INTRODUCTION

The pharmacological approach to atrial fibrillation (AF) involves heart-rate control, anticoagulation, and restoration of normal sinus rhythm. Whereas there is strong evidence demonstrating a reduction in morbidity and mortality using anticoagulation for AF, there are no large-scale prospective studies demonstrating a similar benefit for using antiarrhythmic drugs to maintain sinus rhythm. This chapter reviews the data regarding anticoagulation, and presents an approach to managing clinical scenarios involving rate-slowing drugs and antiarrhythmic therapy.

ANTICOAGULATION IN PATIENTS WITH AF

Firm data now exist for the use of anticoagulation in patients with AF. It is a well-established fact that AF leads to thrombus formation in the heart. Initially, it was believed that thrombus formation in the atria took several days to develop. More recent studies utilizing transesophageal echocardiography have demonstrated that in the absence of anticoagulation, the incidence of left atrial appendage thrombus formation
within 72 h of the onset of AF is 14%. One-half of the thrombi were mobile. Among patients with a recent thromboembolic event, the prevalence of left atrial thrombus did not differ between the group of patients with new-onset AF and those patients with AF of greater than 3 d duration. There was no significant difference between the incidence of left atrial thrombus formation in patients with AF of less than 2 d duration and those in individuals whose arrhythmia was present for 2–3 d.

**Acute Anticoagulation for AF**

Anticoagulation is a two-step process involving acute and chronic treatment. The risk of cardioversion in the absence of anticoagulation increases proportionally with the time an individual is in AF. Although it is generally considered to be safe to cardiovert an unanticoagulated patient who has been in AF for less than 48 h, left atrial thrombus, spontaneous echo contrast, and stroke have been reported to occur within this 48-h window. Weigner et al. (2) retrospectively identified 375 patients with AF lasting less than 48 h. They found the incidence of thromboembolic events after resumption of normal sinus rhythm to be 0.8%. Interestingly, these individuals who had thromboembolic events had normal left ventricular function and no history of AF or prior thromboembolism, and would not have been traditionally characterized as “high risk” for embolism. Although this incidence of clinical thromboembolism is low compared to the 5–7% risk of stroke in unanticoagulated patients who have AF for greater than 48 h who undergo cardioversion, it is nevertheless a significant problem given the overall prevalence of AF in the population. Additionally, there is no difference in atrial mechanical function in patients who cardiovert spontaneously, pharmacologically, or electrically (3). Therefore, it would not be unreasonable to anticoagulate an individual as soon as AF is initially documented if you plan to cardiovert the patient or the patient is at high risk of thromboembolism.

Given the risk of thromboembolism with cardioversion—whether spontaneous, pharmacologic, or electrical—in patients with AF of greater than 48–72 h duration, anticoagulation is needed prior to and after cardioversion. Two approaches are currently acceptable. One method is to place the patient on warfarin and maintain an International Normalized Ratio (INR) of 2 or greater for at least 3–4 wk prior to cardioversion. After cardioversion, the patients need to remain on warfarin for at least 3 wk prior to discontinuing anticoagulation, since there can be a significant lag between the restoration of sinus rhythm and resumption of normal atrial mechanical function. This can lead to thromboembolism after cardioversion if the patient is not adequately anticoagulated. The importance of maintaining strict anticoagulation has been demonstrated in a study performed at the Ochsner Medical Institutions (4). One hundred and fifty patients with AF underwent elective electrical cardioversion. All patients were anticoagulated for at least 3 wk with weekly blood tests to ensure an INR of 2 or greater. If the patient’s INR dipped below 2, the “clock” was restarted and the patient would have to demonstrate three more consecutive weeks of therapeutic INRs. Ninety-five percent of the patients were successfully cardioverted, and there were no thromboembolic events. Therefore, by maintaining a very strict level of precardioversion anticoagulation (INR greater than or equal to two or three consecutive weekly measurements) restoration of sinus rhythm can be achieved with minimal morbidity. In clinical practice, this can translate into several weeks or months for an individual patient to achieve this goal. This scenario can potentially result in a decreased effectiveness of cardioversion and antiarrhythmic