necrolysis was reported by independent authors.[2]

1. Stricker BHC, Biriell C. Skin reactions and fever with indapamide. *BMJ* 1987; **295:** 1313–14.
2. Black RJ, *et al*. Toxic epidermal necrolysis associated with indapamide. *BMJ* 1990; **301:** 1280–1.

Interactions
As for Hydrochlorothiazide, p.1309.

Pharmacokinetics
Indapamide is rapidly and completely absorbed from the gastrointestinal tract. Elimination is biphasic with a half-life in whole blood of about 14 hours. Indapamide is strongly bound to red blood cells. It is extensively metabolised. About 60 to 70% of the dose has been reported to be excreted in the urine; only about 5 to 7% is excreted unchanged. About 16 to 23% of dose is excreted in the faeces. Indapamide is not removed by haemodialysis but does not accumulate in patients with renal impairment.

◊ References.

1. Beermann B, Grind M. Clinical pharmacokinetics of some newer diuretics. *Clin Pharmacokinet* 1987; **13:** 254–66.

Uses and Administration
Indapamide is a diuretic with actions and uses similar to those of the thiazide diuretics (see Hydrochlorothiazide, p.1310) even though it does not contain a thiazide ring system. It is used for hypertension (p.1171), and also for oedema, including that associated with heart failure (p.1165).

In some countries indapamide is described as the hemihydrate. In the treatment of **hypertension** the usual oral dose is 1.25 to 2.5 mg once daily, either alone, or with other antihypertensives; a modified-release preparation may be given in a dose of 1.5 mg daily. At higher doses the diuretic effect may become apparent without appreciable additional antihypertensive effect although US licensed product information suggests that the dose may be increased to 5 mg after 4 weeks. In the treatment of **oedema** the usual dose is 2.5 mg once daily increasing to 5 mg daily after 1 week if necessary.

◊ Reviews.

1. Chaffman M, *et al*. Indapamide: a review of its pharmacodynamic properties and therapeutic efficacy in hypertension. *Drugs* 1984; **28:** 189–235.
2. Robinson DM, Wellington K. Indapamide sustained release: a review of its use in the treatment of hypertension. *Drugs* 2006; **66:** 257–71.

Preparations
BP 2008: Indapamide Tablets;
USP 31: Indapamide Tablets.

Proprietary Preparations (details are given in Part 3)

Arg.: Bajaten; Duremid; Natrilix; Noranat; ***Austral.:*** Dapa-Tabs; Indahexal; Insig; Napamide; Natrilix; ***Austria:*** Fludex; ***Belg.:*** Docindapa; Fludex; ***Braz.:*** Indapen; Natrilix; ***Canad.:*** Lozide; ***Chile:*** Indapress; Natrilix; ***Cz.:*** Indap; Rawel; Tertensif; ***Denm.:*** Fludex†; Indacar; Natrilix; ***Fin.:*** Natrilix; ***Fr.:*** Fludex; ***Ger.:*** Inda-Puren; Natrilix; Sicco†; ***Gr.:*** Dixamid†; Fludex; Magniton-R; Transipen; ***Hong Kong:*** Agelan†; Dapa-Tabs; Diflerix; Frumeron; Indalix; Millibar; Natrilix; ***Hung.:*** Apadex; Pretanix; Rawel; ***India:*** Indicontin; Inditor; Lorvas; Natrilix; ***Indon.:*** Natrilix; ***Irl.:*** Agelan†; Inamide†; Natrilix; ***Israel:*** Pamid; ***Ital.:*** Damide; Indaflex; Indamol; Indolin†; Ipamix; Millibar; Natrilix; Pressural; Veroxil; ***Malaysia:*** Dapa; Diflerix; Napamide; Natrilix; Rinalix†; ***Mex.:*** Natrilix; ***Neth.:*** Fludex; ***NZ:*** Napamide; Naplin†; Natrilix; ***Philipp.:*** Natrilix; ***Pol.:*** Apo-Indap; Diuresin; Indapen; Indapres; Indapsan; Indix; Ipres; Rawel; Tertensif; ***Port.:*** Arifon; Fludex; Fluidema; Tandix; Vasodipin; ***Rus.:*** Akripamide (Акрипамид); Arifon (Арифон); Arindap (Ариндап); Indap (Индап); Indiur (Индиур); Ionik (Ионик); Rawel (Равел); Retapres (Регапрес); ***S.Afr.:*** Catexan; Dapamax; Daptril; Hydro-Less; Indalix; Lixamide; Natrilix; ***Singapore:*** Dapa-Tabs; Millibar†; Napamide; Natrilix; Rinalix; ***Spain:*** Extur; Tertensif; ***Switz.:*** Fludapamide; Fludex; ***Thai.:*** Frumeron; Inpamide; Napamide; Natrilix; ***Turk.:*** Flubest; Fludex; Fludin; Flupamid; Flutans; Indamid; Indapen; Indurin; ***UAE:*** Indanorm; ***UK:*** Natrilix; Nindaxa†; ***USA:*** Lozol; ***Venez.:*** Natrilix.

