2. *Niacin.* The mechanism of action of niacin is not fully understood, but it appears to inhibit the secretion of lipoproteins containing apo B100 from the liver. Niacin decreases both total and LDL cholesterol approximately 15 to 25 percent, reduces VLDL levels by 25 to 35 percent, and raises HDL cholesterol levels by as much as 15 to 25 percent. Thus, on the basis of its effects on three major lipoproteins, VLDL, LDL, and HDL, niacin would appear to be an optimal agent. Efficacy of monotherapy was confirmed in a long-term secondary prevention trial in which niacin significantly reduced the incidence of myocardial infarction. An even longer-term follow-up of that study (15 years total) showed an 11 percent decrease in all-cause mortality among patients randomized to niacin. Like the bile acid–binding resins, niacin is safe, having been in use for almost 30 years. Niacin, however, has unpleasant side effects that limit patient acceptability, including uncomfortable and potentially dose-limiting cutaneous flushing with or without pruritus. However, the cutaneous symptoms tend to subside after several weeks and may be minimized by initiating therapy at low doses. Less common adverse effects include elevations of liver enzymes, gastrointestinal distress, impaired glucose tolerance, and elevated serum uric acid levels with or without gouty arthritis. Liver enzymes may be elevated in 3 to 5 percent of patients on full doses of niacin (>2 g/d).