etabonate is sometimes used orally because unlike other quinine salts, which are intensely bitter, it is tasteless. They all contain about the same amount of quinine and any of them can be used when the dose is cited in terms of "quinine salt"; this is not the case for the bisulfate, which contains a correspondingly smaller amount of quinine. Quinine formate is sometimes given parenterally.

A course of treatment with quinine for falciparum malaria usually lasts 7 days and in uncomplicated infections treatment should preferably be given by the oral route. The usual *oral* dose is 600 mg of quinine salt given every 8 hours for 7 days. For children, a dose of 10 mg of quinine salt per kg body-weight given every 8 hours for 7 days is recommended.

In severe or complicated falciparum malaria, or when the patient is unable to take oral medication, quinine should be given parenterally by slow intravenous infusion, but this can be hazardous and patients generally need monitoring, particularly for signs of cardiotoxicity. Therapy should be changed to the oral route as soon as possible to complete the course. To obtain therapeutic concentrations rapidly with parenteral therapy, quinine is often given in an initial loading dose followed by maintenance doses. A recommended *intravenous* dosage regimen suggested by WHO is an initial loading dose of 20 mg of quinine dihydrochloride per kg (up to a maximum of 1.4 g) given over 4 hours with maintenance infusions being started 8 hours later, calculated from the start of the previous infusion. Alternatively, in intensive care units, an initial loading dose of 7 mg/kg may be given over 30 minutes followed immediately by the first of the maintenance infusions. Maintenance infusions consist of 10 mg/kg (up to a maximum of 700 mg) given over 4 hours every 8 hours. A loading dose should not be given if the patient has received quinine, quinidine, mefloquine, or halofantrine, during the previous 24 hours. If parenteral therapy is required for more than 48 hours the maintenance dose of quinine dihydrochloride should be reduced to 5 to 7 mg/kg.

If intravenous infusion is not possible, quinine dihydrochloride has been given *intramuscularly*. Doses, including the loading dose, are the same as those used intravenously; the drug should be diluted in sodium chloride 0.9% to a concentration of 60 to 100 mg of the dihydrochloride per mL, and the total dose divided between two injection sites, preferably each anterior thigh (not the buttock). However, intramuscular injection can be irritant and there have been concerns regarding its safety and efficacy (see under Malaria, below).

When used for the relief of nocturnal **leg cramps**, quinine is given at night in an oral dose of 200 to 300 mg of the sulfate or bisulfate. Quinine benzoate has also been used.

Babesiosis. Although there is no established specific treatment for babesiosis (p.823), a combination of quinine and clindamycin has been used for *Babesia microti* infections.[1,2] For suggested doses see under Clindamycin, p.253. Quinine with azithromycin was reported to be effective in a patient who had not responded to quinine with clindamycin.[3]

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3. Shaio MF, Yang KD. Response of babesiosis to a combined regimen of quinine and azithromycin. *Trans R Soc Trop Med Hyg* 1997; **91:** 214–15.

Flavouring. The Joint FAO/WHO Expert Committee on Food Additives concluded that quinine levels in soft drinks of up to 100 mg/litre (as quinine base) were not of toxicological concern.[1] However, because of the possibility of hypersensitivity reactions in some individuals, the committee recommended that consumers be informed of the presence of quinine in food or beverages.

1. FAO/WHO. Evaluation of certain food additives and contaminants: forty-first report of the joint FAO/WHO expert committee on food additives. *WHO Tech Rep Ser 837* 1993.

Malaria. Quinine has had an important role in the treatment of falciparum malaria (see p.594) being used where there is chloroquine or multidrug *Plasmodium falciparum* resistance,[1-4] and also (in view of the widespread problem of *P. falciparum* resistance) where the infective species is not known, or if the infection is mixed. Treatment should be with one of the quinine salts given orally, with a dose of 600 mg of quinine salt for adults, or 10 mg/kg for children, every 8 hours for 7 days. The dose of quinine salt applies to the hydrochloride, dihydrochloride, sulfate, and etabonate, but not to the bisulfate. Any quinine lost through vomiting within one hour of an oral dose should be replaced by additional doses.[1]

The oral route may not provide effective treatment in severe infection and in such cases quinine should be given as the dihydrochloride by slow intravenous infusion, with the patient being observed closely, particularly for any signs of cardiotoxicity.[4] Loading doses of quinine are often used to obtain therapeutic blood concentrations as soon as possible in severely ill patients but they should not be given to patients who have received quinine, quinidine, mefloquine, or halofantrine, within the previous 24 hours.