Multi-ingredient: ***Arg.:*** Bipreterax; Preterax; ***Austral.:*** Coversyl Plus; ***Austria:*** Delapride; Predonium; Preterax; ***Belg.:*** Bi Preterax; Coversyl Plus; Preterax; ***Braz.:*** Coversyl Plus; ***Canad.:*** Coversyl Plus; Preterax; ***Cz.:*** Noliprel; Prenewel; Prestarium Combi; Prestarium Neo Combi; ***Denm.:*** Coversyl Comp; ***Fin.:*** Coversyl Comp; ***Fr.:*** Bipreterax; Preterax; ***Ger.:*** Coversum Combi; Preterax; ***Gr.:*** Dinapres; Preterax; ***Hong Kong:*** Predonium; ***Hung.:*** Armix Komb; Armix Prekomb; Co-Prenessa; Coverex Komb; Coverex Prekomb; Noliprel†; Noriplex†; ***India:*** Coversyl Plus; Perigard D; Perigard DF; Tenolol-D†; ***Irl.:*** Bipreterax; Coversyl Plus; Preterax; ***Ital.:*** Atinorm; Delapride; Dinapres; Nor-Pa; Normopress; Prelectal; Preterax; ***Malaysia:*** Coversyl Plus; ***Mex.:*** Preterax; ***Neth.:*** Coversyl Plus; Predonium; Preterax; ***NZ:*** Coversyl Plus; Predonium; ***Philipp.:*** Bi-Preterax; Preterax; ***Pol.:*** Noliprel; Prestarium Plus; ***Port.:*** Bi Predonium; Bi Preterax; Predonium; Preterax; ***Rus.:*** Enzix (Энзикс); Noliprel (Нолипрел); Sonoprel (Сонопрел); ***S.Afr.:*** Bipreterax; Coversyl Plus; Preterax; Prexum Plus; ***Singapore:*** Coversyl Plus; Preterax; ***Spain:*** Bipredonium†; Bipreterax; Preterax; ***Switz.:*** Coversum Combi; Preterax; ***Turk.:*** Coversyl Plus; Preterax; ***UK:*** Coversyl Plus; ***Venez.:*** Bipreterax; Preterax.

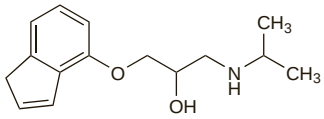
Indenolol Hydrochloride *(BANM, rINNM)* ⊗

Hidrocloruro de indenolol; Indénolol, Chlorhydrate d'; Indenololi Hydrochloridum; Sch-28316Z (indenolol); YB-2.

Инденолола Гидрохлорид

$C_{15}H_{21}NO_2.HCl$ = 283.8.

CAS — 60607-68-3 (indenolol); 68906-88-7 (indenolol hydrochloride).



(indenolol)

Description. Indenolol hydrochloride is a 2:1 tautomeric mixture of 1-(inden-7-yloxy)-3-isopropylaminopropan-2-ol hydrochloride and 1-(inden-4-yloxy)-3-isopropylaminopropan-2-ol hydrochloride.

Pharmacopoeias. In *Jpn.*

Profile

Indenolol is a non-cardioselective beta blocker (p.1225). It is reported to possess potent membrane-stabilising properties and intrinsic sympathomimetic activity.

Indenolol has been used orally as the hydrochloride in the management of various cardiovascular disorders.

Preparations

Proprietary Preparations (details are given in Part 3)
Ital.: Securpres†.

Indobufen *(rINN)*

Indobufén; Indobufène; Indobufenum; K-3920. (±)-2-[4-(1-Oxoisoindolin-2-yl)phenyl]butyric acid.

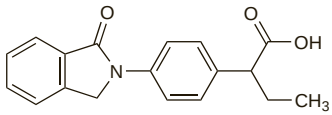
Индобуфен

$C_{18}H_{17}NO_3$ = 295.3.

CAS — 63610-08-2.

ATC — B01AC10.

ATC Vet — QB01AC10.



Profile

Indobufen is an inhibitor of platelet aggregation used in various thromboembolic disorders (p.1187) in oral doses of 200 to 400 mg daily given in 2 divided doses. For patients over the age of 65, the dose should be reduced to 100 to 200 mg daily. Doses should also be reduced in renal impairment (see below). Indobufen has also been given parenterally as the sodium salt.

◊ References.

1. Wiseman LR, *et al.* Indobufen: a review of its pharmacodynamic and pharmacokinetic properties, and therapeutic efficacy in cerebral, peripheral and coronary vascular disease. *Drugs* 1992; **44:** 445–64.
2. Bhana N, McClellan KJ. Indobufen: an updated review of its use in the management of atherothrombosis. *Drugs Aging* 2001; **18:** 369–88.

Administration in renal impairment. In patients with renal impairment the dose of indobufen should be reduced to 100 mg twice daily; it should not be used if the creatinine clearance is under 30 mL/minute.

Preparations

Proprietary Preparations (details are given in Part 3)
Austria: Ibustrin; ***Cz.:*** Ibustrin; ***Ital.:*** Ibustrin; ***Mex.:*** Ibustrin; ***Pol.:*** Ibustrin; ***Port.:*** Ibustrin; ***Venez.:*** Ibustrin.

Indoramin Hydrochloride

(BANM, USAN, rINNM)

Hidrocloruro de indoramina; Indoramine, Chlorhydrate d'; Indoramini Hydrochloridum; Wy-21901 (indoramin). *N*-[1-(2-Indol-3-ylethyl)-4-piperidyl]benzamide hydrochloride.

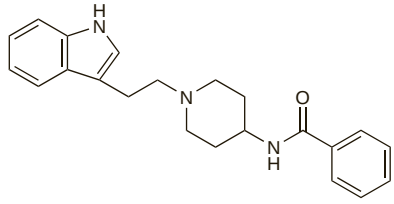
Индорамина Гидрохлорид

$C_{22}H_{25}N_3O.HCl$ = 383.9.

CAS — 26844-12-2 (indoramin); 33124-53-7 (indoramin hydrochloride); 38821-52-2 (indoramin hydrochloride).

ATC — C02CA02.

ATC Vet — QC02CA02.



(indoramin)

Pharmacopoeias. In *Br.*

BP 2008 (Indoramin Hydrochloride). A white or almost white powder. It exhibits polymorphism. Slightly soluble in water; sparingly soluble in alcohol; very slightly soluble in ether; soluble in methyl alcohol. A 2% suspension in water has a pH of 4.0 to 5.5. Protect from light.

Adverse Effects, Treatment, and Precautions

The most common adverse effects in patients receiving indoramin are sedation and dizziness; dry mouth, nasal congestion, headache, fatigue, depression, weight gain (almost certainly due to fluid retention), and failure of ejaculation may also occur. Tachycardia does not seem to be a problem with therapeutic doses but orthostatic hypotension may occur and may produce syncope. Extrapyramidal disturbances have been reported.

After overdosage, coma, convulsions, and hypotension may occur; hypothermia has been reported in *animals*. In acute poisoning appropriate symptomatic and supportive care should be given; if the patient presents within 1 hour, activated charcoal may be considered.

Indoramin should be avoided in patients with heart failure; it has been recommended that incipient heart failure should be controlled before giving indoramin. Caution should be observed in patients with hepatic or renal impairment, a history of depression, epilepsy, or Parkinson's disease. Elderly patients may respond to lower doses.

Because indoramin can cause drowsiness care should be taken in patients who drive or operate machinery.

Cataract surgery. For a warning about intraoperative floppy iris syndrome during cataract surgery in patients taking alpha blockers, see Surgical Procedures, under Precautions for Tamsulosin Hydrochloride, p.2197.

Effects on mental function. Sleep disturbances and vivid dreams were reported during a study in hypertensive patients when indoramin was added to therapy with a thiazide diuretic and a beta blocker.[1]

1. Marshall AJ, *et al.* Evaluation of indoramin added to oxprenolol and bendrofluazide as a third agent in severe hypertension. *Br J Clin Pharmacol* 1980; **10:** 217–21.

Overdosage. A 43-year-old woman with a long history of heavy alcohol intake died after taking 100 tablets of indoramin 25 mg.[1] The main clinical features were deep sedation, respiratory depression, hypotension, and convulsions. Although the hypotension was satisfactorily controlled the CNS effects were resistant to treatment and proved fatal. Other clinical features included areflexia, metabolic acidosis, tachycardia, and later bradyarrhythmias.

1. Hunter R. Death due to overdose of indoramin. *BMJ* 1982; **285:** 1011.

Interactions

The hypotensive effects of indoramin may be enhanced by diuretics and other antihypertensives. It has been reported that the ingestion of alcohol can increase the rate and extent of absorption and the sedative effects of indoramin (see below) and that indoramin should not be given to patients already receiving MAOIs.

Alcohol. In a study[1] in 9 healthy subjects alcohol 500 mg/kg significantly enhanced plasma-indoramin concentrations after an oral dose of 50 mg. The effect was most marked in the early period, corresponding to the absorptive phase. The mean maximum plasma-indoramin concentration was increased from 15.0 to 23.7 nanograms/mL by alcohol; the area under the concentration/time curve was increased by 25%. Alcohol did not affect the pharmacokinetics of intravenous indoramin. The results suggest that alcohol increases indoramin bioavailability either by enhancing absorption or reducing first-pass metabolism. The combination was more sedative than either drug alone.

1. Abrams SML, *et al.* Pharmacokinetic interaction between indoramin and ethanol. *Hum Toxicol* 1989; **8:** 237–41.

Pharmacokinetics

Indoramin is readily absorbed from the gastrointestinal tract and undergoes extensive first-pass metabolism. It is reported to be about 90% bound to plasma proteins. It has a half-life of about 5 hours which is reported to be prolonged in elderly patients. It is extensively metabolised and is excreted mainly as metabolites in the urine and faeces. There is evidence to suggest that some metabolites may have some alpha-adrenoceptor blocking activity.

The elderly. The plasma half-life of indoramin in 5 healthy elderly subjects following a single oral dose ranged from 6.6 to 32.8 hours with a mean of 14.7 hours.[1] The increased half-life may have been caused by reduced clearance in elderly patients.

1. Norbury HM, *et al.* Pharmacokinetics of oral indoramin in elderly and middle-aged female volunteers. *Eur J Clin Pharmacol* 1984; **27:** 247–9.

Uses and Administration

Indoramin is a selective and competitive alpha$_1$-adrenoceptor blocker (p.1153) with actions similar to those of prazosin (p.1376); it is also reported to have membrane-stabilising properties and to be a competitive antagonist at histamine H_1 and 5-hydroxytryptamine receptors. Indoramin is used in the management of hypertension (p.1171), and in benign prostatic hyperplasia (p.2178) to relieve symptoms of urinary obstruction. It has also been used in the prophylactic treatment of migraine.

Indoramin is given orally as the hydrochloride, but doses are usually expressed in terms of the base. Indoramin hydrochloride 11.0 mg is equivalent to about 10 mg of indoramin.

In **hypertension**, the initial dose is 25 mg twice daily, increased in steps of 25 or 50 mg at intervals of 2 weeks to a maximum of 200 mg daily in 2 or 3 divided doses.

In **benign prostatic hyperplasia**, the initial dose is 20 mg twice daily, increased if necessary by 20 mg at 2-week intervals, to a maximum of 100 mg daily in divided doses.

Lower doses may be required in the elderly.

◊ Reviews.

1. Holmes B, Sorkin EM. Indoramin: a review of its pharmacodynamic and pharmacokinetic properties, and therapeutic efficacy in hypertension and related vascular, cardiovascular and airway diseases. *Drugs* 1986; **31:** 467–99.

Migraine. Propranolol is probably the most well-established drug for prophylaxis of migraine (p.616). Many other drugs have been used including indoramin. In a double-blind study,[1] indoramin in a dose of 25 mg twice daily was reported to be as effective as dihydroergotamine mesilate in reducing the frequency of migraine attacks.

1. Pradalier A, *et al.* Etude comparative indoramine versus dihydroergotamine dans le traitement préventif de la migraine. *Therapie* 1988; **43:** 293–7.

Preparations

BP 2008: Indoramin Tablets.

Proprietary Preparations (details are given in Part 3)
Austria: Wypresin†; ***Fr.:*** Vidora; ***Ger.:*** Wydora; ***Irl.:*** Baratol†; Doralese; ***S.Afr.:*** Baratol; ***UK:*** Baratol; Doralese.
