nature of the symptoms and helping them develop strategies to achieve relaxation by using forms of cognitive therapy.

Medications for the Alcohol Rehabilitation Treatment Phase

Several medications have modest benefits when used in the first 6–12 months of recovery. The opioid antagonist naltrexone may shorten subsequent relapses, whether used in the oral form (50–150 mg/d) or as a once-per-month 380-mg injection. By blocking opioid receptors, naltrexone decreases activity in the dopamine-rich ventral tegmental reward system and decreases the feeling of pleasure if alcohol is imbibed. A second medication, acamprosate (Campral) (~2 g/d divided into three oral doses), has similar modest effects. Acamprosate inhibits NMDA receptors, decreasing mild symptoms of protracted withdrawal. Several trials of combined naltrexone and acamprosate have reported that the combination is well tolerated and the efficacy might be superior to either drug alone, although not all studies agree.

It is more difficult to establish the asset-to-liability ratio of a third drug, disulfiram, an ALDH inhibitor, used clinically at doses of 250 mg/d, a dose usually selected to avoid the side effects of the more effective 500 mg/d regimen. This drug produces vomiting and autonomic nervous system instability in the presence of alcohol as a result of rapidly rising blood levels of acetaldehyde. This reaction can be dangerous, especially for patients with heart disease, stroke, diabetes mellitus, or hypertension. The drug itself carries potential risks of temporary depressive or psychotic symptoms, peripheral neuropathy, and liver damage. Disulfiram is best given under supervision by someone (such as a spouse), especially during high-risk drinking situations (such as the Christmas holidays).

A 16-week, placebo-controlled trial in patients with relatively severe acute withdrawal reported better outcomes with use of a depressant medication (gabapentin 1200 mg/d), but those results have not yet been replicated. Additional drugs under investigation include another opioid antagonist, nalmefene; the nicotinic receptor agonist varenicline; the serotonin antagonist ondansetron; the α-adrenergic agonist prazosin; the $GABA_B$ receptor agonist baclofen; the anticonvulsant topiramate; and cannabinol receptor antagonists. At present, there are insufficient data to determine the asset-to-