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Short Stay Management of Atrial Fibrillation

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Short Stay Management of Atrial Fibrillation

Humana Press

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Preface

Atrial fibrillation, already the most common cardiac arrhythmia, continues to increase in the frequency of its hospital presentations. Historically, the evaluation and management of atrial fibrillation required several days to complete, thus necessitating an inpatient admission. That is not the case anymore. Changes in the sensitivity and time to results of troponin, ready availability of cardiac ultrasound, and the rapid onset of effect of the direct oral anticoagulant drug class have provided a new paradigm. Inpatient hospitalization is no longer the only clinical pathway choice for managing this arrhythmia. Rate control, evaluation of new-onset atrial fibrillation, and cardioversion all represent potential patient candidates for admission to the short stay unit. Consequently, new care pathways, procedure standards, and patient discharge information are needed. This book outlines the strategies for care and disposition and provides order sheets and process criteria via which institutions can successfully manage the atrial fibrillation patient in the short stay unit, thus optimizing patient outcomes, patient satisfaction, and institutional operational efficiencies. We hope you find this book useful in the management of the atrial fibrillation patient.

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Part I
Impact of the Disease

Chapter 1
Atrial Fibrillation: Epidemiology and Demographics

Karina M. Soto-Ruiz

First identified on electrocardiograms over 100 years ago [1, 2], atrial fibrillation (AF) is the most commonly presented arrhythmia of clinical significance [3, 4]. It is estimated that it affects 46.1 million people globally [5]. Up to a half-million hospitalizations annually in the USA have AF as their primary diagnosis, and AF is estimated to contribute to >100,000 deaths per year in the USA [6]. And hospitalizations have increased by 23 % between 2000 and 2010 [7].

AF has a significant impact on health-care costs, with the major cost drivers being hospitalizations, stroke, and loss of productivity [8–11]. In 2001, it was estimated that $6.65 billion US dollars was spent on AF treatment [12], with more recent findings estimating the costs to have ballooned from $16 to $26 billion US dollars a year [13], and according to findings by the Cost of Care in Atrial Fibrillation survey, hospitalizations represented a large portion of the cost of treating AF: 52 % vs. 23 % for drug therapy [14].

In the USA, the prevalence of AF increased by 4.5 % (from 4.1 to 8.6 %) between 1993 and 2007, with a 0.3 % increase per year, in Medicare beneficiaries older than 65 years of age [15–20]. Over 2.3 million adults in the USA had a diagnosis of AF in the years 1996 and 1997, with estimations that the number would go up to 5.6 million by 2050 [3]; this numbers climbed to 3.03 million in 2005 in the USA alone, and the prediction now is that 7.56 million will have AF by 2050 [21]. Worldwide, a total of 33.5 million patients had a diagnosis of AF in the year 2010, and it's estimated that there will be 5.5 million new cases diagnosed each year [5]; with such a dynamic prevalence, the impact and burden in our health-care system cannot be overlooked. Specially, when the real prevalence of AF could be underrepresented due to the fact that in up to 25 % of cases, AF occurs in the absence of symptoms, potentially underestimating the real prevalence of the disease [22, 23]. Monitoring techniques to detect asymptomatic, or subclinical AF, also have an impact on

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prevalence. Using a 24-h Holter monitor or an event-loop recording device for 7 days, AF was detected in 11 % of the patients sampled [24]. When using a dual-chamber pacemaker or implantable cardioverter defibrillator for 3 months, subclinical AF was detected in 10 % of patients, and at 2.5 years, subclinical AF was detected in 35 % of patients, of whom 16 % had developed clinical AF [25].

AF is associated with significant morbidity, including a 3–5 times increase in the risk of stroke, and a three times increase in the risk of congestive heart failure (CHF) [26, 27], and it's also associated with a twofold increase in comorbidity-adjusted mortality [9]. AF has also been described as a risk factor for dementia, even in patients without a history of stroke. Studies have reported that decreased cognitive scores (OR 1.5–3.5) are associated with the presence of AF in different populations [30–34]. In the ONTARGET and TRANSCEND studies [35], patients with AF showed faster worsening of Modified Mini-Mental State Examination and Digit Symbol Substitution Test scores over the subsequent 5 years than did patients without AF [36]. It has also been described that individuals with AF had an odds ratio of 2.8 for dementia compared with individuals without AF [31]. The increased risk of dementia in patients with AF is also associated with increased mortality (HR 2.9) [37].

The increase in prevalence has been attributed to a greater ability to treat chronic cardiac and noncardiac disease, the aging population, and the improved ability to suspect and diagnose AF [22]; therefore, when talking about prevalence, we must keep in mind it depends on the population we study. AF is not common in infants and children; if present, it will almost always be associated with a structural heart defect: congenital heart disease, cardiomyopathy, or Wolff-Parkinson-White (WPW) syndrome [28].

As the population age increases, so does the prevalence of AF. It is present in 0.12–0.16 % of subjects younger than 49 years of age, 3.7–4.2 % in those aged 60–70 years, and 10–17 % in patients 80 years or older [5, 15–20, 29]. In subjects over 50 years of age, AF was more frequent in Whites than Blacks (2.2 % vs. 1.5 %). Gender also had an impact on prevalence, with AF occurring more frequently in males (1.1 % vs. 0.8 %) than females [3, 4], but despite a greater prevalence in men, women represent the bulk of patients with AF due to their longer survival [5, 15–20, 29].

The impact of race is less clear. Multiple studies have found the risk of developing AF to be lower in Blacks compared to Whites, but it has not been determined whether Blacks are at lower risk or if Whites are at higher risk [38]. Data on Native American patients is scarce, but in a study of 664,754 subjects, of which 27,697 were Native Americans, an analysis looking at AF trends was conducted in a subpopulation of 67 % White and 55 % Native Americans and found that AF was less prevalent in Native Americans than in White adult males after adjusting for age, BMI, and predisposing comorbidities (adjusted odds ratio 1.15; CL 1.04–1.27) [39].

The lifetime risk for developing AF was analyzed in a report from the Framingham Heart Study. They found the risk for developing AF from age 40 to 95 was 26 % for men and 23 % for women. The risk of developing AF from age 80 to 95 was 23 % for men and 22 % for women. Lifetime risk didn't change significantly with increased age due to the fact that AF incidence increases with age [40].

The exact reason for the current trends in prevalence are unknown, but could be partially explained by aging trends in the global population. Temporal trends in prevalence may result from a lead time bias, increased survival from coexistent cardiovascular conditions such as ischemic heart disease and heart failure due to improved management of cardiovascular comorbidities, resulting in a larger high-risk group [41].

Risk Factors

Cardiovascular Disease AF has been associated with cardiovascular disease, in particular with hypertension, coronary artery disease, cardiomyopathy, and valvular disease; it can also occur after cardiac surgery and in the presence of myocarditis or pericarditis [42]. Hypertension can increase the risk of developing AF by 1.42-fold [43], which represents a relatively small increase in risk; however, given the high frequency of hypertension in the general population, hypertensive heart disease is the most common underlying disorder in patients with AF [44].

Coronary Heart Disease AF is not commonly associated with coronary heart disease unless it's complicated by an acute myocardial infarction (MI) or heart failure (HF). AF can occur in 6–10 % of patients with an acute MI, presumably due to atrial ischemia or atrial stretching secondary to HF [41–48]. In patients with chronic stable coronary heart disease, AF can be found in less than 1 % of patients, with a relative risk of 1.98 at 7 years [49].

Valvular Lesions Lesions that lead to significant stenosis or regurgitation are associated with the development of AF. The rate of development of AF can be up to 5 % per year in patients with valvular disease, and the major independent risk factors were age (65 years or older) and baseline left atrial dimension >50 mm [50]. When AF complicates severe mitral regurgitation, valve repair or replacement is indicated [42].

Rheumatic Heart Disease (RHD) RHD and its associated mitral valve disease were a major cause of AF in the Western world in the past, but the availability of early treatments has made the disease rare in developed countries. However, several studies from Africa, Asia, and the Middle East report a substantial prevalence of RHD in their population with AF [51–55].

Cardiomyopathy AF has been reported in 10–28 % of patients with hypertrophic cardiomyopathy [49, 56, 57], although the prognostic importance remains unclear, with some literature showing a negative impact on prognosis [57], while others show no increase in mortality [56].

Venous Thromboembolic Disease Deep vein thrombosis and pulmonary embolism are associated with an increased risk of AF. The exact mechanism for this is not

known, but it has been speculated to be related to an increase in pulmonary vascular resistance and cardiac afterload, which may lead to atrial strain [58, 59]. It has been reported that up to 14 % of patients with a documented pulmonary embolism develop AF [60, 61].

AF can also occur in chronic obstructive pulmonary disease [61–63], peripartum cardiomyopathy [64], lupus myocarditis [65], and both idiopathic and uremic pericarditis [66, 67]. Obstructive sleep apnea may also be related, in which case the provision of continuous positive airway pressure reduces the risk of the recurrence of AF [68].

Obesity This has also been described as a risk factor. Increased left atrial pressure and volume as well as a shortened effective refractory period in the left atrium and proximal and distal pulmonary veins have been identified as potential factors that facilitate and perpetuate AF in this population [69]. The risk of AF among individuals who are obese was >1.6 times that of counterparts with a normal BMI [70]. In the Atherosclerosis Risk in Communities Study (1989) from the USA, the population-attributable fraction of the risk of AF contributed by being overweight or obese was estimated at 17.9 %, making it the second most important risk factor after hypertension [71].

Diabetes This has also been associated with an increased risk of developing AF (OR 1.1 for men and 1.5 for women); increased left ventricular mass and increased arterial stiffness have been put forth as possible mechanisms [72, 73].

Secondhand Smoke This is an emerging risk factor linked to the development of AF in both current and former smokers [74, 75]. Current hypotheses regarding the mechanism by which secondhand smoke leads to cardiac disease include induction of an inflammatory state [76] and direct effects of nicotine on atrial structural remodeling [77, 78] and effects on autonomic function [79, 80]. Each of these mechanisms has been implicated in the pathogenesis of AF [70, 81]. Dixit et al. looked into a subpopulation if 4976 subjects enrolled in the Health eHeart Study and found that patients with AF were more likely to have been exposed to SHS in utero, as a child, as an adult, at home, and at work. However, those without AF were more likely to have visited social environments with significant SHS. When in utero exposure was separated by parent, maternal or paternal smoking during that period was associated with AF: 50 % of those with AF exposed to maternal smoke compared to 32 % of those without AF, $p < 0.001$, and 66 % of those with AF exposed to paternal smoke compared to 48 % of those without AF, $p < 0.001$ [82].

Renal Disease This has also been linked as a risk factor for the development of AF. Compared to individuals with eGFRcys >90 mL/min/m^2, a multivariable hazard ratios for the development of AF were significantly increased at 1.3, 1.6, and 3.2 in those with eGFRcys of 60–89, 30–59, and 15–29 mL/min/m^2, respectively, during a median follow-up of 10.1 years [83].

In the Framingham study, 1409 patients with new-onset AF were evaluated for the risk of subsequent occurrence based on whether they develop a secondary precipitant or not. A precipitant was found in 31 % of patients. They were cardiothoracic surgery (30 %), infection (23 %), noncardiothoracic surgery (20 %), and acute myocardial infarction (18 %). Other precipitants found were acute alcohol consumption, thyrotoxicosis, acute pericardial disease, acute pulmonary embolism, and other acute pulmonary pathologies. While the 15-year cumulative incidence of recurrent AF was lower among those with secondary causes (62 % vs. 71 %), finding a recurrence of AF among the population with secondary causes was still unexpected [84].

Postoperative Period AF has been reported in up to 30–40 % of patients in the postoperative period for those who underwent coronary artery bypass grafting (CABG) surgery [85–88], in 37–50 % after valve surgery [85, 88, 89], and in up to 60 % of those undergoing a valve replacement plus CABG [65, 68, 85, 88]. AF has also been described in up to 24 % of patients with a denervated transplanted heart and often in the absence of significant rejection [88, 89]. Most occur within the first 2 weeks, while developing AF after the 2-week mark has been associated with an increased risk of subsequent death [89, 90]. AF is less common after noncardiac surgeries with incidence rates that vary, from 1 to 40 %. This wide range could likely be due to the variability in patients and surgical characteristics [91, 92].

Hyperthyroidism Patients with hyperthyroidism are at an increased risk of developing AF [93]. This is believed to occur due to an increased beta-adrenergic tone that may contribute to the rapid ventricular response [94]. It has also been suggested that it may be related to an increased automaticity and enhanced triggered activity of pulmonary vein cardiomyocytes, which can be a source of ectopic beats that initiate AF [95]. AF can be seen in up to 20 % of patients over the age of 60 but less than 1 % of patients under 40; men are also more likely to have AF than women (12.1 % vs. 7.6 %) [96].

Other factors associated with an increased risk have also been described:

Family History The presence of AF in a first-degree relative, particularly a parent, has been associated with an increase in risk, independent of standard risk factors such as age, sex, hypertension, diabetes, or clinically overt heart disease [97]. The strength of this connection seems to be greater with first-degree relatives with premature onset (less than 65 years of age) [98].

Polygenic Inheritance This seems to be more common and could explain the modest elevation in the relative risk of AF in first- and second-degree relatives of affected individuals [97, 99].

Monogenic Inheritance Both autosomal dominant and autosomal recessive forms have been identified. Genetic linkage analysis has identified loci at 10q22-q24, 11p15.5, 6q14-16, 3p22-p25, and 4q25 [100–104]. At the 4q25 locus, several single-nucleotide polymorphisms have been identified [105].

Birth Weight A possible relationship between birth weight and AF development has been described. The age-adjusted hazard ratios (with <2.5 kg [5.5 lb] being the referent group) for incident AF increased significantly from the lowest to the highest birth weight category, during a median follow-up of 14.5 years [106].

Inflammation and Infection A relationship has been described after studies looking at levels of C-reactive protein (CRP) and finding elevated levels in people with later development of AF [107], history of atrial arrhythmias [108], failed cardioversion [109], recurrence of AF after cardioversion [110], and development of AF after cardiac surgery. However, inflammation is more likely a marker of conditions associated with AD, as opposed to being a direct or perpetuating cause [111].

Pericardial Fat Pericardial fat is a visceral adipose tissue with inflammatory properties, and patients with AF had a significantly higher pericardial fat volume (102 mL vs. 76 mL in controls with no AF) [112]. Using data from over 3000 individuals in the Framingham Offspring Cohort, a 40% higher odds ratio of AF per standard deviation increase in pericardial far volume was observed. This association remained significant after adjustments for age, gender, heart disease (myocardial infarction, heart failure), BMI, and other regional fat deposits [113].

Autonomic Dysfunction The autonomic nervous system may be involved in the initiation and maintenance of AF. It may be particularly important in patients with paroxysmal AF, as both heightened vagal and sympathetic tone can promote AF. Heightened vagal tone in predominantly normal hearts, which may explain why vagally mediated AF is often seen in athletic young men without apparent heart disease who have slow heart rates during rest or sleep; such patients may also have an electrocardiogram (ECG) pattern of typical atrial flutter alternating with AF [114, 115]. In comparison, AF induced by increased sympathetic tone may be observed in patients with underlying heart disease or during exercise or other activity [115].

Corrected QT Interval Individuals with either congenital long QT syndrome or short QT syndrome have an increased risk of AF [116, 117]. Individuals with a QTc <372 ms (1st percentile) or >419 ms (60th percentile) had an increased risk (adjusted hazard ratios 1.45 up to 1.44, respectively) compared to the reference group (411–419 ms) [118].

Premature Atrial Contractions Premature atrial contractions (PAC) are known triggers of PAC. PAC count (by quartile) on Holter monitoring was associated with incident AF [119].

Other Supraventricular Tachyarrhythmias Spontaneous transition between typical atrial flutter and AF has been observed, although little is known about the mechanism of this [120, 121]. AF is, in some patients, associated with paroxysmal supraventricular tachycardia (PSVT) [122–124]. The most common causes of PSVTs are atrioventricular nodal reentrant tachycardia and atrioventricular reentrant tachycardia, which occurs in patients with WPW syndrome or concealed

accessory pathways. Among patients with WPW syndrome, the mechanism may be retrograde conduction via the accessory pathway of a premature beat, stimulating the atrial myocardium during its vulnerable period [125]. Ablation of the accessory pathway reduces the incidence of subsequent AF [125, 126].

Low Serum Magnesium Low serum magnesium in patients undergoing cardiac surgery has been identified as a risk factor for the development of postoperative AF. Patients in the lower quartile of serum magnesium (≤1.77 mg/dL) were approximately 50 % more likely to develop AF compared to patients in the upper quartiles (≥1.99 mg/dL) after multivariable adjustments [127].

Alcohol AF occurs in up to 60 % of binge drinkers with or without an underlying alcoholic cardiomyopathy [128]. Most cases occur during and following weekends or holidays when alcohol intake increases, a phenomenon which has been termed "the holiday heart syndrome." Moderate, long-term alcohol consumption does not appear to be a risk factor for AF and has no significant association in either men or women [72, 129, 130]. In contrast, heavy alcohol consumption is associated with an increased incidence of AF. Two large cohort studies found an increased incidence among men with heavy alcohol consumption (hazard ratio 1.45 in both) [131, 132]. Neither study found a correlation in female patients, but the ability to detect such a correlation was limited by the small sample size of women with alcohol consumption in this range. Another study found an increased risk (relative risk 1.34, 95 % CI) with consumption of more than 36 g per day (>3 drinks/day) [130].

Fish and Fish Oil Supplements It has been suggested the intake of fish and fish oils rich in long-chain n-3 fatty acids may reduce the incidence of arrhythmias [133], but the data is mixed. Three cohort studies, with sample sizes of 45,000, 48,000, and 5000 patients, found no relationship [134–136], while one study with a cohort of approximately 5000 patients suggested a reduction in AF burden [136].

Medications Certain medications can cause or contribute to AF development [137]. These include theophylline [138], adenosine [114], and drugs that enhance vagal tone, such as digitalis [139]. Case-control studies have suggested an increased risk for developing AF in patients taking nonsteroidal anti-inflammatory drugs (NSAIDs) [140–142]. However, the absence of an accepted biologic mechanism and the susceptibility of case-control studies to unmeasured confounders make us cautious about the strength of this association [143].

Classification and Progression

AF has been classified as paroxysmal, persistent, permanent, or lone [144]. Lone atrial fibrillation refers to the presence of AF with no underlying structural heart disease; it can be present in as much as 45 % of patients with paroxysmal AF [145].

In patients recently diagnosed, it's been found that up to 15 % of patients progress to persistent or permanent AF within 1 year. Heart failure, hypertension, and rate-control therapy, rather than rhythm-control (26 % vs. 11 %), were independently associated with AF progression. A risk stratification rule to assess the probability of AF progression found that age, previous TIA or stroke, and COPD were also associated with AF progression. The clinical outcome of patients demonstrating AF progression is worse compared with patients demonstrating no AF progression [146]. Up to 52 % of newly diagnosed had paroxysmal AF [147]. Patients with a higher CHADs2 score showed more AF progression: 19 % progression when CHADs2 > 1 vs. 14 % when CHADs2 is 1 and 9 % when CHADs2 is 0 ($p < 0.0001$). The use of class 1c antiarrhythmic drugs was also associated with less AF progression, whereas the use of cardiac glycosides was associated with more AF progression at 1 year follow-up [146].

The World Health Organization (WHO) quantifies the burden of disease from mortality and morbidity utilizing disability-adjusted life year (DALY) measurements; one DALY can be thought of a one lost year of "healthy" life. The sum of these DALYs across a population, or the burden of disease, is considered a measurement of the gap between current health status and an ideal health situation where the entire population lives to be of advanced age, free of disease and disability [147].

In the 2010 Global Burden of Disease Study, DALYs were utilized to indicate the overall morbidity contributed by a given disease in the population. For AF, the age-standardized DALYs in Central Asia, China, Russia, South Asia, Southeast Asia, and sub-Saharan Africa were 35–50 per 100,000 people. In comparison, age-standardized DALYs for patients with AF in Australia, Canada, the USA, and Western Europe were >60 per 100,000 people [148, 149]. A comparison of these values with data from the 1990 Global Burden of Disease Study shows that the burden of AF is steadily rising and now comprises an increased percentage of total DALYs for each nation [148].

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Chapter 2
Pathophysiology of Atrial Fibrillation and Clinical Correlations

Ezra Amsterdam, Sandhya Venugopal, and Uma N. Srivatsa

Background

Atrial fibrillation (AF) is the most common sustained cardiac arrhythmia. It increases with age, affecting less than 1 % of those under age 60 and over 10 % of individuals greater than 75 years old [1]. AF occurs primarily in patients with structural heart disease, but a small proportion of patients with this arrhythmia have no evidence of cardiac disease. AF in these cases was previously known as "lone atrial fibrillation," a term no longer used because of its imprecise definitions. Multiple conditions and risk factors associated with AF include age, heart failure, valvular heart disease, hypertension, diabetes, ischemic heart disease, chronic obstructive pulmonary disease, and anemia. AF affects over three million patients in the USA; increases the risk of heart failure, stroke, and hospitalizations; and is associated with an annual economic burden estimated at over $25 billion [2, 3]. To meet the human and economic challenges of this arrhythmia, an increasingly wide array of innovative therapies has been developed.

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Pathophysiology and Clinical Implications

AF is a supraventricular arrhythmia consisting of rapid, disorganized atrial electrical activity (Fig. 2.1) that results in chaotic and ineffective atrial contractile function [4, 5]. The rate of impulse generation is greater than 400/min in the atria and the ventricular response is irregular; it may be rapid or slow depending on atrioventricular (AV) node function. The classical electrocardiogram (ECG) features of AF are the absence of P waves and an irregularly irregular pattern of QRS complexes. The ECG baseline may show fibrillatory activity (fine or coarse) or no excursion (flat line) between the QRS complexes (Fig. 2.2). Other atrial arrhythmias such as atrial flutter and atrial tachycardias are commonly associated with AF. The most frequent of the numerous symptoms caused by AF is fatigue; palpitations, light-headedness, syncope, chest pain, and dyspnea are also common [6].

