TABLE 447-5 CLINICAL STRATIFICATION OF ACUTE SPECIFIC MIGRAINE TREATMENTS

Clinical Situation	Treatment Options
Failed NSAIDs/analgesics	**First tier**
	Sumatriptan 50 mg or 100 mg PO
	Almotriptan 12.5 mg PO
	Rizatriptan 10 mg PO
	Eletriptan 40 mg PO
	Zolmitriptan 2.5 mg PO
	Slower effect/better tolerability
	Naratriptan 2.5 mg PO
	Frovatriptan 2.5 mg PO
	Infrequent headache
	Ergotamine/caffeine 1–2/100 mg PO
	Dihydroergotamine nasal spray 2 mg
Early nausea or difficulties taking tablets	Zolmitriptan 5 mg nasal spray
	Sumatriptan 20 mg nasal spray
	Rizatriptan 10 mg MLT wafer
Headache recurrence	Ergotamine 2 mg (most effective PR/usually with caffeine)
	Naratriptan 2.5 mg PO
	Almotriptan 12.5 mg PO
	Eletriptan 40 mg
Tolerating acute treatments poorly	Naratriptan 2.5 mg
	Almotriptan 12.5 mg
Early vomiting	Zolmitriptan 5 mg nasal spray
	Sumatriptan 25 mg PR
	Sumatriptan 6 mg SC
Menses-related headache	**Prevention**
	Ergotamine PO at night
	Estrogen patches
	Treatment
	Triptans
	Dihydroergotamine nasal spray
Very rapidly developing symptoms	Zolmitriptan 5 mg nasal spray
	Sumatriptan 6 mg SC
	Dihydroergotamine 1 mg IM

Abbreviation: NSAIDs, nonsteroidal anti-inflammatory drugs.

as "possibly" effective in the treatment of migraine. Because the clinical studies demonstrating the efficacy of this combination analgesic in migraine predated the clinical trial methodologies used with the triptans, it is difficult to compare the efficacy of this sympathomimetic compound to other agents.

Nasal A nasal preparation of butorphanol is available for the treatment of acute pain. As with all opioids, the use of nasal butorphanol has little role in migraine treatment.

Parenteral Opioids are modestly effective in the acute treatment of migraine. For example, IV meperidine (50–100 mg) is given frequently in the emergency room. This regimen "works" in the sense that the pain of migraine is eliminated. However, this regimen is clearly suboptimal for patients with recurrent headache. Opioids do not treat the underlying headache mechanism; rather, they act to alter the pain sensation, and there is evidence their use may decrease the likelihood of a response to triptans in the future. Moreover, in patients taking oral opioids, such as oxycodone or hydrocodone, habituation or addiction can greatly confuse the treatment of migraine. Opioid craving and/or withdrawal can aggravate and accentuate migraine. Therefore, it is recommended that opioid use in migraine be limited to patients with severe, but infrequent, headaches that are unresponsive to other pharmacologic approaches or who have contraindications to other therapies.

MEDICATION-OVERUSE HEADACHE

Acute attack medications, particularly opioid or barbiturate-containing compound analgesics, have a propensity to aggravate headache frequency and induce a state of refractory daily or near-daily headache called *medication-overuse headache*. This condition is likely not a separate headache entity but a reaction of the migraine patient to a particular medicine. Migraine patients who have two or more headache days a week should be cautioned about frequent analgesic use (see "Chronic Daily Headache" in Chap. 21).

PREVENTIVE TREATMENTS FOR MIGRAINE

Patients with an increasing frequency of migraine attacks or with attacks that are either unresponsive or poorly responsive to abortive treatments are good candidates for preventive agents. In general, a preventive medication should be considered in the subset of patients with four or more attacks a month. Significant side effects are associated with the use of many of these agents; furthermore, determination of dose can be difficult because the recommended doses have been derived for conditions other than migraine. The mechanism of action of these drugs is unclear; it seems likely that the brain sensitivity that underlies migraine is modified. Patients are usually started on a low dose of a chosen treatment; the dose is then gradually increased, up to a reasonable maximum, to achieve clinical benefit.

Drugs that have the capacity to stabilize migraine are listed in Table 447-6. Drugs must be taken daily, and there is usually a lag of between 2 to 12 weeks before an effect is seen. The drugs that have been approved by the FDA for the prophylactic treatment of migraine include propranolol, timolol, sodium valproate, topiramate, and methysergide (not available). In addition, a number of other drugs appear to display prophylactic efficacy. This group includes amitriptyline, nortriptyline, flunarizine, phenelzine, gabapentin, and cyproheptadine. Placebo-controlled trials of onabotulinum toxin type A in episodic migraine were negative, whereas, overall, placebo-controlled trials in chronic migraine were positive. Phenelzine and methysergide are usually reserved for recalcitrant cases because of their serious potential side effects. Phenelzine is a monoamine oxidase inhibitor (MAOI); therefore, tyramine-containing foods, decongestants, and meperidine are contraindicated. Methysergide may cause retroperitoneal or cardiac valvular fibrosis when it is used for >6 months, and thus monitoring is required for patients using this drug; the risk of fibrosis is about 1:1500 and is likely to reverse after the drug is stopped.

The probability of success with any one of the antimigraine drugs is 50–75%. Many patients are managed adequately with low-dose amitriptyline, propranolol, candesartan, topiramate, or valproate. If these agents fail or lead to unacceptable side effects, second-line agents such as methysergide or phenelzine can be used. Once effective stabilization is achieved, the drug is continued for ~6 months and then slowly tapered to assess the continued need. Many patients are able to discontinue medication and experience fewer and milder attacks for long periods, suggesting that these drugs may alter the natural history of migraine.

TENSION-TYPE HEADACHE

Clinical Features The term *tension-type headache* (TTH) is commonly used to describe a chronic head-pain syndrome characterized by bilateral tight, band-like discomfort. The pain typically builds slowly, fluctuates in severity, and may persist more or less continuously for many days. The headache may be episodic or chronic (present >15 days per month).

A useful clinical approach is to diagnose TTH in patients whose headaches are completely without accompanying features such as nausea, vomiting, photophobia, phonophobia, osmophobia, throbbing, and aggravation with movement. Such an approach neatly separates