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CHAPTER

ATRIAL FIBRILLATION

Daniel M. Witt

INTRODUCTION

Atrial fibrillation (AF) is a common cardiac rhythm disorder. Although AF rarely causes life-threatening hemodynamic compromise, it is an important independent risk factor for cardiogenic embolic stroke and systemic arterial thromboembolism.[1] Approximately 90% of AF thromboembolic complications are stroke related, while the remaining 10% are systemic embolism (see **Table 14-1** for more information on classifications). The following contribute to thromboembolic risk associated with AF:

- Stasis or turbulence of blood flow within the left atrial appendage leads to thrombus formation.
- Dysfunction of vascular endothelium predisposes to local or systemic hypercoagulability.
- Conversion to normal sinus rhythm (NSR)—spontaneous or intentional—may dislodge any existing left atrial thrombi.

MORBIDITY AND MORTALITY ASSOCIATED WITH AF[1,2]

- Nonvalvular AF is associated with a 5-fold increased risk of stroke.
- The annual risk for nonfatal stroke in untreated AF patients varies between 0.8% and 9.6% (average ~5%) depending on concurrent individual risk factors.
- Attributable stroke risk in AF increases with age:[3]
    - 1.5% for patients 50–59 years
    - 23% for patients 80–89 years
- AF-related strokes tend to be more severe than non-AF-related strokes.

Data from high-quality, randomized controlled clinical trials overwhelmingly demonstrates that long-term, adjusted-dose anticoagulation therapy with vitamin K-antagonists (e.g., warfarin) or direct-acting oral anticoagulants (DOACs) (e.g., dabigatran, rivaroxaban, apixaban, edoxaban) virtually eliminates the stroke risk associated with AF.[1] Despite the proven efficacy of anticoagulation therapy in preventing AF-related stroke, only about half of patients who could benefit receive anticoagulation therapy.[3] Increasing age, perceived bleeding risk, and the innate complexity of managing anticoagulation therapy are some reasons underlying why clinicians and patients with AF opt against anticoagulation therapy.

TABLE 14-1: **Classification of Atrial Fibrillation**[1]

Paroxysmal AF	Terminates spontaneously or with intervention within 7 days (may recur)
Persistent AF	Continuously sustained for >7 days
Long-standing persistent AF	Continuously sustained for >12 months
Permanent AF	When patient and clinician jointly decide to stop further attempts to restore/maintain sinus rhythm (a therapeutic attitude not related to inherent pathophysiologic attributes of AF)[a]
Nonvalvular AF	AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair

[a]Attitude may change as symptoms, efficacy of therapeutic interventions, and patient–clinician preferences evolve over time.

AF: atrial fibrillation

TREATMENT OVERVIEW

Rate versus Rhythm Control

- Evidence from randomized clinical trials indicates that cardioversion of AF to normal sinus rhythm (rhythm control) is not necessary nor preferable to allowing AF to continue while controlling ventricular response rate with AV node blockade (rate control) with concurrent anticoagulation in those with high enough risk to justify therapy.[4,5]
    - The AFFIRM trial found no difference in mortality or stroke rate between patients assigned to one strategy or the other.[4]
    - The RACE trial (patients had persistent AF post failed cardioversion) found rate control not inferior to rhythm control for prevention of death and morbidity.[5]
    - Rate- or rhythm-control strategies do not seem to affect quality of life significantly or differently.
    - Ischemic events occurred with similar frequency with either a rhythm or rate control strategy, especially when warfarin was discontinued or when anticoagulation was subtherapeutic.
    - In younger individuals, a combined rate and rhythm approach may minimize the risk of related heart failure.
    - Whether a rate or rhythm control strategy is employed, AF patients *with thromboembolic risk factors* should probably receive chronic anticoagulation.[1]