The initial phase in the generation of sinus rhythm requires exquisitely coordinated interaction between anatomically and physiologically intact sinoatrial node, conduction pathways, and the myocardium. Abnormalities in these structures or physiologic derangements (e.g., electrolyte alterations, hypoxemia, ischemia) can disrupt the stability required for their proper function and result in structural and electrical remodeling. AF can cause or result from cardiac disease. The trigger for

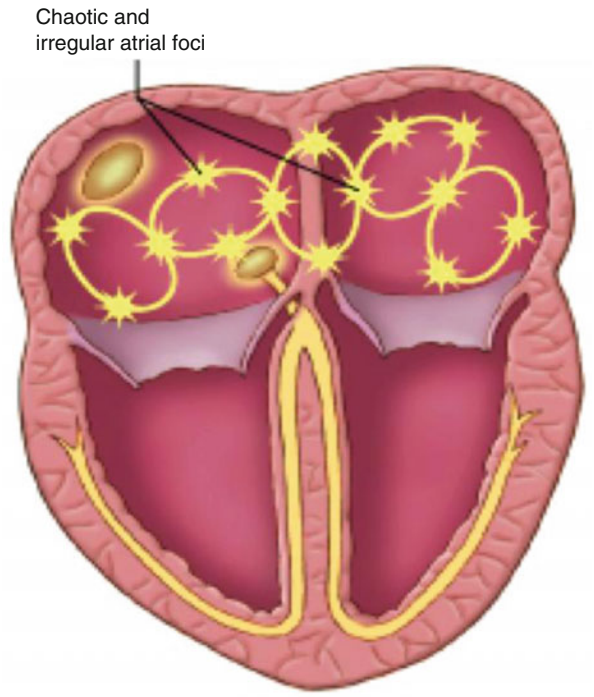

Fig. 2.1 The figure is a representation of the innumerable foci of chaotic electrical activity in the atria during atrial fibrillation

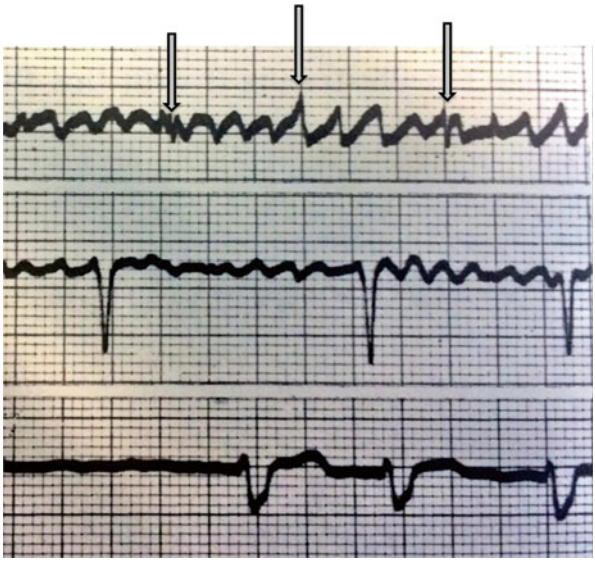

Fig. 2.2 *Top*: Coarse atrial fibrillation. The QRS complexes (*arrows*) are almost completely masked in this example. *Middle*: Fine atrial fibrillation. *Bottom*: Flat-line atrial fibrillation. There are barely visible undulations in the baseline

AF is often a premature atrial complex arising from pulmonary veins in over 80 % of cases; other sites of origin are the posterior wall of left atrium, superior vena cava, vein of Marshall, and rarely the coronary sinus. Atrial remodeling is the structural substrate of AF and comprises enlargement, stretch, and fibrosis that impair normal atrial electrophysiology and promote disorganized rhythm leading to AF [7, 8]. This remodeling usually results from factors involving the left ventricle (e.g., age, hypertension, mitral or aortic valve disease, systolic or diastolic dysfunction) whose impairment may impart retrograde elevated pressure and volume that adversely affect atrial structure and function. Electrical remodeling is a product of structural abnormalities and physiologic mechanisms including inflammation, altered activity of the sympathetic and/or parasympathetic nervous systems, stimulation by the renin-angiotensin-aldosterone system, and oxidative stress [9–11].

The adverse hemodynamic effects of AF are attributable to multiple factors (Table 2.1 [12]). These variables can precipitate heart failure in asymptomatic patients with left ventricular dysfunction and worsen symptoms in those with preexisting heart failure. The degree to which hemodynamic function is impaired by these factors is related to their intensity, duration, and the underlying cardiac substrate. Episodes of AF may be brief, protracted, chronic, or repetitive. The current classification of AF is based on the duration of these episodes (Table 2.2 [13]). AF is an important cause of stroke and other peripheral embolic diseases. Impaired atrial contraction results in relative stasis of the blood, which promotes atrial thrombogenesis particularly in the left atrial appendage, thereby providing the source of systemic embolization.

Table 2.1 Factors leading to adverse hemodynamic function in atrial fibrillation

Abnormal heart rate (fast or slow)
Loss of atrial contribution to ventricular filling
Variable beat-to-beat ventricular function and variable diastolic filling due to the irregular rate
Potential for tachycardia cardiomyopathy
Activation of vasoconstrictor neurohormones

Adapted from Podrid [11]

Table 2.2 Classification of atrial fibrillation

Paroxysmal	Terminates spontaneously or with treatment within 7 days
Persistent	Continuous AF that is sustained for >7 days
Long-standing persistent	Continuous for >12 months
Permanent	Applies to joint decision by patient and clinician to cease further attempts to restore and/or maintain sinus rhythm
Nonvalvular	Absence of rheumatic mitral stenosis, mechanical or bioprosthetic heart valve, or mitral valve repair

Adapted from January et al. [12]

Management of Atrial Fibrillation in Heart Failure

AF promotes the development, recurrence, and persistence of heart failure through the factors noted in Table 2.1. The management of AF in patients with or without heart failure entails three major approaches: *rate control*, *rhythm management*, and *anticoagulation*. These strategies are based on favorable modification of the pathophysiology of the arrhythmia.

Rate Control Adequate control of ventricular rate is crucial in patients presenting with heart failure and untreated or ineffectively treated AF. This concept applies to any of the classes of AF in Table 2.2 to favorably impact quality of life, reduce morbidity, and decrease the potential for tachycardia-induced cardiomyopathy. For example, the development of paroxysmal AF at a ventricular rate of 150/min or greater may precipitate acute cardiac failure in a stable patient in normal sinus rhythm with underlying cardiac disease [14]. Acute decompensation can occur in patients with normal as well as reduced cardiac systolic function. Rate control not only has a direct, beneficial effect on cardiac function but also may alleviate other factors associated with tachycardia such as excess activity of the sympathetic nervous system and additional neurohormonal and inflammatory factors [13].

Rhythm Management Restoration of normal sinus rhythm is a central consideration in patients with AF. The advantages of sinus rhythm include more optimal cardiac rate with its benefits on ventricular function, elimination or marked reduction of cardioembolic risk, cessation of AF-associated symptoms such as palpitations, and improvement in quality of life [15]. Importantly, attainment of sinus

rhythm is the most rapid and efficient approach to rate control. Long-term maintenance of sinus rhythm often utilizes antiarrhythmic drug therapy or catheter ablation of the AF foci. The latter are usually located at the orifices of the pulmonary veins or other intra-atrial sites.

Anticoagulation The benefits of anticoagulation in patients with AF are well established. Risk for cardiac thrombus formation and embolization with its major hazard of stroke are consistently reduced. However, this therapy also confers increased risk of bleeding. Risk scores have been developed to identify probability of stroke in patients with AF and are helpful in patient selection for anticoagulation. The two most commonly utilized are $CHADS_2$ and $CHADS_2VASC_2$ [15]. In this regard, there are also scores for the risk of bleeding from anticoagulation [16, 17]. These evaluation tools aid clinicians in balancing reduction of stroke by anticoagulation and the risk of bleeding from this therapy. Importantly, the risk of bleeding increases virtually in parallel with the risk of stroke [16, 17].

Paroxysmal AF and Acute Decompensated Heart Failure

Paroxysmal AF is an important cause of acute decompensated heart failure (ADHF) [18] through the mechanisms previously described. ADHF may be provoked by paroxysmal AF induced by cardiac or noncardiac factors such as atrial dilation due to chronic progressive heart failure, electrolyte abnormalities, hypoxemia, pulmonary embolism, hyperthyroidism, infections, postoperative state, other systemic diseases, or substance abuse (e.g., cocaine, ethanol, methamphetamines). Each of these factors can disrupt atrial impulse formation at the anatomic or physiologic level resulting in the disorganized electrical activity of AF. It is important to note that the development of AF in a patient with acute myocardial ischemia or infarction is typically related to ventricular failure and consequent atrial stretch rather than atrial infarction and may reflect an extensive infarct and major left ventricular dysfunction.

After the diagnosis of AF is made, a therapeutic plan is established with *control of ventricular rate* as a primary, immediate objective. This can be accomplished directly by pharmacologic blockade of the AV node with selection from among several classes of drugs including digoxin, beta-adrenergic blockers, calcium blockers, and amiodarone, all of which can reduce conductivity and increase refractory period of the AV node. These effects reduce the number of atrial impulses that traverse the node and reach the ventricles. In ADHF, rapid and effective therapy is warranted, and an advantage of these drugs is that each can be administered intravenously, affording relatively prompt reduction of ventricular rate.

The following comments pertain to intravenous use of digoxin, beta-blockers, calcium channel blockers, and amiodarone. Although least effective in achieving rate control, digoxin is the only one of these agents without vasodilating and negative inotropic actions, properties that can worsen cardiac failure and decrease blood

pressure, especially in patients with cardiac decompensation. Beta-blockers are not indicated in acute cardiac failure with sinus rhythm, but in the setting of heart failure and rapid ventricular response, judicious administration can reduce ventricular rate while avoiding untoward effects. Calcium blockers are contraindicated if left ventricular ejection fraction is less than 40 % because of their negative inotropic and vasodilating actions. However, combining digoxin with a calcium blocker such as diltiazem (or a beta-blocker) can frequently achieve the desired effect on ventricular rate without producing hypotension or worsening ventricular dysfunction. Amiodarone, a potent inhibitor of AV node impulse transmission, has negative inotropic effects and vasodilator actions and is usually considered when the previously noted approaches are ineffective or contraindicated. In small doses, amiodarone can be safe and very effective in reducing rapid ventricular rate in acute heart failure with AF. It is important to note that correction of hypoxemia and volume overload with intravenous diuretic therapy in patients with ADHF and AF can have major salutary effects on uncontrolled ventricular rate. Improved oxygenation and alleviation of pulmonary congestion decrease excessive adrenergic drive and are important mechanisms that lower ventricular rate. In addition, correction of excess intravascular volume reduces atrial stretch that may enhance early conversion of AF to sinus rhythm.

In patients with hemodynamic instability or acute myocardial ischemia, or in whom ventricular rate is unresponsive to AV nodal blocking agents, *rhythm management* by urgent external electrical cardioversion is indicated. This method applies a direct current electrical shock to the patient's chest, which is transmitted to the heart. It restores sinus rhythm by depolarization of all excitable myocardium, thereby inducing electrical homogeneity and permitting sinus rhythm to reemerge. This method has a high success rate in stable patients with recent AF but is less successful in unstable patients; it is safe in both settings in the absence of intracardiac thrombi. The advantages of sinus rhythm have been described above.

Anticoagulation is indicated in patients with AF and acute heart failure to reduce the risk of cardioembolic events. It is initiated with heparin and an oral vitamin K antagonist (warfarin). Heparin is discontinued when the international normalized ratio is therapeutic or near therapeutic (usually within 3–5 days). The new oral anticoagulants have provided an alternative to warfarin for long-term anticoagulation. Finally, the basic evaluation of patients with acute heart failure and AF should search for all potential precipitating factors of the arrhythmia, especially correctable ones, such as hyperthyroidism, which, through its association with augmented sympathetic nervous system stimulation, can disorganize atrial electrical function and induce AF.

Persistent or Permanent AF and Heart Failure

Although AF in this patient population is chronic, the principles of management are similar to those in the setting of acute AF and heart failure: *rate control*, *rhythm management*, and *anticoagulation*. An essential aspect of *rate control* is optimal

treatment of heart failure, which in itself will aid in alleviating uncontrolled rate. However, since adequate rate control is a key element of heart failure treatment for those with AF, therapy of both syndromes is complementary and requires coordination. The use of a beta-blocker with documented survival benefits in heart failure (carvedilol, long-acting metoprolol, or bisoprolol) is indicated, and its efficacy on ventricular rate may be enhanced with addition of digoxin. Amiodarone can be employed for rate control in situations in which the previous therapy is inadequate. Rate-limiting calcium channel blockers are contraindicated in patients with left ventricular ejection fraction less than 40 %. In patients refractory to or unable to tolerate medications, ablation of the AV node is an option to accomplish rate control.

Several approaches are available for *rhythm management* to establish sinus rhythm. These include elective pharmacologic cardioversion, electrical cardioversion, and catheter ablation, all of which are covered in detail elsewhere in this volume. Drugs used for this purpose include propafenone, amiodarone, and dofetilide, of which only amiodarone is safe in patients with depressed left ventricular ejection fraction because of its low cardiac side effect profile. In addition, antiarrhythmic drugs are frequently required to maintain sinus rhythm after attainment by any of the foregoing methods. In stable patients with long-standing AF, the success rate of electrical cardioversion in restoring sinus rhythm is inversely related to factors such as severity of hemodynamic dysfunction, duration of AF, and size of the left atrium. Ablation therapy for AF in patients with heart failure is receiving increasingly wide application based on encouraging results.

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Chapter 3
The Economic Impact of Atrial Fibrillation in the US

Sandra Sieck

Introduction

Atrial fibrillation (AF) represents a challenge to the current healthcare system. The rapidly increasing prevalence of AF and its associated comorbid conditions impose a significant economic burden on an already strained healthcare enterprise. Novel diagnostic and treatment approaches add to the overall costs of this condition at a time when resources are increasingly restrained. The shift to outpatient management coupled with innovative models for efficient inpatient care may contain costs and improve clinical outcomes and overall quality of care.

Epidemiology

AF is the most common cardiac arrhythmia. Although estimates vary, AF affects over 3 million people in the USA [1, 2]. New-onset AF incidence is roughly 1 % between 60 and 68 years of age and 5 % after age 69 [3]. AF prevalence is projected to possibly increase with age [4]. Although uncommon in young persons without structural heart disease, the prevalence rises abruptly after age 60. Prevalence in the general population ranges from 0.4–1 %, increasing to 10–12 % in those >75 years of age [3, 5]. In the Medicare population, the incidence is 28 per 1000 person-years [1]. AF is more common in men than in women and less common in Hispanics, Asians, and African-Americans [1].

The incidence and prevalence of AF are expected to increase dramatically in future decades. The increasing age of the overall population and better treatments

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for comorbid conditions will factor into the dramatic increase in the number of people with AF. The incidence is anticipated to double from 1.2 million cases in 2010 to 2.6 million cases in 2030 by some estimates. AF prevalence is projected to likely increase to 12.1 million cases by 2030 and 15.9 million by 2050 [6]. The cumulative lifetime risk of developing AF is high. In the ARIC study, the cumulative risk for developing AF in Caucasians was 21 % in men and 17 % in women and in African-Americans 11 % in both sexes by age 80 [7]. The healthcare system will require efficient approaches of care to deal with burgeoning cases.

Clinical Impact of Atrial Fibrillation

AF patients have concomitant conditions that are a factor in overall morbidity and costs of care. In a 2012 analysis of Medicare beneficiaries, 84 % had hypertension, 36 % heart failure, and 30 % cerebrovascular disease [8]. These associated medical conditions contribute to the costs of treating these AF patients.

Complications related to AF also impact overall economic burden. The most common associated conditions that affect costs are stroke and heart failure. AF in stroke increases overall costs of care by $7,907 per year. AF in heart failure adds $12,117 to the annual cost [9]. Incremental annual costs to the US healthcare system are estimated to be $372 million when AF is combined with heart failure. For AF with acute myocardial infarction, estimates are $244 million [10].

AF is also common following cardiac surgery. Roughly 10–30 % of postoperative patients experience AF. The occurrence of AF following cardiac surgery adds to the hospital stay and resource utilization. An analysis from the multi-institutional Society of Thoracic Surgeons (STS) certified database for cardiac operations (2001–2012) published in 2014 showed that the length of ICU stay increased (56 vs. 40 h), length of stay (LOS) increased (7 vs. 5 days), ICU costs increased ($6673 vs. $4935), and total hospital costs increased ($29,277 vs. $23,706) in patients with postoperative AF compared with postoperative patients without AF [11].

Economic Burden

The main cost drivers related to AF reside on the inpatient side and are predominantly derived from acute hospital admissions and ED visits. Most of the hospital admissions result from complications related to AF, such as stroke, heart failure, bleeding from over-anticoagulation, myocardial infarction, etc. [12, 13] Outpatient cost drivers are medications, consultations, and interventional procedures. Coyne used 2001 data to show that estimated total hospitalization costs for AF in the USA were $2.93 billion with $1.53 billion for outpatient treatment and $235 million for drugs [10]. Kim showed that from 2004 to 2006 data, the total per-patient cost in AF patients that was specific to AF was $1945 and was distributed as follows: $780 for

inpatient, $972 for outpatient medical (includes ED visits, MD visits, lab services), and $193 for outpatient pharmacy [14].

Accurate statistics for the total costs related to AF are somewhat difficult to estimate due to limited information in the literature and the complex nature of AF with its comorbid conditions [15]. But it is clear that the total economic burden of AF has been increasing in the USA. A 2001 database study estimated total AF costs to be $6.65 billion (in 2005 dollars) [10] (Fig. 3.1). Kim estimated that for the US population in 2005, total incremental costs of AF (in 2008 dollars) amounted to $26 billion ($6 billion from AF, $9.9 billion related to other CV disease, and $10.1 billion for noncardiac causes) [14] (Fig. 3.1).

Hospital costs are the main driver for total AF costs in the USA, accounting for 80 % of the total economic burden of AF. Most patients are admitted though the emergency department (ED) and the number of ED visits for AF have gone up. From 1993 to 2004, the number of visits increased 88 %, from 300,000 to 564,000 [16]. Almost two-thirds of AF patients seen in the ED are admitted to the hospital [17].

Between 1996 and 2001, hospitalizations for AF rose 34 % [18]. In 2001, there were 350,000 hospitalizations [10]. In 2010 there were 479,000 hospital discharges with an AF diagnosis, representing an increase over the 460,000 admissions in 2009 [19] (Fig. 3.2). In a Medicare population, AF patients are almost twice as more likely to be admitted than are similar patients without AF (37.5 % vs. 17.5 %) [14].

Direct medical costs are higher in AF patients than in non-AF patients. Medicare and MarketScan databases from 2004 to 2006 estimated total direct costs for an AF patient at $20,670 versus $11,965 in a control group [14]. Costs are related to the AF itself as well as resultant conditions from AF and side effects of treatments. Up to 70–90 % of AF patients have another medical condition that contributes to

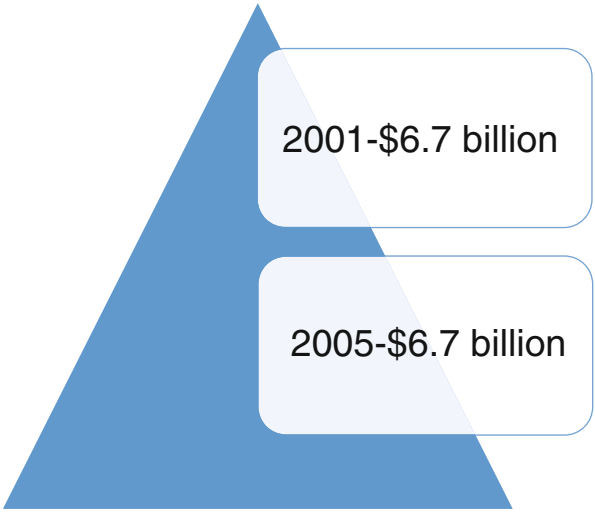

Fig. 3.1 Total annual costs of AF in the USA [10, 14]

Fig. 3.2 Hospital admissions for AF in the USA

increased costs [20]. In 2008, Lee estimated an adjusted mean incremental treatment cost of AF at $14,199 with some of this cost attributable to the incidence of stroke and heart failure at the 1-year post-AF diagnosis [9]. A private plan database from 1999 to 2002 estimated annual direct costs at $15,553 for AF patients compared to $3204 for non-AF patients and annual total costs of $14,875 versus $3579, respectively [20]. Each AF recurrence adds $1600/year to the AF costs. A 2014 study by Patel showed that, adjusted for inflation, average hospital costs for AF increased 24 % between 2001 and 2010 from $6410 to $8439 [21].

Societal costs are even more elusive to quantify. An evaluation by Rohrabacher estimates that lost days related to any cardiac arrhythmias, 40 % of which are AF, were 2.44 adjusted annual mean days of lost work for arrhythmia patients versus 1.85 for employees without arrhythmia [22]. A European study suggests that 9–26 days per year are lost due to AF [23]. Disability related to stroke and/or concomitant heart failure also places increased long-term demand for medical and social services on the healthcare system.

Management Options

The selected treatment modality also has an impact on overall costs. Treatment modalities include noninvasive medical management focusing on medications and invasive treatment with ablation or rarely with surgery.

Noninvasive Approaches

Noninvasive treatment of AF can be broken down into these categories: rate control, rhythm control, and prevention of thromboembolism. In looking at costs related to the therapeutic choices, the AFFIRM study showed that patients in a rate control arm had lower costs and resource utilization than the rhythm control arm by $5077, primarily due to fewer hospital days [24]. Rhythm control can involve drugs and/or DC cardioversion. Costs related to DC cardioversion and the use of medications averaged $4000–5000 in the FRACTAL registry study [25]. Some antiarrhythmic medications are recommended to be started in the hospital or via cardiac monitoring

both by FDA recommendation and specialty society guidelines. Studies show that inpatient monitoring related to sotalol adds $3278 to the cost of AF, and the use of dofetilide adds $3610 [26]. Additional cost studies are required to show comparative cost-effectiveness between different medical management approaches.

Ablation

The evolution of the approach to therapy may alter the total costs of AF. More recent use of ablation to cure and control AF adds to the initial cost of the procedure which is clearly more expensive than typical rate or rhythm control. However, the total cost must be reviewed over the long run, particularly if the procedure reduces recurrences, hospitalizations, and the need for chronic drug treatment. The long-term benefits are in part dependent on user expertise. A Canadian study estimated that the break-even cost point for ablation compared to drug therapy could be up to 3–8 years [27].

Treatment Venue Implications on Costs of AF

As the incidence of AF increases and healthcare resource utilization is subject to increasing constraints, the delivery of healthcare will require more efficient point of care models if the system is to survive and provide quality outcomes. As with other medical treatment options in healthcare, there has been continued emphasis on moving care from the inpatient to the outpatient setting in hopes of reducing overall costs. In looking at options for the AF patient, the categories of patient types play a role in treatment venue: acute AF (either new onset or recurrence) and chronic AF (either with persistent AF or maintenance of sinus rhythm with a history of prior AF). From an economic standpoint, the goal is to efficiently treat acute AF and to monitor and prevent complications related to either AF or its treatment modalities in a cost-effective and quality/evidence-based manner.

For acute care of AF, observation services have become an increasingly acceptable and streamlined approach for evaluation and treatment. For the treatment of the chronic condition related to AF and monitoring needs, specialized atrial fibrillation clinics have become more common in recent years.

Atrial Fibrillation Clinics

In the late 1980s, anticoagulation clinics, or "Coumadin clinics," became popular as a way to safely monitor and control the level of anticoagulation for patients on Coumadin [28, 29]. Rather than depending on a patient's PCP or specialist to constantly adjust Coumadin dose and monitor the level of anticoagulation, a central

clinic took on such functions, often under the auspices of a cardiologist or pharmacist. Patient adherence can be improved, adequate levels of anticoagulation are maintained, and reduced adverse effects of under- or over-anticoagulation are positive outcomes related to these clinics [30]. A 1998 study from Texas also showed that anticoagulation clinic resulted in savings of $162,058 per 100 patients annually by reducing hospitalizations and emergency department visits [31]. A cost-effectiveness Markov analysis showed reduced lifetime costs of AF as a result of an anticoagulation monitoring model of care for patients with AF compared to usual care: $8661 versus $10,746 [32].

More recently outpatient clinics specifically devoted to atrial fibrillation patients have been developed. The idea of a specialized clinic arose from the general lack of adherence to guidelines for treatment of AF and wide variances seen in clinical practice. A study in the Netherlands using cardiologists and specialized nurses showed that coordinated care was associated with improved clinical outcomes compared with usual care [33]. After an average of 22 months, several major adverse events, including stroke, were significantly lower among the atrial fibrillation clinic patients (14.3 % vs. 20.8 %). Reductions were also seen in cardiovascular deaths (1.1 % vs. 3.9 %) and cardiovascular hospitalizations (13.5 % vs. 19.1 %). A cost-effectiveness analysis of this practice model showed promising cost benefits [34]. The outpatient clinic was nurse-led under cardiology guidance with adherence to clinical guidelines using a software-based program. The usual care arm of the study was routine cardiology visits. The nurse-led group had substantial but not statistically significant lower cost per patient. The nurse-led care arm resulted in a slight increase in life-years and quality-adjusted life-years (QALYs) at a lower cost. Therefore, the cost per QALY and cost-effectiveness for total cost per life-year were improved in the nurse-led group: care was less costly and more effective. Outpatient costs related to laboratory tests were higher in the nurse-led group and probably represent up-front costs related to guideline adherence, which would be expected to decrease over time, becoming more cost saving.

The Cleveland Clinic has had an Atrial Fibrillation Clinic and a Stroke Prevention Center, staffed by a multidisciplinary and collaborative team dedicated to evaluation and treatment options for AF patients [35]. In addition to determining the best approach for the treatment of the AF, careful oversight of anticoagulant options is also an arm of the clinic.

Some programs have focused collaboration between the ED and atrial fibrillation clinic. ED visits for AF are sometimes coordinated with an atrial fibrillation clinic for the use of evidence-based treatment and expeditious follow-up after the ED [36]. Further studies are required to corroborate cost-effectiveness of these approaches.

Observation Services

Observation services and dedicated observation units (OUs) for outpatient treatment of medical conditions have been increasing over the last few decades. OUs emerged as a way to provide more efficient and less costly care through a

streamlined, intense therapeutic monitoring and intervention focus. Initial medical conditions that were appropriate for OU included chest pain, asthma, and heart failure, but over time the list of conditions has expanded to include short stay management of patients meeting medical necessity. AF represents another cardiac condition that under certain conditions (e.g., acute onset <48 h, initiation of antiarrhythmic drugs, initiation of anticoagulation, etc.) can be appropriate for OU level of care [37].

Observation services are defined in Medicare's manuals as "a well-defined set of specific, clinically appropriate services, which include ongoing short term treatment, assessment, and reassessment, that are furnished while a decision is being made regarding whether patients will require further treatment as hospital inpatients or if they are able to be discharged from the hospital" [38]. A medical condition that is likely to require a stay <48 h should be considered for observation services (Fig. 3.3).

The Centers of Medicare and Medicaid Services (CMS) studied inappropriate 1-day inpatient hospital admissions. One-day inpatient stays are relatively common in the Medicare program, accounting for over 1 million inpatient admissions (13 % of the total) in 2012 [39]. As part of the Recovery Audit Contractors (RAC), CMS found that short stays not meeting medical necessity criteria for inpatient level of care were responsible for half of the overpayments that CMS made [40]. Observation services represent an ideal approach for such short stays. Diagnostic and therapeutic services are provided in the OU in a stay lasting often fewer than 24 h and rarely lasting longer than 48 h. However, the OU may be seen as reverse

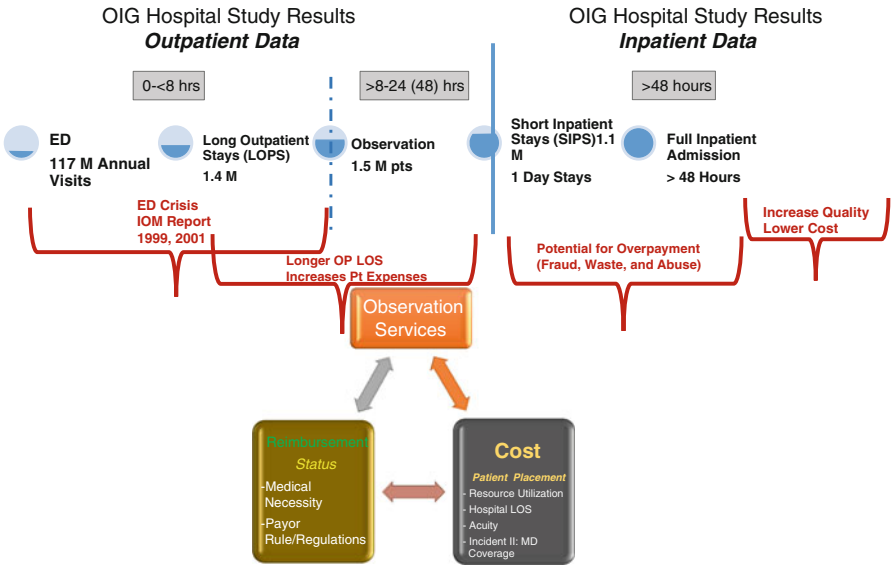

Fig. 3.3 CMS linking hours to expected LOS (Sieck, S, Sieck Healthcare 2015, Department of Health and Human Services, Office of Inspector General Report, July 29, 2013)

incentive for hospital reimbursement. Observation services are considered outpatient services and are currently paid under Medicare Part B, under 1 of 28 comprehensive Ambulatory Payment Classifications (APC). Observation services are paid under composite APC 8009. The estimated 2015 payment is approximately $1,300 [41]. Hospitals generally receive higher payments for clinically similar patients served in an inpatient setting compared with an outpatient setting, and the services provided are similar; therefore, hospitals may have a financial incentive to admit patients [42].

With an aging population, the demand for ED visits and inpatient admissions will increase. Appropriate use of observation unit services have the added benefit of reducing ED overcrowding and ambulance diversion and opening up inpatient beds. Ross performed an interesting analysis comparing inpatient length of stay (LOS) by confidence intervals (rather than averages) and then applying these LOS to the EDOU patients. Based on this methodology, each single EDOU bed was predicted to make 2.35 or 3.16 inpatient beds available [43]. It is reasonable to also conclude that if an OU bed was not located in the ED, overcrowding the ED would be reduced as well.

In the current environment of quality measurement and oversight, the increased use of OU services can also play a role in enhancing compliance with Medicare rules and preparing facilities for upcoming value-based pricing/reimbursement arrangements. But it can also pose challenges to facilities. Hospital's frequently report utilization measures to external agencies. Some of the measures such as inpatient admissions and length of stay (LOS) are likely to change as more patients are seen in OU or outpatient setting. Attempts to compare institutions that record observation status differently, such as in inpatient versus outpatient data, may be difficult. A lack of consistency in patient placement, documentation, and coding has lead payors to make assumptions based on the currently reported data.

The use of observation services for more medical conditions will also impact the cost structure of medical care. A Healthcare Cost and Utilization Report of facility data showed sets of 2- or 3-day stays occurring in the outpatient setting, suggesting patients remained in the outpatient venue for diagnosis or treatment [44]. Correct coding of place of service could impact an institution's case mix index, LOS, costly diagnostic and therapeutic services, and penalties imposed through the new CMS payment methodology value-based purchasing (VBP).

In addition to a potential reduction in costs of care, in order to remain a viable venue setting, the OU must also demonstrate improved or at least similar clinical outcomes. Prior studies in chest pain, heart failure, and asthma show cost benefits and improved clinical outcomes with OU care. Peacock showed that the use of the OU for heart failure resulted in decreased 30-day admission rates, hospitalizations, and length of stay for future hospitalizations [45]. Cost savings through reduced inpatient admission, increased patient safety results, and improved patient satisfaction have also been demonstrated for chest pain [46]. In a literature review, Baugh estimated potential national cost savings from increased use of observation units by hospitals would be $1,572 per patient with resultant annual hospital savings of $4.6 million and national cost savings of $3.1 billion [47]. A study on the use of

an accelerated diagnostic protocol in the OU for TIA patients showed a 20.8-h shorter median stay and lower median associated costs of $1643 [48]. A Georgia study showed the use of an OU resulted in an overall 23–38 % shorter length of stay and a 17–44 % reduction in subsequent inpatient admissions. The authors estimated that 11.7 % of all inpatients admitted across the USA could be treated in an EDOU, with potential savings of $5.5 billion to $8.5 billion annually for the USA [49].

While it might be intuitively plausible to expect such cost savings can be extrapolated to certain segments of the AF population, limited studies on outcomes or cost-effectiveness of the OU in AF are available to date. A 2008 study of 153 acute AF patients (<48 h duration) randomized 75 to the ED observation unit and 78 to inpatient care [50]. OU patients experienced a higher rate of conversion to sinus rhythm (85 % vs. 73 %), and the median length of stay was 10.1 versus 25.2 h ($P < .001$) compared to acute inpatient admission. There was no difference in recurrence of atrial fibrillation during follow-up and no significant difference in the number of adverse events during follow-up between the two groups. This report suggests that AF can be safely and effectively treated in the OU setting. Additional future cost-basis and outcomes studies in AF are needed.

Inpatient Admission

Not all AF patients are appropriate for observation status treatment, and inpatient admission may be most appropriate. Compared to a fully staffed protocol-driven OU, the average inpatient hospital stay often utilizes outdated methods of care that are more geared toward partial workday hours of operation and less adherence to care pathway or programs designed to streamline patient evaluation and management for more routine medical conditions. Variances in care patterns also add to inefficiencies.

For the segment of the AF population that requires acute hospitalization, achieving efficiencies in the hospital flow is critical to resource and cost containment. The process of care in a hospital setting can be analogous to a business model in an industrial setting. Most hospital patients follow a zigzag approach when receiving care/services from the point of entry to discharge (Fig. 3.4). A patient's "flow" through the hospital care system is often not linear. The patient is shuttled through various diagnostic or therapeutic care units (e.g., radiology, laboratory, imaging department, pharmacy, etc.) in a disconnected manner. Each care unit functions more as an independent unit than as an integrated part of a cohesive strategy. Transfer between care units is not always a smooth and seamless interface. Each unit acts as a single entity from the hospital's standpoint, but should not from the patient's perspective. It is incumbent upon the physician to collate the output of the care units' results. Although the final outcome eventually is appropriate care, the zigzag process is generally an inefficient, untimely, and resource-wasteful process.

Several methods have been used in process improvement approaches to enhance inpatient efficiencies and quality of care. Care maps, care pathways, critical pathways,

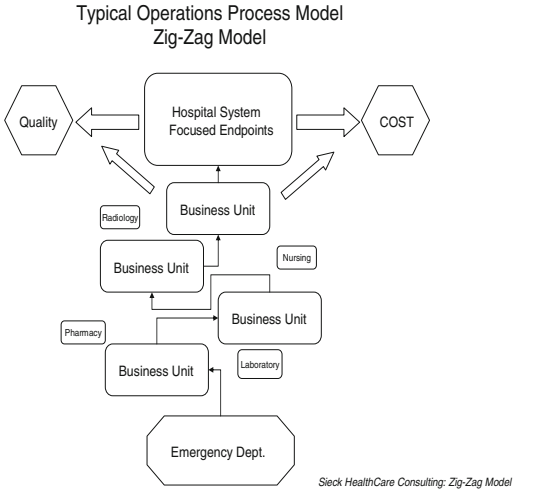

Fig. 3.4 Zigzag model of care (2015 update: Sieck [53])

and integrated pathways are detailed medically appropriate paths that outline daily steps to diagnostic and therapeutic interventions for a particular medical condition that are designed to organize care into an efficient process toward resolution. However, financial concerns were usually not a direct consideration in these pathways. Application of a business design model that merges quality of care and optimal financing in the process of care will insure long-term facility viability.

The Y Model approach affords such a blueprint for this merger [51]. The Y Model focuses on the desired end points of quality and costs. In the business environment, the key to delivering a quality end product at the maximal contribution margin is to streamline manufacturing process and reduce variances in production steps. This translates in medicine by requiring adherence to evidence-based evaluations and treatments performed expeditiously and efficiently through a streamlined process. The Y Model involves placing proper sequencing of services "up front" at the point of entry into the medical care track. Seamless integration between operating care units is the essential core of the Y Model (Fig. 3.5).

Instituting the Y Model in other cardiac conditions has shown positive impact of quality parameters, reduced costs, and improved clinical outcomes. One such example is in treatment of acute coronary syndrome (ACS) [52]. Once a patient was defined as ACS in the ED, stratification was performed and appropriate therapy begun in the ED rather than waiting until ICU bed placement. Treatment was individualized and there was no gap in care services between the ED and ICU. This patient-centric analytic process resulted in identifying care gaps for optimizing outcomes, quality, cost, and patient satisfaction (Fig. 3.6).

While there are no accurate cost estimates for a well-designed process flow for inpatient AF care, it could be opined that cost reductions similar to that seen in the OU could be obtained. The streamlined process for expeditious evaluation paralleled with initiation of monitoring and treatment from the initial point of entry integrates

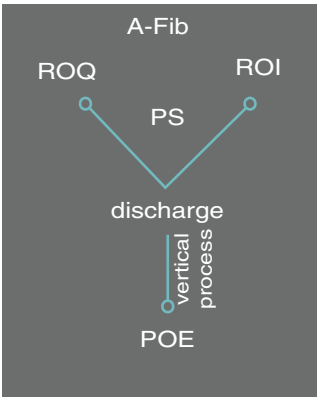

Fig. 3.5 Transformation of data, changing bedside care (2015 update: Sieck [54])

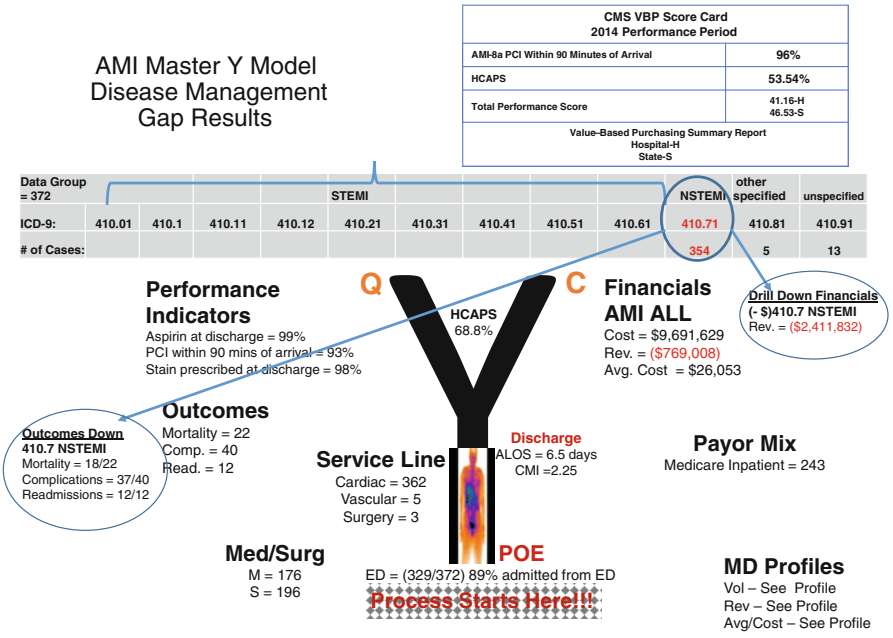

Fig. 3.6 Identifying beside care gaps

a financial strategy that meets both quality metrics and evidence-based case management protocols. Beginning in the ED, this approach focuses on immediate evaluation and initiation of actions centered on seamless integration of ancillary services such as imaging studies, laboratory assessments, skilled nursing and near-continuous provider oversight, and therapeutic/diagnostic adjustments.

Adapting the efficient process flow of the OU to the inpatient setting should result in overall cost efficiencies while maintaining or improving quality. The OU may represent the initial redesign in acute healthcare delivery that will ultimately transform the entire system into a more efficient process. Using this redesign with the Y Model application overlay could result in potential significant cost savings and improved quality of care.

Summary

Atrial fibrillation is a common and costly condition. As prevalence is predicted to increase significantly in the upcoming years and more costly treatment options become available, more efficient approaches to patient management will be needed to constrain burgeoning cost impact of the US healthcare system.

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Part II
Presentation and Management

Chapter 4
Emergency Medical Services and Atrial Fibrillation

Marvin A. Wayne, Andrew M. McCoy, and Richard B. Utarnachitt

Introduction

Emergency medical services (EMS) personnel frequently encounter patients with atrial fibrillation (AF). Atrial fibrillation is an irregularly irregular rhythm which is generated by multiple foci originating above the ventricles in the atria. Approximately 3–6 million residents of the United States have atrial fibrillation [1, 2], accounting for around 270,000 emergency department visits per year [3]. Many of these patients will be transported to the emergency department by emergency medical services (EMS). Therefore, it is prudent that prehospital providers be familiar with how to approach AF in the field.

Field Assessment

The prehospital provider has a limited number of diagnostic tools and therapeutic options to detect and care for the patient with atrial fibrillation. Luckily, teaching an EMS provider to detect AF is not difficult, as this is nearly always the cause of a

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cardiac rhythm that is *irregularly irregular*. As this term is almost pathognomonic for AF and is eminently teachable, this rhythm is easily learned by prehospital providers.

EMS Scope of Practice

It is important to define the scope of practice for prehospital providers. In the United States, several levels of EMS providers exist. These are largely determined by state laws and may be broadly divided into two groups, those that provide Basic Life Support (BLS) and those that provide Advanced Life Support (ALS). Within this dichotomy, many other levels may exist (intermediate, EMT-1, EMT-2, etc.). Differences between these levels depend upon the scope of practice at the national level as well as state, regional, and local protocols. Often those at the BLS level have few interventions to offer to the patient in AF. ALS providers will be able to utilize Advanced Cardiac Life Support (ACLS) protocols.

All providers, no matter their level, should have an understanding of atrial fibrillation and that atrial fibrillation is the most likely origin of a patient in an irregularly irregular heart rhythm on their physical assessment. A BLS provider should, at the very least, recognize this as an abnormal pulse rate and/or rhythm.

Chief Complaint

Patients with atrial fibrillation have a wide variety of primary complaints on evaluation. These can be wide ranging and include palpitations, an elevated heart rate, shortness of breath, chest pain, and weakness.

Physical Examination

Field providers are educated from an early point in their training to perform a careful, yet focused, physical examination. At all levels, this includes taking a pulse, including its inherent rhythm. This is one possible place providers may identify atrial fibrillation, with detection of an irregularly irregular rhythm. Most providers are also taught to evaluate signs of normal vs. altered perfusion including skin temperature, capillary refill, and mental status. Other common physical findings in atrial fibrillation include pale and diaphoretic skin, labored breathing, and jugular venous distention. Determining these findings will help the EMS providers in their evaluation of the patient with atrial fibrillation.

ECG

The 12-lead electrocardiogram (ECG) is the mainstay of diagnosis of atrial fibrillation and should be obtained on every patient. Atrial fibrillation is defined as an irregular R-R interval. Advanced Life Support providers were able to detect atrial fibrillation 71–86 % of the time on 3-lead interpretation examination [4, 5]. While a 3-lead, 4-lead, or 5-lead are often used to detect dynamic changes in the patient's condition, the formal 12-lead ECG is critical to evaluate for potential underlying causes of atrial fibrillation, including acute myocardial infarction.

The EMS Challenge

Interventions

Specific interventions for the patient in atrial fibrillation will vary by the level of the provider, the protocols in effect, and the clinical condition of the patient. Included in this section is a broad cross section of the available treatments. Emergency medical services physician leadership should be heavily involved in the development and refinement of local prehospital treatment protocols and guidelines. If prehospital intervention is warranted, it is focused on rate control in symptomatic patients and rhythm control in unstable patients. Rate control is first considered when AF is accompanied by rapid ventricular response (RVR) defined as a ventricular rate >100 bpm.

General treatment as indicated in the prehospital setting would include:

- Peripheral IV
- 12-lead ECG and continuous cardiac monitoring
- Rate control with either calcium channel-blocking or beta-blocking agent if indicated (not usually required if rate is <140 and hemodynamically stable)
- Cardioversion if patient is hemodynamically unstable (hypotension or signs of hypoperfusion)

Aspirin

Aspirin is commonly available. It is a cheap salicylate drug that is used as an analgesic, antipyretic, and anti-inflammatory agent and most importantly an antiplatelet agent. Aspirin has been shown to significantly decrease mortality in patients with acute myocardial infarction [6]. Some of these patients are in AF secondary to acute ischemia. They may present with chest pain or other possible cardiac complaints. EMS providers are often requested or required per local protocol to treat these patients with aspirin. Anticoagulation in AF will be discussed in more detail in Chap. 9.

Pharmacologic Rate Control

Calcium Channel Blockers

Diltiazem is the most commonly utilized calcium channel blocker for managing narrow complex atrial fibrillation with rapid ventricular response. Diltiazem has been found to lower heart rate in patients with atrial fibrillation with a ventricular response rate over 150 beats per minute when compared with non-pharmacologic treatment. In one study by Wang et al., 81 % of patients were found to have a therapeutic response to diltiazem compared with 17 % in the control group [7]. A second study had similar findings with diltiazem having prehospital efficacy of 73 %, and only 0.7 % (2/278) of patients who received diltiazem had an episode of hypotension [8]. Therefore, diltiazem is considered to be a safe and effective intervention for patients with narrow complex AF with RVR in the prehospital environment.

Verapamil has been used for AF with RVR with success in both the emergency department and the prehospital environment. Little has been published, however, about the prehospital use of verapamil. However, based on pharmacologic similarities, emergency department use, as well as EMS use, it would also be presumed to be an effective treatment choice.

Beta Blockers

No placebo-controlled studies of beta blockade for the treatment of atrial fibrillation with rapid ventricular response in the prehospital environment are available. Metoprolol is the most commonly utilized beta blocker for atrial fibrillation in the hospital setting [9]. Head-to-head trials in the emergency department, however, have been performed.

Calcium Channel Blocker Versus Beta Blocker

While no prehospital studies have been performed, there are several emergency department studies examining calcium channel blockers and beta blockers head to head. Demircan et al. randomized 40 patients to weight-based doses of either diltiazem or metoprolol for atrial fibrillation with heart rate greater than 120 beats per minute. They found diltiazem to have better and earlier heart rate control with a similar side effect profile [10]. Fromm et al. randomized 54 patients to a similar regimen; in 30 min, 95 % of the diltiazem group and 46 % of the metoprolol group had reached target heart rates with no difference in rates of hypotension or bradycardia [11]. While these studies are emergency department based, the pharmacology likely is similar in the prehospital environment. These two randomized clinical

trials are the only high-quality evidence available to base recommendations on calcium channel blockade, specifically diltiazem, over beta blockade as a strategy to treat atrial fibrillation with rapid ventricular response.

It is unadvisable to utilize both beta-blocking and calcium channel-blocking agents simultaneously. Blocking both beta receptors and calcium channels can lead to negative chronotropic, inotropic, and dromotropic effects [12]. The combination, when administered to healthy volunteers, significantly lowered heart rate, compared to either alone. However, the combined drug administration also significantly increased adverse effects, most notably fatigue and first-degree heart block [12].

Adenosine

Some EMS systems allow for prehospital treatment of supraventricular tachycardia (SVT) with adenosine. Adenosine blocks conduction through the AV node. Care must be taken to instruct ALS providers about the risks of this strategy, namely, that if SVT is incorrectly diagnosed, significant adverse outcomes can result. Haynes reported two cases of atrial fibrillation that became fatal when adenosine was used to treat presumed SVT [13]. Gupta et al. described four emergency department patients that went into a ventricular tachycardia after administration of adenosine for presumed SVT that was later found to be atrial fibrillation [14]. These cases likely represent atrial fibrillation in the presence of accessory conduction pathways, also known as Wolff–Parkinson–White (WPW) syndrome. Patients with WPW syndrome will frequently decompensate when adenosine is administered as their accessory conduction pathway takes over and their ventricular rate elevates into the 200s with increasing hemodynamic instability. For this reason, adenosine is generally avoided in cases of atrial fibrillation or irregularly irregular tachycardic patient.

Electrical Rate Control

Cardioversion/Defibrillation

Patients that are in atrial fibrillation and are hemodynamically unstable may require cardioversion. Electrical cardioversion for atrial fibrillation was first described in the 1960s [15]. Atrial fibrillation in a hemodynamically unstable patient should be treated with synchronized cardioversion, where possible, using a biphasic waveform. In a double-blind randomized trial of elective cardioversion, a biphasic waveform was found to be superior to a monophasic waveform [16]. Sedation for cardioversion is outside the scope of this chapter but will be discussed in detail in Chap. 11 of this book. Generally, sedation for cardioversion is recommended whenever possible, for patient comfort. Indications for defibrillation or non-synchronized cardioversion are discussed in Chap. 10.

Conclusions

Atrial fibrillation is a disorder commonly encountered in the prehospital environment. EMS providers play a vital role in extending the patient's care from the emergency department into the prehospital arena. This can be done despite the relatively limited resources in the prehospital setting. Management should be focused on stabilization with rate control using calcium channel blockers or beta blocker therapy with cardioversion reserved for only hemodynamically unstable patients. Through earlier access to advanced therapies, patients may have improved outcomes.

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Chapter 5
Symptoms of Atrial Fibrillation

Shahriar Dadkhah and Korosh Sharain

Introduction

Atrial fibrillation (AF) is the most common clinically significant cardiac arrhythmia and affects over 5 million adults in the United States [1]. It is associated with considerable morbidity and mortality from complications such as heart failure, stroke, and other embolic sequelae. Although up to one third of patients with AF are asymptomatic, those who do experience symptoms of AF often seek emergency medical attention. Additionally, approximately 70 % of first detected AF episodes are diagnosed in the hospital or emergency room [2]. Recent data suggests that over two thirds of patients diagnosed with AF in the emergency department are eventually admitted to the hospital [3]. Therapeutic strategies aim to not only reduce associated morbidity and mortality but also control associated symptoms. Multiple studies have demonstrated that long-term clinical outcomes based on rate versus rhythm control are similar; therefore, an individualized approach based on arrhythmia-related symptoms remains an additional factor when determining treatment goals [4–7]. There are significant costs and complications associated with pharmacologic and non-pharmacologic invasive treatments for AF from a symptom standpoint, making appropriate symptom assessment imperative. However, there is no accepted gold standard for assessing symptoms in AF given its variable nature. The aims of this chapter are to review the symptoms associated with AF and describe their proposed mechanisms, briefly discuss symptom scoring tools, and describe targeted symptom management.

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Symptoms

The symptoms associated with AF are extremely variable and can range from an asymptomatic incidental electrocardiographic (ECG) finding to overt signs of acute heart failure (Table 5.1). However, it is difficult to attribute symptoms of AF solely to the arrhythmia as many of these patients have multiple comorbid conditions such as heart failure or valvular disease which could cause similar symptoms. In fact, up to 90 % of those with AF have been found to have a comorbid condition [8]. Additionally, there exists both inter-patient and intra-patient symptom variability. Symptoms in the same patient can differ based on the disease course, as well. Two large prospective studies by Nabauer et al. and Lévy et al. characterized the numerous clinical presentations observed in the AF population [9, 10]. They were also able to describe symptoms based on type of AF. Their findings along with other selected studies which evaluated symptoms of AF are presented in Table 5.2 [9–12]. The next section will review symptoms that have been associated with AF, with the caveat that coexisting conditions likely play a role or can be primarily responsible for symptom association.

Palpitations

Up to one half of patients with AF may experience the awareness of their irregular heartbeat [9–11]. Palpitations are the most common presenting symptom associated with paroxysmal AF [9, 10]. In fact, palpitations may be the only symptom that occurs more often during an episode of AF compared to sinus rhythm. However, the mechanisms responsible for palpitations have not been fully elucidated. The neural pathways responsible for palpations are unknown and may not even originate from the myocardium itself. Interestingly, denervation of the heart in heart transplant recipients does not prevent the sensation of palpitations [13, 14].

Table 5.1 Symptoms of atrial fibrillation

Palpitations
Dyspnea
Chest discomfort, chest pressure, chest pain
Reduced exercise tolerance
Dizziness, presyncope, syncope
Anxiety
Depression
Fatigue
Polyuria

Table 5.2 Selected trials evaluating symptoms of atrial fibrillation (AF)

		Palpitations	Dyspnea	Chest Pain/discomfort	Syncope, dizzy	Fatigue	Other	None
Nabauer et al. [9][a]	Total ($n=8942$)	41.9	44.3	18.7	25.3	44.9	–	24.5
	Paroxysmal ($n=3928$)[b]	54.7	40.2	21.6	28.3	48.1	–	20.3
	Persistent ($n=1873$)[c]	41.4	47.5	18.8	24.9	49.0	–	23.3
	Permanent ($n=3141$)[d]	26.1	47.5	15.1	21.9	38.4	–	30.4
Lévy et al. [10]	Total ($n=756$)	54.1	44.4	10.1	10.4	14.3	0.9	11.4
	Paroxysmal ($n=167$)†	79.0	22.8	13.2	17.4	12.6	0.0	5.4
	Persistent ($n=200$)[e]	51.5	58.0	11.0	9.5	18.0	0.0	7.0
	Chronic ($n=389$)[f]	44.7	46.8	8.2	8.0	13.1	1.8	16.2
Lok et al. [11]	Total ($n=291$)	42.3	38.1	7.9	16.5	–	–	–
Lip et al. [12]	Total ($n=170$)	25.9	51.8	34.1	18.8	–	–	–

All values represented as percentages (%) unless stated otherwise

[a]Number in each group and percentages were calculated based on published data

[b]Includes first detected AF episode, paroxysmal defined as AF that lasts less than 7 days

[c]Defined as recurrent or sustained AF that lasts more than 7 days

[d]Defined as long-standing AF in which cardioversion has failed or has not been attempted

[e]Defined as AF lasting greater than or equal to 7 days but less than 1 month

[f]Defined as AF present for greater than 1 month

Dyspnea

Dyspnea is also a common symptom and can occur in over 40% of those with AF [9, 10, 12]. Studies demonstrate that dyspnea is the most common presenting symptom of longer-duration AF, such as persistent or permanent AF, which is common among the elderly [9, 10]. The mechanism responsible for dyspnea may be an increase in left-sided pressures including an elevated mean pulmonary wedge pressure and a reduction in stroke volume, however, left ventricular end-diastolic pressure was found to be decreased, in one study that compared induced AF [15]. A secondary tachycardiomyopathy may be another cause of dyspnea in AF [16, 17]. Dyspnea is also one of the most common symptoms in heart failure and may cloud the picture as AF and congestive heart failure (CHF) are closely related and often predict each other's development.

Reduced Exercise Tolerance

Reduced exercise tolerance is also common and may occur in over half of AF patients but can be confused with dyspnea [18, 19]. However, exercise tolerance has been shown to improve after conversion of AF to sinus rhythm [18]. It is estimated that AF can cause a reduction in exercise tolerance by up to 20% [19]. There is some suggestion that increased heart rate variability is associated with improved exercise tolerance [20].

Chest Discomfort, Pressure, Pain

Chest pain may occur in 10–20% of patients with AF although other studies have documented higher rates [9–12]. Such symptomatology may relate to impairment in myocardial perfusion or increased coronary artery resistance and can be present even in those without coronary disease or critical valvular disease [20–22]. Chest pain is present in those with rapid ventricular rates and in those with slow ventricular rates so chest pains are likely not purely rate related and involve other unknown mechanisms [22]. Derangements in the renin-angiotensin and sympathetic systems may also play a role in the sensation of chest pain [21, 22].

Dizziness, Presyncope, Syncope

Dizziness or syncope may occur in up to one quarter of patients with AF [9]. There is suspected to be a very complex interplay between the sympathetic and parasympathetic nervous systems in AF which may explain these symptoms [20]. In fact, Holter monitor data demonstrates that sympathovagal imbalance may cause such

symptoms [23]. Other suspected mechanisms include hemodynamic compromise from the arrhythmia, although this is not as likely without an underlying bypass tract or structural disease [15]. Pauses associated with sinus node dysfunction are another proposed mechanism [16].

Other Symptoms

Anxiety, depression, and fatigue have also been documented symptoms of AF [24]. Almost one third of patients with AF may have depression and anxiety [24]. Symptoms of depression can predict quality of life and atrial fibrillation recurrence after cardioversion [24, 25]. Polyuria from release of atrial natriuretic peptide has also been described [26]. Additionally, 15–25 % of patients with AF present with stroke from embolic phenomenon [4, 27]. Therefore, when evaluating patients with stroke or transient ischemic events, a history of AF may not be present as the cerebrovascular event may be the presenting episode.

Asymptomatic

One of the main challenges in identifying AF is that 11–32 % of those with AF are asymptomatic [4, 9, 10, 28, 29]. Much of this data is derived from the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study and patients with implantable pacemakers or defibrillators [4, 30–33]. When studies evaluated symptomatic calls in those with implantable devices, approximately 31 % of calls were made when patients were in normal sinus rhythm and 69 % while they were in AF [32]. Another study demonstrated that almost 65 % of documented AF episodes were asymptomatic [33]. Ambulatory monitoring studies have demonstrated that the ratio of asymptomatic to symptomatic AF episodes was approximately 12:1 [30]. Therefore, symptoms are an unreliable diagnostic tool overall; however, symptoms remain the leading cause of seeking medical care in those with AF.

A study evaluating AF in post-cardiac surgery patients demonstrated that a first documented episode of AF could be detected up to 21 days post operation, the majority of which were asymptomatic (69 %); the second most common symptom was palpitations (17 %) in this group [14].

Although we have identified symptoms which may be attributed to AF, we do not understand why some patients are symptomatic while others remain asymptomatic. The AFFIRM study did find that those who were asymptomatic were more often male; had lower incidence of coronary disease, heart failure, and pulmonary disease; and had better left ventricular function [4]. However, asymptomatic patients were more likely to present with cerebrovascular events, possibly from delayed medical treatment during their asymptomatic state [4]. Additionally, in those with a longer duration of AF or those who develop permanent or persistent AF, symptoms

may actually reduce or disappear [5, 10, 11]. Interestingly, patients with symptomatic paroxysmal AF have been found to have a tenfold greater likelihood of having an asymptomatic recurrence [30].

Additionally, ventricular rates may play a role in symptom generation. The suppression of paroxysmal atrial tachyarrhythmia trial (SOPAT) and AFFIRM demonstrated a direct correlation between ventricular rate and symptoms of the arrhythmia [4, 34]. Other studies have been unable to establish such a relationship [4, 34–36].

Symptom Scoring

As described above, the symptoms of AF are variable and often overlap with comorbid conditions. Therefore, symptom scoring tools have been created in an attempt to objectify such subjective and variable data. In a short stay unit, such tools are likely less practical; however, they do deserve brief mentioning. Recently, a large push has been made to better characterize symptoms of AF, mainly to better assess optimal management strategies in clinical trials. Tools such as the University of Toronto Atrial Fibrillation Severity Scale, the Canadian Cardiovascular Society Severity of Atrial Fibrillation Scale, European Heart Rhythm Association (EHRA) scale, and the Short Form 36 have all been utilized [37–41]. However, the clinical utility of these scoring systems is unknown. A major criticism of these scoring tools is the difficulty in capturing symptoms attributed to AF versus comorbid conditions.

Symptom-Directed Therapies

Other chapters will address management of atrial fibrillation; however, a brief discussion regarding symptom-directed therapies is warranted. Targeted treatment directed at symptoms has been evaluated in multiple studies. When comparing rate versus rhythm control strategies for AF, no significant difference was found in symptom improvement [4–6, 42, 43]. However, other studies have demonstrated that restoring and maintaining sinus rhythm improved symptoms and functional status [18]. Other studies also showed improvement in exercise capacity with cardioversion as the rhythm control strategy [2, 19, 44, 45]. Additionally, AV node ablation with pacemaker placement did demonstrate improved symptom scores, quality of life, and functional status [46, 47]. Several studies have evaluated the use of pulmonary vein isolation and its effects on symptoms, exercise capacity, and quality of life and found that these parameters were in fact improved [48, 49]. It has been shown, regardless, that if symptoms were improved by a specific strategy, those with more severe symptoms benefited the most.

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Chapter 6
Stroke and Other Complications of Atrial Fibrillation

Michelle Wiener

Atrial fibrillation (AF) has important health implications for patients as well as society, as it is associated with substantially increased rates of hospitalizations, heart failure, mortality, and healthcare costs [1, 2]. Patients that suffer from this condition are at a twofold increase in overall mortality and approximately a fivefold increase in the risk of stroke [3, 4]. The increased morbidity and mortality associated with this condition are multifactorial, with the majority of complications arising from associated comorbidities, the underlying arrhythmia, and the side effects of treatment. For the purpose of this chapter, only the complications directly related to AF will be discussed, as treatment modalities will be presented in future chapters. AF is associated with three major complications: hemodynamic compromise, arrhythmogenesis, and thromboembolism.

Hemodynamic Compromise

The initial evaluation of any AF patient must include an assessment of hemodynamic status, as the arrhythmia may reduce cardiac output by up to 24 % [5] and result in acute hypotension. From a mechanistic standpoint, this is due to a combination of factors that involve the loss of effective atrial contractions and an irregular ventricular rhythm. Often times the patient's already compromised status is then exacerbated by a rapid ventricular rate, which can further exacerbate an already reduced cardiac output. If not treated properly this can lead to severely depressed cardiac function with pulmonary edema, heart failure, or hemodynamic collapse.

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Increased demand from an excessive heart rate can also exacerbate underling cardiovascular disease leading to demand ischemia or myocardial infarction.

Arrhythmogenic Complications

It is estimated that up to 40 % of Wolff-Parkinson-White (WPW) patients will experience paroxysmal AF during their lifetime [5]. These patients have an increased risk of ventricular fibrillation that is often precipitated by AF, especially if they are treated with drugs such as digoxin or calcium channel blockers. While sudden cardiac death outside of this population of people with WPW is not a well-studied complication of AF, a retrospective cohort study of 15,439 patients from the Atherosclerosis Risk in Communities (ARIC) data set found that the risk of sudden cardiac death was doubled in the participants with AF, even when accounting for heart failure or myocardial infarction [6].

Thromboembolism and Stroke

The most common and widely studied complication of atrial fibrillation is thromboembolism. The percentage of strokes attributable to AF is based on age and ranges anywhere from 1.5 % in people 50–59 years of age to 23.5 % of people 80–89 years old [7]. Of note, these numbers are likely conservative estimates as AF is often asymptomatic and therefore underreported. Although the mechanism at present is complex and not fully elucidated, the major contributors to clot formation are thought to be from cardiac stagnation of blood flow, endothelial dysfunction, and atrial stunning [8]. Interestingly, new research suggests that even patients who have no evidence of thrombus on echo prior to undergoing cardioversion may still be at increased risk of clot formation due to atrial stasis from restoration of sinus rhythm and decreased blood flow to the left atrial appendage [9]. This may explain the findings of the Finnish CardioVersion Study, which demonstrated an increased incidence of post-cardioversion thromboembolic complications in patients who were sent home without anticoagulation (cardioverted within 48 h of AF onset). Patients with heart failure (odds ratio [OR], 2.9), diabetes (OR, 2.3), or female sex (OR, 2.1) were at the highest risk, with diabetic heart failure patients reaching a 9.8 % stroke rate in the study [8].

Similarly, while it seems intuitive that paroxysmal AF would be associated with lower rates of thromboembolism, studies suggest that even short episodes (15 min) are enough to cause local platelet activation and endothelial damage that increase a patient's risk of stroke [10]. This risk was further assessed by a study that utilized dual chamber pacemaker and ICD devices to detect subclinical atrial tachyarrhythmias in patients without a history of AF, which found that this was independently associated with a 2.5-fold increase in stroke risk [11].

Based on the increased rates of stoke in this population, it follows logically that the major goal of therapy in these patients is to prevent clot formation. Unfortunately, it is well established that warfarin is more effective than aspirin in preventing thromboembolic events, which also carries an increased risk of bleeding complications [12]. Consequently, risk stratification schemes have been developed in order to identify the subset of patients whose benefit from anticoagulation outweighs their risk of hemorrhage.

The two strongest risk factors associated with ischemic stroke are mitral stenosis and a history of previous stroke or transient ischemic episode. In patients with nonvalvular AF, the next most important risk factors include diabetes mellitus, hypertension, heart failure, female sex, and age. While many risk stratification scoring systems exist for thromboembolism, the 2014 American Heart Association/American college of Cardiology/Heart Rhythm Society (AHA/ACC/HRS) guidelines recommend the CHA_2DS_2-VASc score for evaluation of stroke risk in nonvalvular AF with level 1B evidence [13] (Fig. 6.1). Stroke risk based on the CHA_2DS_2-VASc score is listed in Fig. 6.2. While this system has similar accuracy to the CHADS2 score that preceded it for identifying high-risk patients (but lacks its simplicity), it better differentiates patients that are at low to medium risk [14–18]. This is important as the threshold for anticoagulation is decreasing as the use of non-vitamin K antagonist oral anticoagulants (NOACs) has increased in clinical practice, as they consistently demonstrate a lower risk of major bleeding and less intracranial bleeding when compared to the use of warfarin (but similar incidence of clinically relevant bleeding) [19, 20]. In one analysis based on the use of NOACs, the study threshold recommendations for initiation of oral anticoagulation were as low as a 0.9 % stroke risk [21], which translates to any patient with a CHA_2DS_2-VASc score of ≥ 1.

Fig. 6.1 CHA_2DS_2-VASc scoring system for the evaluation of stroke risk in AF patients [16]

CHA_2DS_2-VASc	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥ 75	2
Diabetes mellitus	1
Stroke/TIA/Thromboembolism	2
Vascular disease (prior MI, CAD, aortic plaque)	1
Age 65 to 74 years	1
Sex (female)	1
Maximum score	9

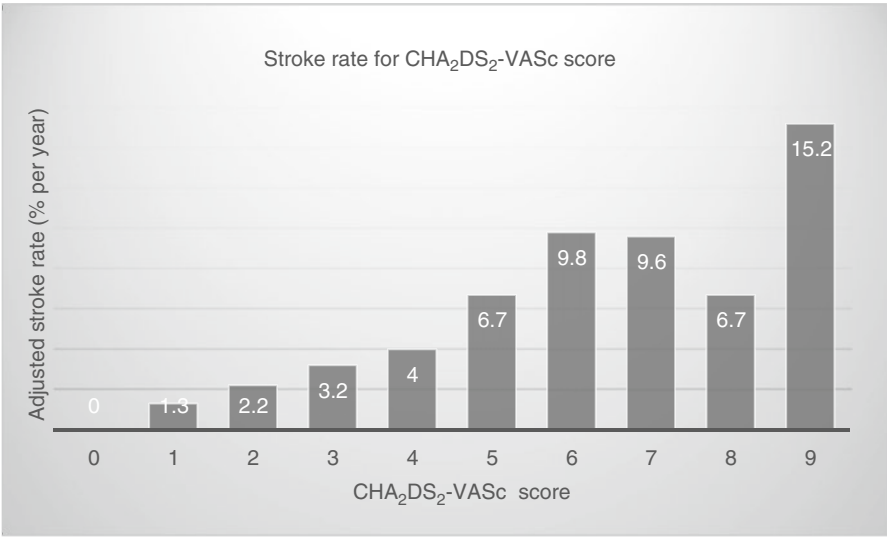

Fig. 6.2 Adapted from 2014 AHA/ACC/HRS guidelines. Adjusted stroke rate may vary depending on population studied [13]

It should be noted that several large studies have also demonstrated that renal failure is also an independent risk factor for stroke in AF patients, with a directly proportional relationship between the severity of renal dysfunction and the risk of thromboembolism [22–24]. This is encompassed by the ATRIA score, which combines the CHADS2 score with female sex, proteinuria, and low estimated glomerular filtration rate or end-stage renal disease. The ATRIA score has been independently validated and is superior at predicting severe events [22]; however, in direct comparison to the CHA_2DS_2-VASc score, the ATRIA classification had the same limitations of the CHADS2 score and is poor at identifying low-risk patients [25]. Perhaps, future risk stratification will examine the addition of chronic kidney disease/end-stage renal disease to the CHA_2DS_2-VASc score.

Dementia

There has been debate as to the role of AF in the development of dementia, which stems largely from the fact that both conditions increase with age as well as are associated with many of the same comorbidities. Several observational meta-analysis studies found an independent association between AF and the risk of dementia (hazard ratio 1.42) [26, 27] and loss of independence in performing activities of daily living (HR 1.35) [28], but this link has not been established in the prospective population trials [29, 30]. Recently, a new study examining increased rates of cognitive decline among AF patients without a history of stroke found that this increase was due to an in increased rate of subclinical cerebral infarcts that were

discovered on magnetic resonance imaging (MRI) [31] This provides a potential mechanism as well as further evidence for the role of anticoagulation in preventing long-term complications of AF, but more research is needed to clarify the relationship between AF and dementia.

Summary

Early diagnosis and treatment of AF are essential in order to reduce the risk of serious complications that are associated with this condition. As the population ages, the burden of AF to patients and hospitals is going to increase. Having a better understanding of the long-term complications and developing a systematic approach to risk stratification will ensure that patients are treated more consistently and aggressively, especially in the face of better methods of anticoagulation.

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Chapter 7
Rate Control in Atrial Fibrillation

Maria Aini and Judd E. Hollander

Rate Versus Rhythm Control

There has been much attention placed on atrial fibrillation, subsequent risk of stroke [1–4], and differing treatment options. Studies consistently showed that patients with atrial fibrillation benefit from long-term anticoagulation to prevent thromboembolic events [5–7]. Therefore, logically there have also been discussions about the importance of rhythm control over rate control in atrial fibrillation to reduce the risk of thromboembolism [8–12]. The rationale supporting rhythm control is less frequent symptoms, improved exercise tolerance, and the discontinuation of anticoagulation. The argument favoring rate control is that there are safer pharmacologic options and cardioprotective advantages if using beta-blockers. There have been multiple trials comparing rhythm control to rate-control therapies in atrial fibrillation (see Table 7.1) [13, 14].

The Atrial Fibrillation Follow-Up Investigation of Rhythm Management (AFFIRM) and Rate Control Versus Electrical Cardioversion for Persistent Atrial Fibrillation trial (RACE) are large, multicenter clinical trials that both show no benefit from aggressive pharmacologic rhythm control compared to rate control. Additionally, both trials concluded that antiarrhythmic drugs are not efficacious in maintaining sinus rhythm and have serious side-effect profiles in comparison to rate-controlling agents [15, 16].

AFFIRM is a randomized multicenter trial comparing rate versus rhythm control with primary end point being overall mortality in 4060 patients 65 years old or older. Although overall, there was not an excess of deaths in the rhythm-control group, the differences approached significance (hazard ratio, 1.15 [95 % confi-

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Table 7.1 Rate versus rhythm trials

Trial	Patients (*n*)	Mean age (years)	Mean follow-up (years)	Inclusion criteria	Primary outcome parameter	Rate-control patients reaching primary outcome	Rhythm-control patients reaching primary outcome	*P* value
PIAF (2000)	252	61.0	1.0	Persistent AF (7–360 days)	Symptomatic improvement	76/125 (60.8 %)	70/127 (55.1 %)	0.32
AFFIRM (2002)	4060	69.7	3.5	Paroxysmal AF or persistent AF	All-cause mortality	310/2027 (25.9 %)	356/2033 (26.7 %)	0.08
RACE (2002)	522	68.0	2.3	Persistent AF or flutter for <1 years	Composite: cardiovascular death, CHF, severe bleeding, pacemaker implantation, thromboembolic events, severe adverse effects of antiarrhythmic drugs	44/256 (17.2 %)	60/266 (22.6 %)	0.11
STAF (2003)	200	66.0	1.6	Persistent AF (>4 weeks and <2 years), LA size >45 mm, CHF NYHA II–IV, LVEF <45 %	Composite: overall mortality, cerebrovascular complications, CPR, embolic events	10/100 (10.0 %)	9/100 (9.0 %)	0.99

HOT CAFÉ (2004)	205	60.8	1.7	First clinically overt persistent AF	Composite: death, thromboembolic events; intracranial/major hemorrhage	1/101 (1.0 %)	4/104 (3.9 %)	>0.71
AF-CHF (2008)	1376	66	3.1	LVEF <35 %m CHFm h/o AF	Cardiovascular death	175/1376 (25 %)	182/1376 (27 %)	0.59
J-RHYTHM (2009)	823	64.7	1.6	Paroxysmal AF	Composite of total mortality, symptomatic cerebral infarction, systemic embolism, major bleeding, hospitalization for heart failure, or physical/ psychological disability	89/405 (22.0 %)	64/418 (15.3 %)	0.012

Adapted from *European Heart Journal* (2010) 31, 2369–2429. doi:10.1093/eurheartj/ehq278

dence interval, 0.99–1.34]). The rhythm-control strategy was associated with a higher risk of death than the rate-control strategy among older patients, those without congestive heart failure, and those with coronary artery disease. Even after adjustment for these prespecified covariates, the trend toward a higher risk of death in the rhythm-control group than in the rate-control group persisted (hazard ratio, 1.18 [95 % confidence interval, 0.99–1.41]). Interestingly, during the course of the study, 594 patients assigned to the rhythm-control group crossed over to the rate-control group (16.7 %, 27.3 %, and 37.5 % after 1, 3, and 5 years, respectively). The inability to maintain sinus rhythm and drug intolerance was the chief reasons for abandonment of a rhythm-control strategy. This study concluded that sinus rhythm maintenance showed no advantage either in the primary end point or in any secondary end points, including quality of life, stroke, or worsening functioning class. The conclusions also suggest that antiarrhythmic drug therapies often fail (see Table 7.2) [15].

RACE was a similar prospective study of 522 patients with persistent atrial fibrillation. The primary end point was a composite of cardiovascular death, heart failure, thromboembolism, bleeding, pacemaker insertion, or severe side effects of antiarrhythmic drugs. After a mean follow-up of 2.3 years, the primary end point occurred in 44 of the 256 rate-control patients (17.2 %) and 60 of the 266 rhythm-control patients (22.6 %). The study concluded no benefit of rhythm control over rate control [16]. Patients enrolled in the RACE study who were New York Heart Association Class II and III heart failure were further analyzed. The primary end point was a composite of cardiovascular mortality, CHF hospitalization, thromboembolic

Table 7.2 AFFIRM results

Event	Overall $N=4060$	Rate-control group $N=2027$	Rhythm-control group $N=2033$	P value
Primary end point (death)	666 (26.3)	310 (25.9)	356 (26.7)	0.08
Secondary end point (composite of death, disabling stroke, disabling anoxic encephalopathy, major bleeding, and cardiac arrest)	861 (32.3)	416 (32.7)	445 (32.0)	0.33
Torsade de pointes	14 (0.5)	2 (0.2)	12 (0.8)	0.007
Sustained ventricular tachycardia	15 (0.6)	9 (0.7)	6 (0.6)	0.44
Cardiac arrest followed by resuscitation				
Ventricular fibrillation or ventricular tachycardia	19 (0.6)	10 (0.7)	9 (0.5)	0.83
Pulseless electrical activity, bradycardia, or other rhythm	10 (0.3)	1 (<0.1)	9 (0.6)	0.01
Central nervous system event total	211 (8.2)	105 (7.4)	106 (8.9)	0.93
Myocardial infarction	140 (5.5)	67 (4.9)	73 (6.1)	0.60
Hospitalization after baseline	2594 (76.6)	1220 (73.0)	1374 (80.1)	<0.001

AFFIRM trial

events, bleeding, pacemaker implantation, and serious drug side effects. Quality of life was also compared. It was concluded that in patients with mild to moderate CHF, rate control is non-inferior to rhythm control. Finally, in another study in patients with atrial fibrillation and congestive heart failure, a routine strategy of rhythm control did not reduce the rate of death from cardiovascular causes, as compared with a rate-control strategy [17].

Thromboembolic events are a major adverse outcome of atrial fibrillation. Even when sinus rhythm is achieved, there is ongoing risk of stroke among patients with a history of atrial fibrillation. This is likely related to asymptomatic, recurrent episodes of paroxysmal atrial fibrillation [13]. Both AFFIRM and RACE trials showed that embolic events occurred irrespective of rate versus rhythm control and that these events occurred in the setting of discontinuation or subtherapeutic anticoagulation. Therefore, the data suggests that longer-term anticoagulation may be needed in all high-risk patients with history of atrial fibrillation whether paroxysmal or permanent (see Table 7.3 [15, 16]).

Furthermore, it is recommended that rhythm-control measures, whether electrical or pharmacologic, should be avoided in patients with atrial fibrillation greater than 48 h duration, those who have a high risk of stroke such as mechanical heart valve, or patients with a history of rheumatic heart disease, recent stroke, or transient ischemic attack. Rhythm control should also be avoided in patients at high risk of dysrhythmias, such as digoxin toxicity or hypokalemia [18].

Though rate control generally seems safer and in most patients may be a better alternative to pharmacologic rhythm control, there is a patient cohort that may benefit from early mechanical rhythm control. This cohort includes younger patients without an underlying treatable cause for atrial fibrillation and who are without advanced heart failure. In this group, aggressive, mechanical rhythm control can prevent progression to permanent atrial fibrillation [19–21].

Table 7.3 ATRIAL fibrillation types

Type of atrial fib	Definition
Paroxysmal	Terminates spontaneously or with intervention within 7 days Episodes may recur with variable frequency
Persistent	Continuous AF that is sustained >7 days
Long-standing persistent	Continuous AF >12 months in duration
Permanent	The term “permanent AF” is used when the patient and clinician make a joint decision to stop further attempts to restore and/or maintain sinus rhythm Acceptance of AF represents a therapeutic attitude on the part of the patient and clinician rather than an inherent pathophysiological attribute of AF Acceptance of AF may change as symptoms, efficacy of therapeutic interventions, and patient and clinician preferences evolve
Nonvalvular	AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair

Adapted from ACC/AHA Guidelines; January et al. [3]

Rate Control

Atrial fibrillation with rapid ventricular response usually approaches rates of 120–160 bpm, assuming the patient has a healthy AV node. Patients with lone atrial fibrillation can tolerate higher ventricular rates than the typical patient with underlying heart failure or other underlying heart disease. When atrial fibrillation with rapid ventricular response occurs, the rapid irregular pattern of ventricular contraction causes decreases in stroke volume and may result in hypotension [2]. Early recognition of rapid atrial fibrillation along with expedient rate control is paramount to hemodynamic stability and avoidance of chronic tachycardia-induced cardiomyopathy. When treating the acute presentation of atrial fibrillation with rapid ventricular response of unknown duration, rate control can frequently be achieved using an AV nodal blocking agent. Non-dihydropyridine calcium channel blockers, beta-blockers, and digoxin all slow conduction through the AV node and can be used. Specifically, non-dihydropyridine calcium channel blocker or beta-blocker can be given via IV route with a response rate of minutes, then continued orally for longer-term rate control [22, 23].

2014 AHA/ACC/HRS Guideline for the Management of Patients with Atrial Fibrillation Class I recommendations include using a beta-blocker or non-dihydropyridine calcium channel antagonist for rate control in paroxysmal, persistent, or permanent atrial fibrillation and use of IV beta-blocker or non-dihydropyridine calcium channel blocker to slow ventricular heart rate in the acute setting in patients without preexcitation [3].

European Society of Cardiology recommendations for acute rate control give Class I recommendations for IV administration of beta-blockers or non-dihydropyridine calcium channel antagonists to slow the ventricular response to atrial fibrillation, exercising caution in patients with hypotension or heart failure. Also, in the acute setting, IV administration of digoxin or amiodarone is recommended to control the heart rate in patients with atrial fibrillation and concomitant heart failure or in the setting of hypotension. Class III evidence supports that beta-blockers, non-dihydropyridine calcium channel antagonists, digoxin, and adenosine are contraindicated when preexcitation is present [13, 14].

Special Considerations

Specifics of treatment choice will depend upon hemodynamic stability and underlying medical conditions and whether the patient has heart failure (see Table 7.4) [3, 18].

Diltiazem is a non-dihydropyridine calcium channel blocker that is easily titratable and has a fast onset of action. Diltiazem is widely considered the agent of choice for treating atrial fibrillation with rapid ventricular response in the emergency department. Verapamil works in a similar mechanism to diltiazem though verapamil has a longer duration of action which could lead to prolonged hypotension. Diltiazem and verapamil have negative ionotropic effects though less so with

Table 7.4 Initial rate-control medication and common dosage

	Common dosage	Notes
First line		
Diltiazem	0.25 mg/kg IV initially, can repeat 0.35 mg/kg IV if needed	Initial oral dose is 60 mg every 6 h
Verapamil	0.075–0.15 mg/kg IV initially then additional 10 mg if no response, then 0.005 mg/kg/min infusion	Increased risk for hypotension than diltiazem
Metoprolol	5 mg IV every 15 min up to three doses	Initial oral dose is 25–50 mg every 12 h
Esmolol	500 mcg/kg IV followed by 50 mcg/kg/min infusion	Short half-life
Second line		
Digoxin	0.25 mg IV, up to 1.5 mg in 24h	Slow onset, good choice with CHF
Amiodarone	5 mg/kg IV over 30 min then 1200 mg over 24 h	Contraindicated in preexcitation, risk of cardioversion

Adapted from ACC/AHA Guidelines; January et al. [3]
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diltiazem. Non-dihydropyridine calcium channel blockers are preferred in patients that have severe chronic obstructive pulmonary disease [3, 22]. Dihydropyridine calcium channel blockers are not effective in rate control.

Metoprolol is a common and effective agent with a fast onset of action. It is especially useful in the setting of myocardial ischemia because of its negative inotropic effect [19]. It is also a good choice in high adrenergic settings like postoperative atrial fibrillation and thyrotoxicosis [3].

Esmolol is a beta-blocker that is particularly useful. Because it has an extremely short half-life, it needs constant titration. This can be helpful if beta-blocker tolerance is questioned.

In the AFFIRM trial, more patients reached adequate rate control in the beta-blocker group (70%) compared to the calcium channel blocker group (54%) [15, 18, 23]. Overall the evidence between the efficacy of beta-blockade and calcium channel blockade is inconclusive, and several other studies show no difference in outcome [24]. The general consensus is that calcium channel blockers are preferred in the setting of active chronic obstructive pulmonary disease and beta-blockers are preferred in the setting of coronary artery disease [18, 22].

Rate Control in Heart Failure

Both calcium channel and beta-blockers will control ventricular rate at rest and exercise. Because metoprolol, esmolol, verapamil, and, to a lesser extent, diltiazem have negative inotropy, they should be used with caution in patients with

decompensated heart failure. However, it is important to note that in a patient with a history of compensated heart failure, calcium channel and beta-blockade can improve oxygen delivery, ventricular filling, and therefore cardiac output through rapid rate control [3].

Digoxin will control the heart rate mostly at rest but not during exercise. Once considered first-line treatment for rapid atrial fibrillation, it has a slower onset of action of 15–30 min with peak effect in 1–5 h. It is not as effective as beta or calcium channel blockade and thus is considered second-line therapy [22, 25, 26]. However, because of its positive inotropic effect, it may be useful in the setting of decompensated systolic heart failure where it may reduce ventricular rate and improve contractility. Digoxin can also be used as an adjunct to first-line therapies, but because of associated toxicities, levels must be monitored periodically [25, 26]. Digoxin has a role for rate control for atrial fibrillation in sedentary patients that have reduced ejection fraction [3].

Amiodarone is interesting because it can be used for both rate and rhythm control. In the acute setting, especially in setting of CHF with hypotension, amiodarone can be used for its rate-control effect since its cardioversion properties usually take longer to take effect (4–6 h after IV dosing) [3]. Because of amiodarone's antidysrhythmic effect, it's advisable to anticoagulate in this setting to mitigate the risk of an iatrogenic embolic event if conversion to sinus rhythm occurs [18].

Lenient Versus Strict Rate Control

Rate-control goals are the subject of controversy. According to the RACE II trial, lenient rate control was defined as a resting heart rate less than 110 bpm, and strict control was defined as a resting heart rate less than 80 bpm in patients with permanent atrial fibrillation. The trial evaluated primary outcomes of cardiovascular death and related hospitalization and concluded the primary outcomes were similar between the two groups. Of importance, the lenient rate-control group had fewer outpatient visits to achieve goal rate control, were on fewer rate-control agents, had fewer side effects, and had lower costs (see Table 7.5) [27].

Table 7.5 Race II outcomes

Outcome	Lenient rate control $N=311$	Strict rate control $N=303$	Hazard ratio (90 % CI)
Composite primary outcome	38 (12.9)	43 (14.9)	0.84 (0.58–1.21)
Death from cardiovascular cause	9 (2.9)	11 (3.9)	0.79 (0.38–1.65)
Heart failure	11 (3.8)	11 (4.1)	0.97 (0.48–1.96)
Stroke	4 (1.6)	11 (3.9)	0.35 (0.13–0.92)
Bleeding	15 (5.3)	13 (4.5)	1.12 (0.60–2.08)

Aberrancy and Preexcitation

An EKG is the mainstay of atrial fibrillation diagnosis. In settings of atrial fibrillation with a wide complex QRS, aberrancy versus preexcitation should be considered. If the cause of the wide QRS complex is an underlying bundle branch block, the ventricular conduction rate often remains under 180 bpm. In the setting of atrial fibrillation where the ventricular rate approaches 200 bpm or above, an accessory pathway must be considered. In this scenario, AV nodal blockers are contraindicated, and IV procainamide is the pharmacologic treatment of choice if patient is hemodynamically stable. Otherwise prompt cardioversion is warranted [3]. AV nodal blockade is contraindicated because blocking the AV node will preferentially send atrial impulses through the accessory pathway where impulses travel fast without a refractory period, and this phenomenon can quickly degenerate into ventricular fibrillation [3].

Unstable Atrial Fibrillation

Hemodynamic instability is an indication for urgent electric synchronized cardioversion. Patients require procedural sedation then typically 100 J of biphasic current initially followed by 150–200 J of current delivery if initial is unsuccessful. Electric cardioversion has shown to have a higher success rate than pharmacologic cardioversion [2–4].

Cocaine-Induced Atrial Fibrillation

Cocaine is an exogenous cause of various arrhythmias including atrial fibrillation. Chronic cocaine use is also associated with myocardial ischemia so treating atrial fibrillation in this setting can be complex [28, 29]. The unopposed α-adrenergic effect of beta-blockers in cocaine toxicity leads to worsening coronary vasoconstriction and increased blood pressure; therefore, the use of beta-blockers for the treatment of cocaine toxicity should be avoided when ischemia is suspected. In a patient presenting in rapid atrial fibrillation with concern for cocaine-induced myocardial ischemia, beta-blockade should be avoided and treatment should be supportive [30].

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Chapter 8
Diagnostic Testing in the Emergency Department of Atrial Fibrillation

Adriana Thomas and Zubaid Rafique

History

As with all medical encounters in the emergency department (ED), a thorough yet focused history is paramount in a patient's workup. When considering atrial fibrillation (AF), aspects that should be considered are age (as prevalence increases significantly with age [1]), symptoms, precipitating factors, pattern of the arrhythmia, response to therapy received in the past, and medical, family, and social history.

AF has many associated symptoms and some patients may even be asymptomatic. Typical symptoms include palpitations, shortness of breath, tachycardia, fatigue, weakness, dizziness, angina, and pre-syncope. Infrequently, it can present with syncope or an embolic event or right-sided heart failure. Characterizing the pattern of the arrhythmia (i.e., paroxysmal, persistent, long-standing persistent, or permanent) is important as management is based on the clinical type of AF. As for precipitating factors, it is important to inquire about life events or substance abuse history leading to AF, since alcohol, emotional stress, and exercise are some of the common triggers [2–4]. Past medical history is important as various medical conditions carry an increased risk of AF. These include chronic kidney disease (CKD) [15, 16], cerebral vascular accident (CVA) [9], diabetes mellitus (DM) [17–20], hypertension (HTN) [8], and chronic obstructive pulmonary disease (COPD) [5]. You may also elicit reversible causes such as thyroid disease [10–13] and alcohol abuse.

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Table 8.1 Important historical aspects to obtain from patients presenting to an emergency department in AF

History
Age [1]
Symptoms
Precipitating factors [2–4]
Pattern of arrhythmia
Response to prior treatment
Past medical history
Family history
Social history

And lastly, obtaining a family history and social history can elicit other underlying risks. Table 8.1 summarizes the historical elements important in the workup of AF.

Physical Exam

Physical exam findings consistent with AF include an irregularly irregular rhythm upon auscultation of the heart. This same rhythm can be appreciated on distal pulses along with an irregular jugular venous pulsation. Valvular heart disease causing stenosis or regurgitation has a significant association with the development of AF; hence auscultation for cardiac murmurs is important. Since AF can lead to heart failure, physical exam should evaluate for evidence of heart failure as well [22].

Diagnosis of Atrial Fibrillation on ECG

The diagnosis of AF is typically confirmed with a 12-lead electrocardiogram (ECG). The ECG is characterized by the absence of discrete P waves and an irregularly irregular ventricular rhythm. You may also find fibrillatory waves (f waves) and usually a narrow QRS complex.

Atrial Activity in Atrial Fibrillation

In AF there is no organized atrial activity. Numerous pacemaker cells within the atria generate electrical impulses at the same time. This results in rapid and irregular f waves of varying morphology and amplitude and a lack of discrete P waves. They are best seen in leads V1, V2, V3, and aVF and are typically present at a rate of 350–600 beats per minute (bpm) [23]. “Coarse” AF occurs when f waves, typically in lead V1, have large amplitudes. This is most commonly seen in early-onset AF and can make it difficult to differentiate between AF, atrial flutter, and multifocal atrial tachycardia (MAT).

Ventricular Response in Atrial Fibrillation

Ventricular response can be as high as 200 bpm. Higher ventricular rates (>200 bpm) occur by means of accessory pathways or a shortened refractory time of the AV node (due to increased catecholamine circulation or a sympathetic response) [21].

In patients with a preexcitation syndrome, ventricular rates can reach 280–300 bpm. This occurs because atrial impulses bypass the AV node and conduct via an accessory or intranodal pathway. Intranodal pathways can have very short refractory periods allowing for more frequent conduction [6]. On the other hand, accessory pathways do not have a refractory period and can conduct faster than the AV node. In cases where accessory pathways are used, the His-Purkinje system is bypassed as well, and ventricular depolarization is lengthened resulting in wide (aberrant) QRS complexes, which can be mistaken for ventricular tachycardia (but still with an irregularly irregular rhythm). A defining feature of AF with preexcitation is the relationship between the heart rate and the width of the QRS: the faster the rate, the wider the QRS [5].

In patients who are said to have "regularization of atrial fibrillation," a regular ventricular rate is seen. This is a result of complete AV nodal blockade and a resulting junctional or ventricular escape rhythm.

The QRS Complex in Atrial Fibrillation

The QRS complex is typically narrow in AF as the His-Purkinje system is usually intact, and thus activation of the ventricles is not altered once atrial conduction has made its way through the AV node. However, the width of the QRS complex is altered in the presence of the following underlying conditions:

- A bundle branch block
- A functional block in the His-Purkinje system (typically rate related)
- An atrioventricular (AV) bypass tract capable of antegrade conduction

All of these conditions lead to aberrant ventricular conduction (a wide QRS), and the resulting rhythm is termed "AF with aberrancy" (Fig. 8.1).

Indicated Blood Work and Imaging

Aside from an ECG, various blood tests are indicated in patients presenting in AF. This is depicted in Table 8.2 below.

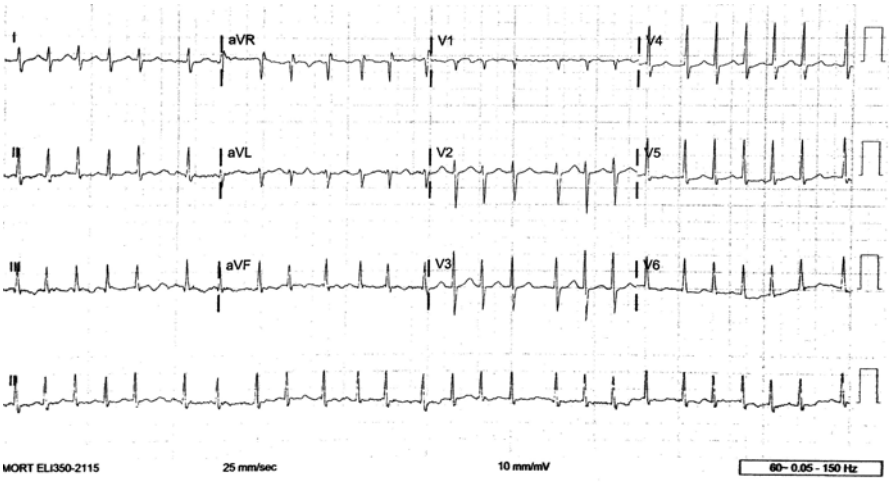

Fig. 8.1 ECG demonstrating AF with RVR, defined by the lack of discrete P waves and irregularly irregular rhythm and a ventricular rate of ~160 bpm

Table 8.2 Indicated workup for patients presenting to an emergency department with AF

Blood work/imaging	Rationale
Serum creatinine, urine protein	CKD is an independent risk factor for AF [15, 16]
CBC	Anemia in combination with CKD has an even higher associated risk of AF [12, 14]
Thyroid stimulating hormone (TSH) and free T4	TSH level-dependent relationship exists between the degree of thyroid dysfunction and the risk of new-onset AF. Even subclinical hypothyroidism can be associated with an increased risk of AF. However, both subclinical and overt hypothyroidism are associated with a lower risk of AF when compared to hyperthyroidism [10–13]
Blood glucose	Diabetes is associated with higher risk of developing AF, with a higher risk in patients with longer duration of untreated diabetes and worse glycemic control [17–20]
B-type natriuretic peptide (BNP) and chest radiograph (CXR)	Heart failure (HF) and AF often occur together; the prevalence of AF varies with the severity of HF [22]
Troponin	Although the incidence of AF and acute coronary artery disease is low, it is still recommended that patients be screened for it as CAD has significant implications when choosing antiarrhythmic therapy [7]

Echocardiogram

The use of ultrasound in the emergency department is quickly becoming mainstay.

Its use is highly recommended in aiding with the management of AF. Although, it has limited sensitivity, a TTE can be used to detect a left atrial or left atrial appendage thrombus. Detection of thrombus is important for patients in which electrical cardioversion is being considered. It can also be used to evaluate chamber size, valvular pathology, ventricular function, and pericardial disease.

Transesophageal echocardiography is more sensitive in detecting atrial thrombi and thus more helpful in the management of AF [24, 25].

Summary

Workup of AF, much like any condition being evaluated in an ED, begins with a thorough yet focused history and physical. This should focus on obtaining the following:

- Symptoms
- Circumstances surrounding onset
- Past medical, family, and social histories
- Cardiac exam
- Evidence of heart failure on exam and history

The diagnosis of AF is confirmed by an ECG demonstrating the absence of discrete P waves and an irregularly irregular rhythm.

Ventricular response is usually up to 200 bpm; however, the presence of an accessory pathway can allow much higher rates.

Recommended workup of AF includes CBC, serum creatinine, urine protein, Thyroid stimulating hormone (TSH) and free T4, blood glucose, BNP, troponin, and a chest radiograph.

Echocardiography is helpful in detecting atrial thrombus and thus dictates the need for anticoagulation before cardioversion.

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Chapter 9
Anticoagulation for Atrial Fibrillation in the Emergency Department or Observation Unit

Deepak L. Bhatt and Andrew E. Noll

Introduction

Patients with atrial fibrillation (AF) are at an increased risk of thromboembolic stroke, and anticoagulation to reduce the risk of stroke and other thromboembolic events has become a standard part of the management of this arrhythmia. The benefit of anticoagulation must be weighed against the increased risk of bleeding, and this is an important part of the initial management of most patients with newly diagnosed AF. Historically, heparin followed by the vitamin K antagonists such as warfarin was the treatment of choice, but recently a number of novel oral anticoagulants, or non-vitamin K antagonist oral anticoagulants (NOACs), have been developed which offer simplified dosing and perhaps some benefit over warfarin. The decision to start anticoagulation in the emergency department (ED) or observation unit (OU) requires thoughtful consideration of all of these factors [1].

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Pathophysiology of Thromboembolism in Atrial Fibrillation

The precise mechanisms of thrombus formation in AF are not known, but it is hypothesized that stasis of blood flow, hypercoagulability, and damaged endothelium may all contribute. Atrial fibrillation is characterized by the disorganized and erratic contraction and relaxation of the myocytes of both atria. As a result the atria do not contract in an organized and hemodynamically efficient manner leading to stasis of blood and thrombus formation, most often in the left atrial appendage. A left atrial thrombus may then dislodge and embolize to the cerebral circulation causing an ischemic stroke, or, less commonly, to the systemic circulation causing infarction of the limbs or other organs.

Anticoagulation to Prevent Thromboembolism

The 2014 American Heart Association (AHA)/American College of Cardiology (ACC) guidelines for the management of nonvalvular atrial fibrillation (NVAF) recommend long-term anticoagulation with warfarin (class 1A recommendation) or a NOAC (class 1B recommendation) in patients with history of prior stroke/transient ischemic attack (TIA) or a CHA_2DS_2-VASc score ≥ 2 [2]. They also recommend that antithrombotic therapy be based on "shared decision making, discussion of the risks of stroke and bleeding, and patient preferences." Starting anticoagulation for stroke reduction in AF is not an emergency, as the day-to-day risk of stroke is very small, and the decision to start anticoagulation should be shared between the patient, ED/OU physician, and outpatient physicians. An exception to this is the patient who will undergo electrical or pharmacologic cardioversion; in this case anticoagulation should be started prior to the cardioversion to reduce the risk of stroke when sinus rhythm is restored [3].

This chapter primarily concerns patients with nonvalvular atrial fibrillation. Valvular AF refers to patients with prosthetic heart valves, mitral stenosis, or any severe valvular disease likely to require imminent repair. These patients were excluded from most major trials of AF, and distinct guidelines have been published for the management of prosthetic heart valves and mitral stenosis [4, 5]. Many patients with valvular AF do require anticoagulation, and, for the time being, heparin or warfarin should be used exclusively. The NOACs should be avoided in these patients, as only dabigatran has been evaluated for this purpose, and it did not adequately protect against stroke when compared with warfarin [6].

The decision to start an anticoagulant for a patient with new NVAF in the OU can be safe and effective if the following four factors are considered:

1. Risk of thromboembolism/stroke
2. Risk of bleeding
3. Choice of anticoagulant
4. Practical considerations

Risk of Thromboembolism/Stroke

Certain patients with NVAF are at higher risk of stroke than others, and stratification of patient risk has proven useful in guiding the decision to start anticoagulation. The preferred method is the CHA_2DS_2-VASc score, a recently developed model that has superseded the better-known $CHADS_2$ score, as it is more predictive and better able to stratify risk among patients with lower scores (Tables 9.1 and 9.2) [7, 8].

Comparing the two scoring systems, it can be seen that the CHA_2DS_2-VASc better distinguishes the risk among patients with scores ≤2 (0 correlates with 0 % risk, 1 with 1.3 %, 2 with 2.2 %), whereas a $CHADS_2$ score of 0 connotes a 1.9 % risk. Other notable features of the CHA_2DS_2-VASc score include a score of 2 or greater for all patients who have a history of stroke or TIA and for all patients aged ≥75 years; in both of these groups, anticoagulation is strongly recommended, but it must be kept in mind that both of these groups are also at increased risk of bleeding.

Table 9.1 The $CHADS_2$ and CHA_2DS_2-VASc scores

$CHADS_2$	Risk factor	CHA_2DS_2-VASc
1	Congestive HF	1
1	Hypertension	1
1	Age ⩾75	2
1	Diabetes mellitus	1
2	History of stroke/ TIA	2
	Vascular disease[a]	1
	Age 65–74	1
	Sex (female)	1
6	*Maximum score*	*9*

HF heart failure, *TIA* transient ischemic attack
[a]Myocardial infarction, peripheral arterial disease, or aortic plaque

Table 9.2 Adjusted risk of stroke/thromboembolism stratified by $CHADS_2$ and CHA_2DS_2-VASc scores

$CHADS_2$ score	Annual risk of stroke (%) [9]	CHA_2DS_2-VASc score	Annual risk of thromboembolism (%) [10]
0	1.9	0	0.0
1	2.8	1	1.3
2	4.0	2	2.2
3	5.9	3	3.2
4	8.5	4	4.0
5	12.5	5	6.7
6	18.2	6	9.8
		7	9.6
		8	6.7
		9	15.2

Anticoagulation is strongly recommended in patients with CHA_2DS_2-VASc $\geqslant 2$, as in these patients, the benefit of anticoagulation outweighs the risk of bleeding and intracranial hemorrhage; this holds true for both warfarin and the NOACs [11–13]. Further support for this recommendation comes from meta-analyses of the major trials comparing warfarin with placebo which have demonstrated that warfarin significantly reduces the risk of all stroke, disabling stroke, and all-cause mortality [14]. In general, oral anticoagulation reduces the risk of ischemic stroke by about two thirds; thus the absolute risk reduction depends on the patient's baseline risk [15].

Whether anticoagulation benefits patients with a CHA_2DS_2-VASc score of 1 is less clear, and there is variability in practice. The AHA/ACC does not recommend for or against anticoagulation in this group, and thus these patients require careful individualized decision-making. The true risk of stroke in these patients is uncertain; a number of recent retrospective cohorts have cited estimates of annual risk varying from as low as <1 % to as high as 3.5 % [16, 17]. This discrepancy seems to be in part due to the definition of "stroke" used and to variability in the actual risk imparted by the various risk factors that can give a patient a CHA_2DS_2-VASc score of 1, and prospective studies are needed in order to better define these patients' true risk. In patients with a CHA_2DS_2-VASc score of 1, consideration of the patient's risk of bleeding is imperative, and it may be advisable to defer the initiation of anticoagulation to the outpatient setting, where a patient's long-term physicians can more thoroughly discuss the matter with the patient.

The risk of stroke with a CHA_2DS_2-VASc score of 0 is minimal, and there is a general consensus that anticoagulation does not offer a meaningful benefit. These patients should be followed closely for the development of any conditions that would increase their CHA_2DS_2-VASc score and thus prompt consideration of anticoagulation.

Risk of Bleeding

The inevitable consequence of anticoagulation with warfarin and the NOACs is an increased risk of bleeding. A patient's individual risk of hemorrhage should always be considered prior to starting an anticoagulant.

The average rate of major hemorrhage in patients taking warfarin has been estimated as 1–3 % per year in the major clinical trials; community-based cohorts have shown a slightly higher rate of 3–4 % with a significantly increased risk during the first 30 days of therapy (as high as 11.8 % per person-year) [18]. Intracranial hemorrhage (ICH) is the most feared complication of anticoagulation therapy. Warfarin increases the risk of ICH by two to five times, and higher degrees of anticoagulation are associated with increased rates of hemorrhage and death [19, 20]. Estimates of the incidence of ICH vary, but one large community-based cohort found the incidence of ICH to be 0.23 per 100 person-years among those patients not taking

warfarin and 0.46 per 100 person-years among those taking warfarin [21]. The major NOAC trials have all demonstrated a reduced rate of ICH with NOACs as compared with warfarin, with rates ranging from 0.3 to 0.8 % per year. Thus, while ICH is a serious and often deadly or disabling consequence of anticoagulation, it occurs at a significantly lower rate than ischemic stroke in patients with CHA_2DS_2-VASc $\geqslant 2$ and does not negate the benefit of anticoagulation in these patients.

The rates of bleeding correlate with a number of risk factors, including increasing age, history of gastrointestinal bleeding, concurrent use of antiplatelet agents, uncontrolled hypertension, chronic kidney disease, chronic liver disease, history of uncontrolled international normalized ratio (INR), and increasing $CHADS_2$ score, among others [22]. A number of risk assessments such as the HAS-BLED and RIETE scores have been developed with the intent of predicting an individual patient's risk of hemorrhage with anticoagulation, but they unfortunately have poor predictive value and have not been prospectively validated [23]. Therefore, none of them are ideal for routine clinical use. When estimating a patient's risk of bleeding with anticoagulation, clinical judgment and consideration of the risk factors above are the standard.

Choosing an Anticoagulant

For decades warfarin, with or without a heparin "bridge," has been the preferred oral anticoagulant for thromboprophylaxis in patients with atrial fibrillation, but since 2010 several direct-acting oral anticoagulants (dabigatran, rivaroxaban, apixaban, and edoxaban) have been developed and approved by the US Food and Drug Administration (FDA). Patients and providers now face a choice and must weigh the risks and benefits of the available drugs when choosing an anticoagulant (Table 9.3).

Warfarin

Warfarin is a vitamin K antagonist that leads to production of hemostatically inactive forms of the clotting factors II, VII, IX, and X. It has been in regular use since the 1950s and its efficacy is well established [14]. Warfarin has a narrow therapeutic window and must be closely monitored to ensure that the patient is neither over- nor under-anticoagulated. This requires regular laboratory INR checks, every few days upon initiating warfarin and at least every few weeks thereafter. The goal INR is typically 2–3. Most health centers have "Coumadin clinics" with staff dedicated to monitoring and titrating patients' warfarin, but, despite this, the average time spent in the therapeutic range for most patients is only 50–66 % [24, 25]. This puts patients at increased risk of both treatment failure and bleeding, and warfarin is among the top ten medications associated with serious adverse events as monitored by the FDA. Bleeding complications

Table 9.3 Summary of anticoagulants for nonvalvular atrial fibrillation

Drug	Mechanism of action	Dosing	Renal dosing	Monitoring/ safety	Comments
Warfarin (Coumadin)	Vitamin K antagonist; inhibits production of factors II, VII, IX, and X	2.5–5 mg daily, use lower dose if liver disease, CHF, elderly, poor nutrition, high risk of bleeding Adjust dose to meet INR goal	No adjustment necessary; increased risk of bleeding in patients with advanced CKD Preferred anticoagulant in patients with severe CKD or ESRD on dialysis	Check INR every 3–4 days initially; may reduce checks to once monthly once stable regimen is achieved	Patient must be able to adhere to frequent INR checks Patient should consume a steady amount of vitamin K in the diet
Dabigatran (Pradaxa)	Direct thrombin inhibitor	150 mg twice daily	CrCl 30–50 ml/ min: reduce to 75 mg twice daily only if concurrently taking ketoconazole or dronedarone CrCl <30 ml/ min: avoid use	No routine monitoring of anticoagulation necessary Monitor renal function annually	May increase the risk of major GI bleeding compared with warfarin Avoid in patients >80 years of age Avoid in patients taking Pgp inducers or inhibitors
Rivaroxaban (Xarelto)	Direct factor Xa inhibitor	20 mg once daily with evening meal	CrCl 30–50 ml/ min: Reduce dose to 15 mg daily CrCl <30 ml/ min: avoid use	No routine monitoring of anticoagulation necessary Monitor renal function annually	May reduce ICH and fatal bleeding compared with warfarin Avoid in patients concurrently taking medications that are Pgp and strong CYP3A4 inhibitors

(continued)

Table 9.3 (continued)

Drug	Mechanism of action	Dosing	Renal dosing	Monitoring/ safety	Comments
Apixaban (Eliquis)	Direct factor Xa inhibitor	5 mg twice daily, unless patient has any two of the following: age ≥80 years, weight ≤60 kg, creatinine ≥1.5 mg/dl, then decrease to 2.5 mg twice daily	SCr 1.5–2.5 mg/dl: reduce dose to 2.5 mg twice daily if age ≥80 years or weight ≤60 kg SCr >2.5 mg/dl or CrCl ≤25 ml/min: avoid use	No routine monitoring of anticoagulation necessary Monitor renal function annually	May reduce stroke/SEE, major bleeding, and death compared with warfarin In patients taking strong inhibitors of CYP3A4 and Pgp concomitantly, reduce the 5 mg twice-daily dose by 50%, and if already on 2.5 mg twice-daily dose, then avoid coadministration with the strong dual inhibitors
Edoxaban (Savaysa)	Direct factor Xa inhibitor	60 mg once daily in patients with CrCl 51–95 ml/min. Do not use in patients with normal renal function	CrCl >95 ml/min: Avoid use CrCl 51-95 ml/min: no adjustment necessary CrCl 15-50 ml/min: reduce dose to 30 mg daily CrCl <15 ml/min: avoid use	No routine monitoring of anticoagulation necessary Monitor renal function annually	May reduce major bleeding and ICH compared with warfarin.

CHF congestive heart failure, *INR* international normalized ratio, *CKD* chronic kidney disease, *CrCl* creatinine clearance, *GI* gastrointestinal, *Pgp* P-glycoprotein, *ICH* intracranial hemorrhage, *SCr* serum creatinine, *SEE* systemic embolic event

associated with warfarin account for about 29,000 emergency department visits each year [26].

Warfarin is subject to many drug-drug interactions, most importantly antibiotics and a number of drugs that affect warfarin metabolism. All concurrent medications and any new medications should be carefully reviewed for interactions in patients taking warfarin. Finally, the vitamin K content of the diet significantly impacts the effect of warfarin, and all patients should be counseled about maintaining a steady intake of vitamin K.

Despite these limitations, many patients do well with warfarin and maintain stable INRs with monthly laboratory visits. INR correlates well with the anticoagulant effect of warfarin, allowing clinicians both to monitor adherence and to assess a patient's risk of thrombosis or bleeding. This is especially useful in cases of treatment failure or significant bleeding events. Reversal of warfarin's anticoagulant effect is sometimes desired in cases of bleeding or prior to invasive procedures, and protocols for reversal with vitamin K, plasma transfusion, and prothrombin complex concentrates are well established.

Heparin and Low-Molecular-Weight Heparins

The anticoagulant effect of warfarin is not immediate, and three to seven days' administration is often required for the INR to reach the therapeutic range. For those patients at high risk of thromboembolism and low risk of bleeding, it may be reasonable to provide a heparin or low-molecular-weight heparin (LMWH) "bridge" concurrently with warfarin until the INR is therapeutic. In patients without a prior history of thromboembolism, the daily risk of stroke is very low, and thus a heparin or LMWH bridge is often not prescribed. Patients at higher risk of stroke, such as those with a prior history of thromboembolism, a mechanical valve, or mitral stenosis, are generally bridged with heparin or LMWH as long as their risk of hemorrhage is not too great. This strategy has not been prospectively studied to any great extent, and thus, when deciding whether to bridge with a parenteral anticoagulant, each individual's likelihood of benefit should be weighed against his or her risk of hemorrhage [2]. Low-molecular-weight heparin is administered subcutaneously and is often preferred to intravenous unfractionated heparin, as it does not generally require laboratory monitoring and may be administered by the patient at home. However, in patients with significant renal dysfunction or extreme obesity, LMWH dosing can be more challenging than unfractionated heparin. Unfractionated heparin and LMWH have demonstrated equivalence in achieving short-term anticoagulation in patients with atrial fibrillation [27]. These considerations apply not only when initiating warfarin but also when an oral anticoagulant is being held in preparation for an invasive procedure, though recent data have called routine use of bridging into question. Bridging is not required when starting NOACs, as their onset of action is a matter of a few hours.

Dabigatran

Dabigatran is a direct thrombin inhibitor and was the first non-vitamin K oral anticoagulant approved for the prevention of thromboembolism in NVAF. The RE-LY trial compared dabigatran to warfarin and showed that the 150 mg twice-daily dose of dabigatran reduced the primary outcome of stroke or systemic embolism by one third; the rates of intracranial hemorrhage and fatal bleeding were also reduced, but there was an increased rate of gastrointestinal bleeding with dabigatran (1.6 % per year versus 1.0 % per year with warfarin) [28]. There was no significant difference in mortality between the two groups. This trial and a subsequent meta-analysis have demonstrated that there may be a slightly higher risk of myocardial infarction (MI) with dabigatran as compared with warfarin, and this should be taken into consideration when considering its use [29]. Certain subgroups are at an increased risk of bleeding complications, most notably African-Americans and those with chronic kidney disease [30].

Dabigatran is hepatically metabolized and renally cleared, and patients with severe liver disease or creatinine clearance (CrCl) <30 ml/min were excluded from the RE-LY trial. A reduced dose, 75 mg twice daily, has been approved for use in patients with CrCl 15–30 ml/min, but this dose has not been prospectively tested, and warfarin is probably a safer anticoagulant in this population [31]. Dabigatran should be avoided in all patients with CrCl <15 ml/min, in those on dialysis, and in those with advanced liver disease. Its use should also be avoided in the presence of P-glycoprotein (Pgp) inducers (e.g., rifampin, phenytoin, carbamazepine, St. John's wort) and inhibitors (e.g., verapamil, ketoconazole, amiodarone, dronedarone, quinidine, clarithromycin).

A distinct advantage of dabigatran and all the NOACs is that no routine laboratory monitoring is required; the unfortunate corollary is that there is no widely available laboratory test that measures the anticoagulant effect of dabigatran. Most reliable is the dilute thrombin time, but this assay is not commonly available and varies between laboratories [32]. A normal activated partial thromboplastin time (aPTT) rules out a clinically significant effect of dabigatran, but the aPTT does not linearly correlate with dabigatran levels.

Rivaroxaban

Rivaroxaban was the first factor Xa (FXa) inhibitor approved to prevent stroke and embolism in atrial fibrillation. The ROCKET-AF trial showed that rivaroxaban 20 mg once daily was noninferior to warfarin in the prevention of stroke and systemic embolism [33]. There was a reduction in the rate of intracranial hemorrhage and fatal bleeding and no change in the rates of clinically relevant bleeding, MI, or death.

Rivaroxaban is metabolized by the liver and principally excreted by the kidneys. It should be avoided in patients with severe liver or end-stage kidney disease. A

reduced dose of 15 mg once daily has been approved for patients with CrCl 15–50 ml/min, but caution should be used in patients with CrCl <30 ml/min, as rivaroxaban has not been prospectively evaluated in this group. Rivaroxaban is a substrate of both CYP3A4 and Pgp and should be avoided when a patient's medical regimen includes medications that inhibit both of these concurrently. There is no laboratory test approved to monitor the anticoagulant effect of rivaroxaban, although a normal INR can rule out clinically significant levels of the drug [32]. Assays for anti-Xa activity can correlate with rivaroxaban activity when the assay is calibrated to the drug itself.

Apixaban

Apixaban is an FXa inhibitor that demonstrated superior efficacy and safety when compared with warfarin in the ARISTOTLE trial [34]. Apixaban achieved a reduction in stroke or systemic embolism, major bleeding, intracranial hemorrhage, and death. The standard dose of 5 mg twice daily should be reduced to 2.5 mg twice daily in patients with two or more of the following factors: age ≥80 years, weight ≤60 kg, or a serum creatinine ≥1.5 mg/dl. Apixaban should be avoided in patients with creatinine >2.5 mg/dl or with CrCl <25 ml/min, as these patients were excluded from the major trials of the drug. The FDA has recently approved apixaban for use in patients with end-stage renal disease on stable hemodialysis, but the 2014 ACC/AHA guidelines do not support this recommendation due to a lack of experience with the drug in these patients. In patients taking concomitant strong inhibitors of the CYP3A4 and Pgp systems, the 5 mg twice-daily dose should be decreased by 50 %, and the 2.5 mg twice-daily dose should be avoided in favor of warfarin. Apixaban activity can be determined by an anti-Xa activity assay calibrated to apixaban, but INR and PTT do not reliably reflect apixaban activity.

Edoxaban

Edoxaban is the most recently approved FXa inhibitor for thromboprophylaxis in atrial fibrillation. It was compared with warfarin in the ENGAGE AF-TIMI 48 trial and demonstrated similar efficacy to warfarin with fewer major bleeding events, intracranial hemorrhages, and cardiovascular deaths [35]. It should be used at a 60 mg once-daily dose in patients with CrCl 51–95 ml/min and at 30 mg once daily for CrCl 15–50 ml/min. Note that, per the FDA, it should be avoided in patients with normal renal function (CrCL >95 ml/min), as these patients experienced an increased rate of stroke and thromboembolism in the trial. As with all NOACs, it should not be used in patients with severe liver dysfunction. Edoxaban is best measured by anti-Xa activity calibrated to either heparin or edoxaban [36] (Table 9.3).

Antiplatelet Agents

Aspirin and clopidogrel have been studied as alternatives to anticoagulants for the prevention of stroke in atrial fibrillation, but they have poor efficacy with a similar or greater risk of bleeding [15]. Antiplatelet agents should not be considered adequate prophylaxis against stroke and systemic embolism in patients with atrial fibrillation. The addition of an anticoagulant to antiplatelet therapy in patients with coronary artery disease and atrial fibrillation is known to increase the risk of bleeding, and a benefit to warfarin plus aspirin over warfarin alone has not been demonstrated [37]. Patients with atrial fibrillation who have undergone recent percutaneous coronary intervention are at an even higher risk of bleeding if warfarin is added to aspirin and a $P2Y_{12}$ inhibitor such as clopidogrel ("triple therapy"); in these cases it may be preferable to omit aspirin from the regimen to reduce the risk of bleeding [38]. Overall, there is little evidence to guide decision-making when patients require both an anticoagulant and antiplatelet medications, and these cases should be considered on a patient-by-patient basis in consultation with the patient's longitudinal physicians [39, 40].

Practical Considerations

Because anticoagulation for the prevention of thromboembolism in AF is generally not urgent, it is prudent to carefully consider the practical and long-term implications of starting a patient on anticoagulation in the ED or OU.

Decision to Anticoagulate

For some patients the decision to start anticoagulation will not be straightforward, and in these cases consulting with the outpatient physicians prior to starting an anticoagulant can be helpful. Such patients include those at marginal risk of thromboembolism (CHA_2DS_2-VASc of 1), those at high risk of bleeding, and those on interacting medications, especially dual antiplatelet therapy (aspirin plus a $P2Y_{12}$ inhibitor) which significantly increases the risk of bleeding with an oral anticoagulant [41]. Some patients may prefer to discuss starting an anticoagulant with their primary care physician or cardiologist, and this is a reasonable option as long as there is no evidence of an intracardiac thrombus on echocardiography and no plan for cardioversion.

Choice of Anticoagulant

The recent development of the NOACs as alternatives to warfarin therapy demands that the physician and patient be informed and thoughtful when choosing an oral anticoagulant. Many physicians are comfortable with warfarin given its many

years on the market, the ability to monitor levels by INR, and the widespread availability of reversal agents. Drawbacks include problematic drug-drug and drug-food interactions and the need for frequent laboratory visits to monitor the INR. The NOACs offer fewer drug interactions, no dietary restrictions, and no need for routine laboratory monitoring. Furthermore, in some cases they appear to have superior safety profiles when compared with warfarin. These drugs are, however, more expensive than warfarin, and some clinicians are hesitant to use them due to the limited amount of long-term safety data and the lack of reversal agents in case of emergencies. Patients prescribed NOACs should also be aware that missing even a single dose results in loss of anticoagulant effect and puts them at increased risk of stroke. Finally, it is generally not necessary to "bridge" a patient with a parenteral anticoagulant such as heparin or LMWH when initiating a NOAC for NVAF. Bridging may be considered when starting warfarin in a patient at high risk of stroke, with evidence of active thrombosis, or with valvular AF, but is generally not necessary in nonvalvular AF with NOACs which take effect within a couple of hours.

Patient Follow-Up

If a patient is started on oral anticoagulation for new AF in the ED/OU, appropriate outpatient follow-up must be arranged. For patients on warfarin, this includes an INR check 3–4 days after initiation and a visit with the provider who will monitor the INR. Any patient prescribed a NOAC should have an outpatient appointment to monitor for signs of bleeding, but no routine laboratory monitoring is necessary.

Conclusion

Atrial fibrillation is an increasingly prevalent and costly disease. Hospitalizations for AF in the United States increased by 23 % between 2000 and 2010, and three quarters of the $6.7 billion spent on AF care annually is attributed to these hospitalizations [42]. The thoughtful and timely initiation of anticoagulation in the ED or OU could have a substantial impact on these figures by avoiding unnecessary admissions and increasing adherence to the anticoagulation guidelines for patients with atrial fibrillation.

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Chapter 10
Cardioversion and Acute Atrial Fibrillation Management

Chad E. Darling, Christian G. Klaucke, and David D. McManus

Introduction

There are more than six million people with known atrial fibrillation (AF) in the United States, and approximately 1.6 million new cases occur annually [1, 2]. Many such patients present to the emergency department (ED) and require acute care for the management of symptoms or an unfavorable hemodynamic profile. Traditionally patients with new-onset AF who present to the ED have been managed with a rate control strategy and admitted. Attempts to cardiovert appropriately selected patients to sinus rhythm is a patient-centered approach that has been shown to be a safe and cost-effective strategy that can negate the need for hospital admission for many lower-risk patients [3–6]. The decision to pursue an early rhythm control strategy depends on a variety of factors, including patient stability, age, precipitants, coinciding heart failure, duration of the AF episode, and more. However, there is tremendous variation across providers, hospital systems, and even regions with regard to how new-onset AF is managed in acute setting. US physicians infrequently attempt early cardioversion (26 % of the time) compared to higher rates as observed in the United Kingdom and Canada of 50 % and 66 %, respectively [7]. With an increasing focus on patient-centered care, crowded hospital wards, and enhanced systems to obtain prompt cardiology follow-up, many US hospitals are developing programs to cardiovert and discharge from the ED an increasing proportion of patients.

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AF presents in a variety of ways in the acute setting. Patients may be symptomatic or asymptomatic, and in a minority of cases, the AF may result in hemodynamic instability. Generally, patients with hemodynamic instability from AF have other cardiovascular diseases (e.g., aortic stenosis or coronary artery disease). AF rarely is sufficient in and of itself to cause hypotension or shock. Stable patients with AF, where the exact onset is unknown, or patients with AF whose onset is greater than 48 h prior to the arrival and not on therapeutic anticoagulation for >3 weeks, are not candidates for elective cardioversion in the ED or observation unit (OU) and require further treatment and assessment prior to any attempt at rhythm control. The general rationale herein is that a transesophageal echocardiogram is frequently needed for such patients to rule out a left atrial thrombus.

The purpose of the present review is to outline indications and techniques for cardioversion of new-onset AF – both pharmacologic cardioversion (PC) and electrical cardioversion (EC) – in the first 24–48 h after a patient arrives to the hospital/ED. Of note, the decision to manage patients with persistent or permanent AF with a focus on rate versus rhythm control has been studied in detail [8–10] and will not be discussed in this review. One of the central tenets of cardioversion is that symptomatic patients benefit from cardioversion whereas asymptomatic patients may not benefit.

The Unstable Patient with AF

A minority of patients will present with AF and have symptoms and signs of instability such as chest pain, persistent hypotension, mental status changes, or heart failure. Typically these patients will demonstrate AF with a rapid ventricular response and have heart rates greater than 150 beats per minute [11]. When these signs and symptoms are felt to be due to AF, advanced cardiovascular life support (ACLS) guidelines recommend immediate synchronized EC (see Fig. 10.1).

Approach to Emergency Electrical Cardioversion

The goal of EC in the unstable patient is to convert the destabilizing AF rhythm to sinus rhythm as safely and quickly as possible. Despite the urgency of the situation, there are several management considerations that need to be addressed such as airway support, pain control, procedural sedation, energy selection, optimal defibrillator pad placement, management of other medical conditions, and anticoagulation to mitigate stroke risk. At minimum the patient should be placed on a cardiac monitor, given supplemental oxygen, and have an IV placed from which point-of-care labs (blood and chemistry counts) may be obtained. Electrolyte abnormalities should be addressed as appropriate, and the EKG should be reviewed for abnormal findings (e.g., ischemia, hyperkalemia). In addition, the unstable patient with unknown onset

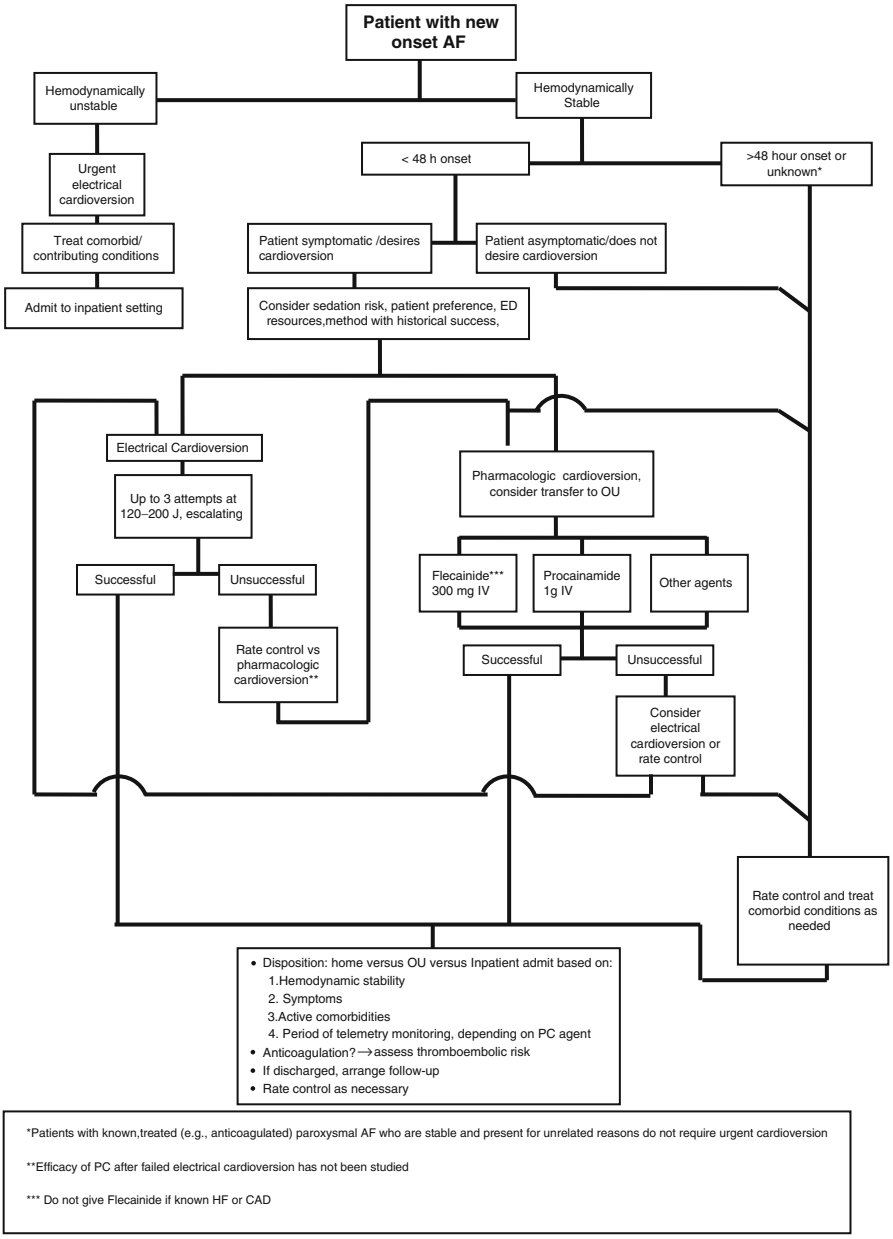

Fig. 10.1 Flow Chart

of AF, who is not already on anticoagulation and does not have a contraindication to anticoagulation, should be anticoagulated with an appropriate agent (see Chap. 20) before electrical cardioversion, if at all possible, or immediately after regardless of stroke risk (e.g., CHA2DS2-VASc) score [12].

Procedural sedation (see Chap. 20) should be considered for the unstable patient; however, limited time and unstable vital signs may limit options. After ensuring the patient has adequate airway support, pre-procedural pain control is paramount, and a bolus of intravenous medication (e.g., fentanyl) with quick onset of analgesia, and little effect on lowering blood pressure, can be considered. Short-acting sedative agents such as midazolam or propofol may be considered cautiously due to their potential effect of lowering blood pressure. Etomidate, which has less of an effect on blood pressure, may be a viable option for sedation of these patients. However, electrical cardioversion should not be significantly delayed for sedation of these high-risk patients.

Once the patient is deemed ready for the procedure, recommendations are to begin with synchronized cardioversion at 120–200 J biphasic or 200 J monophasic. If unsuccessful, one can repeat the cardioversion with increased energy, although there are no specific recommendations on escalating doses. It is also important to note that at equivalent energies, biphasic machines have a higher success rate than monophasic machines and with less thermal injury [13, 14].

There have been no randomized controlled trials to date evaluating ideal defibrillator pad position for patients needing electrical cardioversion specifically for recent-onset AF. Most studies have employed a strategy of anterior-posterior (A-P) placement or right parasternal and left midaxillary position [referred to as anterior-lateral or (A-L)]. A recent systemic review of 13 AF studies found overall no statistical difference in cardioversion rate between these two approaches. Subgroup analysis of only biphasic shocks found a trend toward superiority of A-L placement [15]. Among the included studies, A-P placement varied from right infraclavicular and left infrascapular to left infraclavicular and left infrascapular. Other variants are the right upper chest sternal body and left third intercostal space at the angle of the left scapula [16]. Cardioversion has been shown to be safe for patients with implanted pacemakers/defibrillators although recommendations include biphasic current and A-P placement with pads at least 8 cm from the device [17]. If time allows, any moisture may be wiped off the skin and excessive chest hair removed as is recommended per ACLS defibrillation guidelines [11]. Another consideration for patients with defibrillators is the use of internal cardioversion, a lower energy (typically 20 J) and less painful option in lieu of external cardioversion.

Management After EC, a Focus on Anticoagulation

After successful cardioversion, there are several other management considerations. If not done before cardioversion, stroke risk needs to be assessed based on the duration of the acute AF episode (greater than or less than 48 h), current comorbidities,

contraindications to anticoagulation, and thromboembolic risk as defined by CHA2DS2-VASC stroke risk score. For patients deemed high risk (typically 2 or more points), intravenous or subcutaneous anticoagulation should be administered prior to cardioversion, or as soon as possible thereafter. Transition to an oral agent (warfarin or target-specific anticoagulant) should be made and continued for at least 4 weeks for patients with a CHA2DS2-VASc risk score ≥2. Previously unstable patients with a CHA2DS2-VASC score of 0 or 1 that had AF for less than 48 h (if this can be accurately determined) do not require further anticoagulation treatment. Aspirin may also be considered for score of 1 [12]. Keep in mind, however, that irrespective of CHA2DS2-VASc score, if AF is of greater than 48 h duration, anticoagulation should be prescribed for 4 weeks post-cardioversion (see Chap. 20).

The disposition for previously unstable patients with AF who have undergone cardioversion will generally be admission for further diagnosis and treatment including that required for any concomitant acute medical conditions. Any exception to this approach, such as admitting these patients to observation following cardioversion, would necessitate close coordination and consultation with Cardiology for management in the OU.

The Stable Patient with AF

The vast majority of patients who present with acute AF will be hemodynamically stable and do not require emergent cardioversion. Initially the focus for these patients will be on control of heart rate if they are presenting with AF and tachycardia. The subsequent management approach to these patients is guided by several key factors such as whether the time of onset of this episode of AF can be accurately determined, how symptomatic the patient is, what medications they are taking, what comorbidities are present, and patient/caretaker treatment preferences regarding early rhythm control. Patients with persistent AF will require coordinated long-term outpatient treatment, and acute management for these patients will focus on rate control and stroke prevention as needed.

In appropriately selected patients with new-onset AF of less than 48 h duration, an approach focused on early rhythm control has the potential to improve patient satisfaction and may decrease short-term healthcare costs [18, 19]. Often younger, healthier patients may prefer to be immediately converted back to normal sinus rhythm (NSR) for convenience or if the AF is producing symptoms such fatigue, dyspnea with exertion, or palpitations. Early attempts at cardioversion may also be more successful when compared to strategies that delay this procedure for days or weeks, possibly due to atrial remodeling, a process that encompasses structural and electrical changes that promote maintenance of AF [20, 21].

Previous Literature

Several studies have examined the safety and efficacy of early rhythm control in patients with new-onset AF of less than 48 h in duration [3, 5, 22]. The 48-h time period identifies patients who are at very low risk of left atrial thrombus formation and therefore do not require evaluation with echocardiography and treatment with anticoagulation prior to cardioversion. One caveat pertains to patients with AF less than 48 h that are at a high stroke risk (e.g., CHA2DS2-VASC = 2 or greater). It is advised that these patients receive heparin, factor Xa, or a direct thrombin inhibitor "as soon as possible before or immediately after cardioversion, followed by long-term anticoagulation" [12].

Considerations Before Cardioversion

Unlike the unstable patient, there is much more time to prepare for the cardioversion of patients with new-onset AF who are hemodynamically stable. This time allows for more detailed history, exam, and consideration and treatment of precipitating events such as electrolyte abnormalities, anemia, hypothermia, acute decompensated heart failure, pericarditis, and others. When the choice is made to move forward with cardioversion in the ED/OU, the first attempt can be done either pharmacologically with an antiarrhythmic agent or with electrical cardioversion as previously discussed. Some have advocated using antiarrhythmics before cardioversion as a majority will convert to sinus rhythm, and this does not require the resources of procedural sedation for electrical cardioversion [3, 23].

Method of Cardioversion

Pharmacologic Cardioversion

The major agents for PC with Level 1A evidence include flecainide, ibutilide, propafenone, and dofetilide [12]. (Table 10.1) Procainamide, although not in the current AHA recommendations, is present in the 2010 Canadian guidelines [38] and has been used with success in ED-based studies of elective cardioversion [3, 23]. Medication selection is guided by practitioner preference and familiarity, potential contraindications (e.g., history of prior myocardial infarction, heart failure), and route of administration (oral vs. intravenous). If a patient presents in AF who is on a certain class of antiarrhythmic at home, it may be worthwhile to attempt pharmacologic cardioversion with a medication from a dissimilar class to take advantage of a different mechanism of action, or to consult a Cardiac Electrophysiologist for advice.

Table 10.1 Drug dosing for pharmacologic cardioversion of AF

Agent	Dosage and route	Considerations	Contraindications	Refs
Flecainide	200–300 mg PO × 1	Hypotension, ventricular arrhythmias	CAD, structural heart disease including CHF	[24–27]
Procainamide	1 g IV over 60 min	Hypotension, QT prolongation, arrhythmias	Electrolyte abnormalities, long QT, others	[3, 23, 28]
Dofetilide	500 mg PO × 1 (CrCl >60), 250 mg PO × 1 (60 < CrCl < 40), 125 mg Pox 1 (40 < CrCl < 20)	Renal dosing, QT prolongation, torsades de pointes	CrCl < 20	[26, 29, 30]
Ibutilide	1 mg IV over 10 min, may repeat × 1 (use 0.01 mg/kg if <50 kg)	QT prolongation, torsades de pointes, hypotension	Electrolyte abnormalities, long QT, others	[31–34]
Propafenone	450–600 mg PO × 1	Hypotension, ventricular arrhythmias	CAD, structural heart disease including CHF	[16, 24–27, 35]
Amiodarone	150 mg IV over 10 min, followed 1 mg/min for 6 h, then 0.5 mg/min for 18 h	Hypotension, bradycardia, QT prolongation, increased INR, phlebitis	Electrolyte abnormalities, thyrotoxicosis, long QT, others	[26, 36, 37]

Of note, rapid AF in the context of an antegradely conducting accessory pathway (e.g., Wolff-Parkinson-White syndrome) is a circumstance where ibutilide and procainamide have shown particular efficacy, while nodal blocking agents should generally be avoided [31]. "FBI" is a term often used to describe the characteristic appearance of AF in the presence of a manifest accessory pathway (fast, broad, and irregular). Should a patient present with these electrocardiographic features, procainamide and ibutilide are the preferred agents [31].

Flecainide and Propafenone

A placebo-controlled trial of patients with AF for 7 days or less found no statistical difference in conversion rates for 300 mg flecainide versus 600 mg propafenone at 3 h (59 % vs. 51 %) and 8 h (78 % vs. 72 %), respectively [24]. An additional study found that both medications were efficacious in the out-of-hospital setting for recent-onset (<48 h) arrhythmias in patients who responded to a loading dose of either medicine in the ED or inpatient setting [35]. Regarding propafenone's intravenous (IV) formulation, a review of trials comparing this to the oral route found the

parenteral option superior within the first hour of administration but no difference in conversion rates at 3 and 8 h [25]. Flecainide and propafenone are both Class IC agents and are contraindicated in patients with left ventricular dysfunction, sick sinus syndrome, congestive heart failure, or a QRS duration >110 msec [26].

Ibutilide

In early studies, ibutilide, a Class III antiarrhythmic, demonstrated a 30–50 % conversion rate of acute-onset AF, usually 60–90 min after IV infusion [32]. If unsuccessful, a repeat infusion can be attempted. Magnesium and potassium deficits must be repleted prior to infusion for optimal conversion success and to minimize QTc prolongation that could result in ventricular tachyarrhythmias [39]. Close cardiac monitoring is important as ibutilide carries a 2–3 % risk of inducing torsades de pointes, and this usually occurs during or shortly after the infusion [32]. Additionally, structural heart disease does not appear to confer increased risk or limit cardioversion success, nor does pretreatment with a patient's home medications.

Dofetilide

Dofetilide is a Vaughan-Williams Class III antiarrhythmic drug that has never specifically been studied for cardioversion of new-onset AF in the ED. Nevertheless, dofetilide is listed in the 2014 American Heart Association AF guidelines for pharmacologic cardioversion and has been shown to be safe in patients with advanced heart failure [12, 26]. A double-blind study found dofetilide converted approximately 24 % of patients with chronic AF (defined as >7 day duration) at the highest dose of 500 mg in 24 h [29]. Dofetilide is contraindicated in patients with a QTc >440 due to risk of torsades de pointes (QTc >500 if the patient has a ventricular conduction delay). Of note, dofetilide requires renal adjustment and QTc monitoring post-conversion [12]. Given the potential prolonged time to conversion to sinus rhythm, and the subsequent need for QTc monitoring, an OU or inpatient admission is likely to be necessary when using dofetilide. Generally, dofetilide administration is restricted to approved providers who have completed an online safety course.

Vernakalant

A relatively atrial-selective drug with both Class I and III properties, vernakalant is currently used in Europe, Canada, and Australia for pharmacologic cardioversion of AF. In a recent clinical trial, it showed a 58 % conversion rate of AF to sinus rhythm at 90 min after infusion for arrhythmias lasting between 3 and 48 h. The large majority (98 %) of patients remained in sinus rhythm at 24 h, although this included individuals with initial symptoms up to 45 days in duration [40]. Vernakalant is not yet available in the United States.

Procainamide

A type 1A antiarrhythmic, procainamide was used as the antiarrhythmic of choice in the Ottawa Aggressive Protocol for pharmacologic cardioversion of patients presenting with AF onset less than 48 h due to its relatively rapid effect and reasonable safety profile. In the Ottawa study, procainamide IV administration resulted in 58 % of patients converting to sinus rhythm. In addition there were no significant complications such as death or stroke [3, 23]. Minor complications were primarily limited to transient hypotension and treatable arrhythmias, mostly asymptomatic bradycardias. As part of the protocol, patients who did not convert with pharmacologic cardioversion were then given the option for electrical cardioversion, with a resultant 92 % success rate [3].

Amiodarone

Amiodarone's ability to convert recent-onset AF is often delayed relative to other pharmacologic cardioversion options, as it usually requires a bolus followed by 24 h of infusion. A meta-analysis including 1174 patients showed that amiodarone converted 82 % of patients at 24 h versus placebo at 56 % [36]. This medication may be preferred in the context of left ventricular dysfunction and acute ischemia as well as when blood pressure is lower than the patient's baseline [26].

Pharmacologic Cardioversion Summary

In summary, there are a number of antiarrhythmics that can be used in attempted pharmacologic cardioversion of new-onset AF to normal sinus rhythm. The choice of what agent to use will be guided by patient comorbidities such as heart failure, coronary disease, and renal insufficiency; practitioner preference; as well as ED and pharmacy resources. Attempting pharmacologic cardioversion before electrical cardioversion has a reasonably high chance of success and uses less clinical resources. In addition, pharmacologic cardioversion can be used in conjunction with the OU as a place to monitor the patients over time for conversion to sinus rhythm or for the need for subsequent EC (Fig. 10.1).

"Antiarrhythmic" Effect of Rate-Controlling Agents

Anecdotally, AV nodal blocking drugs (e.g., beta-blockers and calcium channel blockers) have been correlated with a return to normal sinus rhythm in the ED. This is often in the context of controlling a rapid ventricular response. A 2008 study found that when randomizing patients with AF of less than 48 h duration to ED observation versus admission, 32 % of patients treated with rate control in the OU converted to sinus rhythm within 6 h without having the need for electrical cardioversion [41].

Electrical Cardioversion

Electrical cardioversion may be used for those patients who fail pharmacologic cardioversion as well as for those patients/providers that prefer electrical cardioversion as an initial therapy. Not infrequently, patients who have a history of episodes of AF that responded to electrical cardioversion may request that it be attempted first, in lieu of waiting the hours sometimes necessary for pharmacologic cardioversion to work. The current literature supports a higher success rate for electrical cardioversion compared to pharmacologic cardioversion. A recent prospective randomized control study found that electrical cardioversion was superior to IV propafenone for cardioversion (89 % vs. 74 %) with less time in the ED (180 min vs. 420 min) [16]. Regarding safety after electrical cardioversion, studies have demonstrated an excellent safety record with few complications [16, 18].

As mentioned previously, various energy intensities for electrical cardioversion of AF are used. The 2015 AHA/ACC/HRS guidelines do not provide specific recommendations, although most studies start at 100-200 J biphasic with increased energy for any needed subsequent shocks up to 200 J. Adhesive defibrillator pad placement is generally the same as for unstable patients described above. There is variability among clinicians regarding the number of unsuccessful shock attempts before pursuing a rate control strategy (if warranted), although most studies have employed up to three shocks in the hemodynamically stable patient with AF [5, 16].

Sedation

In patients with new-onset AF who are hemodynamically stable, there are various sedation options worth considering for EC. Common drugs for pain or sedation include morphine, fentanyl, etomidate, propofol, midazolam, ketamine, and others (see Chap. 20). Practitioners should use what they are most comfortable with, taking into account the specific patient comorbidities and hemodynamics (e.g., relative hypotension with propofol). To date, no studies have demonstrated that one sedation scheme is superior to another with regard to cardioversion success rate, and there is significant variability of agents used in various countries [7]. The safety and efficacy of the sedation portion of electrical cardioversion rely on detailed preparation for the procedure, appropriate agent selection and usage, as well as careful monitoring of the patient throughout the periprocedural period.

Considerations/Contraindications for Cardioversion of Stable AF

The key consideration when deciding whether or not to cardiovert a patient with AF who is not hemodynamically unstable is whether or not the patient is symptomatic. Asymptomatic patients can be managed safely with rate control and anticoagulation as appropriate based on stroke and bleeding risk. Cardioversion of the stable patient

with AF should not be attempted in instances where a precipitant (thyrotoxicosis, pericarditis, valve disease, hypovolemia, sepsis, etc.) has been identified but not yet treated. Multiple other factors should be considered when deciding between electrical and pharmacologic cardioversion. Electrical cardioversion has the advantage of being immediate but the risks from the use of sedation may make it less desirable in a busy ED setting. Other factors to consider include the fact that older, more frail patients, those with relative hypotension, patients who have recently eaten, and those with difficult airways' may make procedural sedation for cardioversion higher risk. Additionally, procedural sedations for electrical cardioversion can be relatively resource intensive in the acute setting, potentially requiring significant time for the physician, nursing, and respiratory staff, as well as additional equipment resources. This may limit the use of electrical cardioversion for any setting with limited resources.

Disposition

After cardioversion of a stable patient with AF, the disposition can vary from admission to the inpatient floor, transfer to the OU, or discharge to home with cardiology follow-up (see Chap. 20). Generally the goal of early cardioversion is to facilitate discharge to home and avoid a potentially costly admission. However, utilizing the OU is an appropriate strategy for these patients and has been shown to be safe and result in a shorter length of stay versus patients admitted to the hospital [41]. For patients in whom cardioversion was unsuccessful, the need for rate control and anticoagulation (based on their CHA2DS2-VASC score) should be considered, and many of these patients may still be discharged with appropriate Cardiology and primary care follow-up. A key issue is transitional care management of anticoagulation since the 2–3 weeks after cardioversion are a particularly high-risk time period for thrombus formation. Cardiology consultation can be considered for a discussion of the risks and benefits of bridging anticoagulation after cardioversion.

Conclusion

Historically, therapy for the stable patient with new-onset AF involved rate control and admission to the hospital despite the fact that most patients with AF are at low risk for near-term adverse events [42]. In the last 10–15 years, in part driven by a better understanding of AF and its prognosis, AF is increasingly being managed by ED physicians without admission by providing select, lower-risk patients the opportunity for cardioversion to restore their native rhythm safely and expediently. In rare cases, urgent electrical cardioversion is needed to regain hemodynamic stability, but the vast majority of ED cardioversions are carried out electively for symptomatic patients when onset of AF is determined to be 48 h or less. While electrical cardioversion has generally shown greater success across many studies, it is reasonable to

try pharmacologic cardioversion first in stable patients since it requires fewer resources (e.g., sedation and monitoring). Among pharmacologic cardioversion agents, flecainide and procainamide have reasonable success in a short-time period with overall favorable side effect profiles. Other antiarrhythmic agents may be selected for certain patient populations based on comorbidities and other considerations. In summary, utilizing cardioversion for stable and unstable patients with new-onset AF in the acute setting is a safe and efficacious strategy that practitioners in the ED should be prepared to provide. Physicians should familiarize themselves and their staffs with contemporary cardioversion treatment strategies in light of the increasing number of patients being diagnosed with new-onset AF and presenting for acute care.

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Chapter 11
Procedural Sedation for Atrial Fibrillation Patients

Sharon E. Mace

Definitions and Goals of Procedural Sedation and Analgesia

Procedural sedation and analgesia, previously referred to as conscious sedation, is the process of administering sedatives or dissociative medications simultaneously with analgesics, as needed, to produce an altered state of consciousness which permits the patient to undergo unpleasant and/or painful procedures while preserving cardiovascular function [1]. Analgesia is defined as pain relief without intentional alteration of mental status. Anxiolysis refers to the condition in which the patient experiences decreased apprehension without affecting their level of awareness. Sedation occurs along a continuum that ranges from minimal to moderate, followed by deep sedation and finally general anesthesia [2]. Dissociative sedation is a trance-like cataleptic state in which spontaneous ventilation, airway, and cardiopulmonary function are maintained, while the patient is unresponsive to all stimuli [3]. The classic and most widely used dissociative agent is ketamine.

Eliminating or at least minimizing the anxiety, suffering, and pain that may occur during diagnostic or therapeutic procedures is the avowed purpose of procedural sedation and analgesia [2].

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Considerations in the Selection of Sedatives and Analgesics for Procedural Sedation: Patient Factors

The selection of an appropriate sedation and analgesic regimen for any patient undergoing a medical procedure, including a patient with atrial fibrillation, is dependent upon multiple variables incorporating patient considerations, the properties of the specific sedative and or analgesics, and other factors, including the practitioner's knowledge base and the site or location where the procedural sedation is to occur [4].

Obviously, the specific properties of the sedative(s) and analgesic(s), and particularly their effect on the various organ systems, especially the cardiovascular, respiratory, and neurologic systems, are key items for consideration [4].

Patients need to be assessed regarding their risk for procedural sedation and analgesia in order to determine the potential for adverse effects when undergoing procedural sedation and analgesia. This allows the practitioner to anticipate potential problems and ideally avoid or minimize any adverse events related to the procedural sedation and analgesia [4].

Patients presenting with atrial fibrillation range in complexity from essentially healthy young patients with a structurally normal hearts to hemodynamically unstable geriatric patients with end-stage heart failure and multiple comorbidities. Patients range in age from the newborn or infant with congenital heart disease to the geriatric patient with multiple comorbidities and New York Heart Association (NYHA) class IV [5, 6]. Providing procedural sedation and analgesia to a patient with atrial fibrillation may create additional difficulties or problems and often requires additional consideration and usually adds another layer of complexity to the process.

Patients at Risk for Adverse Events During Procedural Sedation

Who is at increased risk for adverse events during procedural sedation? Various factors have been linked with an increased incidence of adverse events during procedural sedation and analgesia. Patients with significant comorbidity such as cardiovascular disease, respiratory disorders, or neurologic disease have a higher incidence of adverse events or complications than patients without any comorbidities [7, 8]. Moreover, patients whose comorbid condition is well controlled have a lower occurrence of adverse events than patients whose comorbid condition is poorly controlled.

Obviously, a patient with asthma who is experiencing an acute exacerbation of their asthma and is wheezing and hypoxic would pose more problems for sedation than an asthmatic who is well controlled, not experiencing an acute asthmatic attack, is not wheezing, and is not hypoxic. Similarly, a patient with atrial fibrillation who

is rate controlled, with stable vital signs, and is asymptomatic is different from a patient with symptomatic atrial fibrillation who is having chest pain or shortness of breath, has a heart rate of 180, and is hypotensive. The symptomatic, hypotensive, tachycardic atrial fibrillation patient whose heart rate is not well controlled would be more likely to experience a complication from procedural sedation and analgesia than the stable, asymptomatic patient.

This is reflected in the American Society of Anesthesiology (ASA) physical status classification [4] (Table 11.1). Patients with a higher ASA physical status classification, especially 3 or above, are at greater risk than lower physical status classification, with the greatest risk in the highest ASA physical status classification (Table 11.2). ASA 1 is a normal healthy patient with no medical illnesses or diseases. ASA 2 is a patient with mild disease. An ASA 2 patient has no functional limitations and has well-controlled disease of one body system. An example of an ASA 2 patient would be a well-controlled asthmatic with mild, intermittent asthma who is not having an asthmatic attack. A hypertensive patient on antihypertensive medications whose blood pressure is well controlled and in the normal range would be an ASA category 2 (Table 11.1).

For the young healthy atrial fibrillation patient with no structural heart disease and no other comorbidities who is asymptomatic with stable vital signs whose heart rate is rate controlled (e.g., within the normal range), he/she might be deemed as

Table 11.1 American Society of Anesthesiology (ASA) physical status classification

ASA 1: Normal healthy patient. No organic, physiologic, or psychiatric disease
ASA 2: Patients with mild disease: No functional limitations. Well-controlled disease of one body system. Examples: well-controlled asthma, well-controlled hypertension
ASA 3: Patients with severe systemic disease: Some functional limitation. Controlled disease of ≥ 2 body systems with no immediate danger of death
ASA4: Patients with severe systemic disease that is a constant threat to life: ