Atrial fibrillation is the most common chronic rapid arrhythmia requiring the attention of internists and cardiologists. Patients with this arrhythmia have higher rates of morbidity and death than similar patients with normal sinus rhythm, and they do so for a number of reasons.

Patients with atrial fibrillation have a slew of comorbidities, including hypertensive and ischemic heart disease. Patients undergoing cardiac surgery have a dramatically higher risk of a postoperative bout of atrial fibrillation. The main concerns are the risk of stroke and the symptoms of heart failure and fatigue (often with exercise intolerance).

Information from registries of patients with atrial fibrillation has permitted the development of prognosticators of stroke risk. The CHADS2 score (congestive heart failure, hypertension, age > 75, diabetes, and prior stroke or transient ischemic attack) is an amazingly simple way to identify patients with atrial fibrillation who are at highest risk of stroke. This in turn has allowed stratification of patients for entrance into various anticoagulation studies. And perhaps surprisingly, when many factors are considered, nothing turns out to be dramatically better than warfarin (Coumadin)—if the international normalized ratio (INR) can be appropriately controlled.

Not many options are available to prevent atrial fibrillation. Postoperative atrial fibrillation may be prevented with high-dose steroids or colchicine (Colcrys), but this is often a self-limited, situational event. Chronic or recurrent intermittent atrial fibrillation is not readily prevented in most patients, and many symptomatic patients, as discussed by Dr. Bruce Lindsay in this issue (page 553), may benefit from drug therapy or radiofrequency ablation.

Studies suggest that trying to convert atrial fibrillation to normal sinus rhythm (vs controlling the rate) may not be worth the effort and the risk in many patients with asymptomatic atrial fibrillation. Furthermore, in patients with symptomatic atrial fibrillation, determining the cause of symptoms is difficult. For example, it may not always be easily determined if fatigue in an elderly patient with chronic atrial fibrillation is due to mild rate-related congestive heart failure, decreased left ventricular output due to the loss of the atrial “kick,” chronic ischemia, or the sedating effect of a beta-blocker given in an effort to control the tachycardia.

Despite many large, well-done studies comparing antiarrhythmic drugs, ablation techniques, and anticoagulants, patients will still benefit most from an experienced clinician’s reflective, individualized assessment before embarking on algorithm-driven long-term therapy. We have more choices, more data, and more management algorithms, but there is still no panacea for patients with atrial fibrillation.