WHO[3,4] has given recommendations for intravenous regimens comprising an initial loading dose and maintenance infusions (for details see Uses and Administration, above). Patients are transferred to oral therapy as soon as possible, and treatment continued until a total of at least 7 days of therapy has been given. If intravenous formulations of quinine are unavailable quinidine may be used as an alternative; for further details, see under the Uses and Administration of Quinidine, p.1385.

If facilities for intravenous infusion, including monitoring, are not available quinine may be given by deep intramuscular injection.[4] A loading dose of quinine dihydrochloride 20 mg/kg is given by injection in divided sites followed by injections of 10 mg/kg every 8 hours;[4] a dose interval of 12 hours has also been used. Patients should be transferred to oral therapy as soon as possible. The use of the intramuscular route has been controversial because of concerns over safety and efficacy. However, some studies have shown that it can safely be used in adults and children with severe infections.[5-8] Intramuscular injections of quinine can be irritant and have caused pain, focal necrosis, and abscess formation; fatal tetanus has developed in some patients.[9] It has been suggested that some such reactions may be related to the use of preparations formulated in urethane or other irritant substances. Diluted solutions of quinine dihydrochloride 60 mg/mL adjusted to neutral pH appear to be less painful than the usual undiluted preparation of 300 mg/mL.

If facilities do not exist to give quinine parenterally then patients with severe malaria should receive quinine by mouth or nasogastric tube.

Quinine as formerly standardised used to contain a higher concentration of cinchona alkaloids and there might be synergy between mixtures of these alkaloids.[10] In practice no advantage has been shown by such mixtures over quinine alone in the treatment of chloroquine-resistant falciparum malaria.[11]

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Muscle spasm. Quinine (usually as quinine sulfate or bisulfate) has traditionally been used for nocturnal cramps (p.1887) but there has been concern over its efficacy and potential for adverse effects, especially in the elderly. In the USA, for example, the FDA ruled that quinine products should no longer be used for the management of nocturnal cramps.[1,2] A similar ban has been imposed in Australia.[3] Meta-analyses[4,5] concluded that although quinine was effective in the treatment of nocturnal cramps in ambulatory patients the risk of serious adverse effects should be borne in mind. It was recommended that patients should be closely monitored while the efficacy of quinine is assessed over a period of at least 4 weeks. Some[6] have recommended that treatment be stopped every 3 months to see whether it is still needed. Haemodialysis-induced cramp (p.1671) has been reported to respond to treatment with quinine[7,8] but similar concerns apply.

1. FDA. Drug products for the treatment and/or prevention of nocturnal leg muscle cramps for over-the-counter human use. *Fed Regist* 1994; **59:** 43234–52.
2. Nightingale SL. Quinine for nocturnal leg cramps. *ACP J Club* 1995; **123:** 86.
3. Adverse Drug Reactions Advisory Committee (ADRAC). Quinine indications—cramps deleted. *Aust Adverse Drug React Bull* 2004; **23:** 20. Also available at: http://www.tga.gov.au/adr/aadrb/aadr0410.htm (accessed 01/11/04)
4. Man-Son-Hing M, Wells G. Meta-analysis of efficacy of quinine for treatment of nocturnal leg cramps in elderly people. *BMJ* 1995; **310:** 13–17.
5. Man-Son-Hing M, *et al.* Quinine for nocturnal leg cramps: a meta-analysis including unpublished data. *J Gen Intern Med* 1998; **13:** 600–606.
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Preparations

BP 2008: Quinine Bisulphate Tablets; Quinine Dihydrochloride Intravenous Infusion; Quinine Sulphate Tablets;
USP 31: Quinine Sulfate Capsules; Quinine Sulfate Tablets.

Proprietary Preparations (details are given in Part 3)
Arg.: Circonyl; ***Austral.:*** Biquinate; Myoquin†; Quinate; Quinbisul; Quinoctal†; Quinsul; ***Braz.:*** Palukin; Paluquina†; ***Denm.:*** Kinin; ***Fr.:*** Quinoforme†; Surquina; ***Ger.:*** Limptar N; ***Gr.:*** Kinin†; ***India:*** Cinkona; Quinarsol; Quininga; ***NZ:*** Q200; Q300; ***Swed.:*** Kinin; ***Thai.:*** Genin; ***USA:*** Qualaquin.

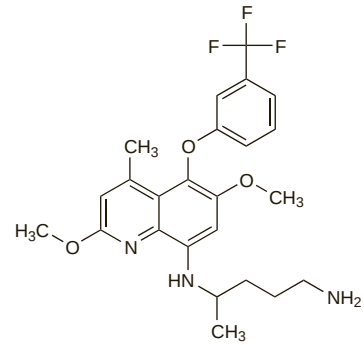
Multi-ingredient: ***Austria:*** Dilatol-Chinin; Iromin-Chinin-C; Limptar; Seltoc; ***Braz.:*** Monotrean; Monotrean B6; ***Fin.:*** Crampiton; Relapamil; ***Fr.:*** Dinacode†; Hexaquine; Okimus; Quinimax; ***Ger.:*** Limptar†; Togal Classic; ***Irl.:*** Anadin; ***Ital.:*** Monotrean†; ***Neth.:*** Aflukin C; ***NZ:*** Nicobrevin; ***Port.:*** Broncosil†; Rectopulmo Adultos†; ***Rus.:*** Analgin-Chinin (Анальгин-Хинин); ***S.Afr.:*** Ilvico; ***Spain:*** Brota Rectal Balsamico; ***UK:*** Nicobrevin.

Tafenoquine *(BAN, rINN)*

Tafenoquina; Tafénoquine; Tafenoquinum; WR-238605. (±)-8-[(4-Amino-1-methylbutyl)amino]-2,6-dimethoxy-4-methyl-5-[(α,α,α-trifluoro-*m*-tolyl)oxy]quinoline; (*RS*)-*N*⁴-[2,6-Dimethoxy-4-methyl-5-(3-trifluoromethylphenoxy)quinolin-8-yl]pentane-1,4-diamine.

Тафенохин

$C_{24}H_{28}F_3N_3O_3$ = 463.5.
CAS — 106635-80-7.



Profile

Tafenoquine is an 8-aminoquinoline antimalarial. It acts as a tissue schizontocide and is under investigation as the succinate for the radical cure and prevention of relapse in vivax malaria. It may also have a role in the prophylaxis of falciparum malaria.

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tient may involve trial and error. Triptans should not be used in patients with major risk factors for, or suffering from, cardiovascular disease. The main concern with all triptans is their potential for coronary vasoconstriction and no triptan appears to be safer than the others.

If **ergotamine** is used it should be given at the first warning of an attack; the earlier it is given, the more effective the treatment. Since its oral bioavailability is poor and may be reduced further during a migraine attack, ergotamine has sometimes been given in sublingual or rectal preparations. Ergotamine can also exacerbate nausea and vomiting; metoclopramide or domperidone, or in severe cases the phenothiazines chlorpromazine or prochlorperazine, may be given. Dihydroergotamine may be of use if parenteral treatment is required; it can also be given intranasally but there is less experience with this route.

Patients who rapidly develop severe migraine may be given **parenteral** dihydroergotamine or sumatriptan. Some consider parenteral metoclopramide to be suitable first-line treatment. If there is no response to these drugs, dopamine antagonists such as chlorpromazine or prochlorperazine given parenterally may be effective in relieving the pain of acute migraine attacks. Prolonged attacks (status migrainosus) may require intravenous administration of dihydroergotamine with metoclopramide.

Other drugs that may be given alone or in combination include corticosteroids or pethidine. Lidocaine has been given intravenously for the emergency treatment of migraine; intranasal lidocaine has also been tried. The opioid agonist-antagonist butorphanol, given by nasal spray, has been advocated, but its place in therapy, if any, remains to be established. Other drugs under investigation include botulinum A toxin and CGRP antagonists; intravenous valproic acid has also shown promise in aborting acute attacks.

Guidelines have been issued for the treatment of migraine in **children and adolescents**. For acute treatment, ibuprofen and paracetamol were found to be effective in children aged 6 years and over; sumatriptan nasal spray should be considered in those aged 12 years and over.

Prophylactic treatment should be considered for patients in whom abortive measures are ineffective or migraine attacks occur frequently, or for those with less frequent but severe or prolonged attacks. Some recommend prophylaxis if attacks occur more often than once or twice a month. Prophylaxis can reduce the severity and/or frequency of attacks but does not eliminate them completely and patients still need additional abortive or symptomatic treatment. Drugs suggested for prophylaxis have a range of actions which reflects uncertainty over the pathogenesis of migraine. It is important to give prophylactic drugs for an adequate period before assessing their efficacy. Once an optimum effect has been achieved the need for continuing prophylaxis should be reviewed at intervals of about 3 to 6 months.

The main prophylactic drugs are **beta blockers**, tricyclic **antidepressants**, and the **antiepileptics**, topiramate and valproate. Propranolol is considered by many to be the prophylactic drug of choice. Lethargy appears to be the most common adverse effect. Other beta blockers reported to be effective are those that, like propranolol, possess no intrinsic sympathomimetic activity, which include atenolol, metoprolol, nadolol, and timolol. The potential for beta blockers to interact with some serotonin ($5\text{-}HT_1$) agonists and ergotamine should be borne in mind. Tricyclic antidepressants, particularly amitriptyline, given in gradually increasing doses at night are useful for preventing migraine, especially in patients who also have depression or tension-type headache, although antimuscarinic adverse effects may occur. Valproate is also used for preventing migraine. Nausea appears to be the most common adverse effect. Topiramate is the main alternative to valproate. Weight loss and paraesthesia are commonly reported adverse effects. Topiramate and valproate are particularly useful in patients who also have epilepsy or bipolar disorder.

Other drugs have been used for the prophylaxis of migraine: pizotifen, an antihistamine and serotonin antagonist, has been widely used but evidence for its efficacy is limited; it may be tried in children. Of the drugs with calcium-channel blocking activity, flunarizine appears to be effective, and has been suggested for use in children, and verapamil may be useful, but evidence for the efficacy of other calcium-channel blockers such as diltiazem, nifedipine, or nimodipine is less convincing; NSAIDs may be worth trying. The use of methysergide, a potent serotonin antagonist, has declined because of serious adverse effects, in particular retroperitoneal fibrosis. MAOIs such as phenelzine have been used occasionally but are best reserved for severe cases refractory to other forms of prophylactic treatment. Cyproheptadine, an antihistamine and serotonin antagonist, has been used for migraine prophylaxis, particularly in children. Other drugs used for the prophylaxis of migraine have included butterbur, clonidine, cyclandelate, indoramin, feverfew, and the ergot derivative metergoline. Positive results have been seen with magnesium and riboflavin. Other drugs still under investigation, which have shown potential for prevention of migraine attacks are: baclofen, botulinum A toxin, candesartan, gabapentin, lisinopril, and venlafaxine.

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Post-dural puncture headache

For the management of headache associated with lumbar puncture or spinal anaesthesia, see Post-dural Puncture Headache under Local Anaesthetics, p.1851.

Tension-type headache

Tension-type headaches, also referred to as muscle-contraction headaches, are probably the commonest form of headache. They are characterised by bilateral pain, which unlike migraine is continuous and non-pulsatile. The pain is often described by the patient as feeling like a tight band pressed around the head. Headaches of this type may be precipitated by many factors including psychosocial stress or muscular stress. Many patients also have associated symptoms of anxiety or depression. Tension-type headaches and migraine often co-exist and may then be referred to as combination or mixed headaches. Some patients only experience isolated acute attacks of tension-type headache (episodic tension-type headache), but others may develop chronic tension-type headache which is difficult to treat.

Treatment is aimed at removing the underlying causes where these can be identified. Simple massage may help if muscle contraction is a prominent component of the pain. Non-opioid analgesics, such as aspirin or other NSAIDs and paracetamol, may be tried for individual acute attacks of headache, but analgesic overuse must be avoided as this can lead to chronic headache resistant to other measures (see Medication-overuse Headache, above). Opioids alone or in combination preparations with other analgesics should also be avoided. Hypnotics or sedatives have sometimes been used in combination preparations with analgesics in the management of tension-type headache that disrupts sleep but, because of the potential for abuse, they should be avoided in chronic headaches. Muscle relaxants appear to have little place in the management of tension-type headache; although some patients may respond, results are generally disappointing. Other drugs that have been tried include valproate and botulinum A toxin. **Prophylaxis** is preferable to regular short-term use of analgesics in controlling chronic tension-type headache. Tricyclic antidepressants, particularly amitriptyline, are generally considered as first choice, although benefit is rarely complete. The mode of action is unclear and appears to be independent of any antidepressant action. In most cases, improvement is seen with low doses, but full antidepressant doses are necessary in the presence of underlying depression. Addition of a beta blocker such as propranolol may sometimes be of benefit for patients with some migraine features.

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Almotriptan Malate *(BANM, USAN, rINNM)*

Almotriptan, Malate d'; Almotriptani Malas; LAS-31416 (almotriptan); Malato de almotriptán; PNU-180638E (almotriptan malate). 1-[({3-[2-(Dimethylamino)ethyl]indol-5-yl}methyl)sulfonyl]pyrrolidine malate (1:1).

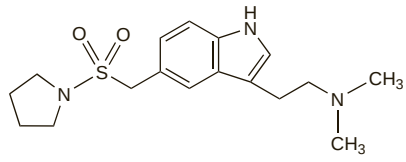
Альмотриптана Малат

$C_{17}H_{25}N_3O_2S,C_4H_6O_5$ = 469.6.

CAS — 154323-57-6 (almotriptan); 181183-52-8 (almotriptan malate).

ATC — N02CC05.

ATC Vet — QN02CC05.



(almotriptan)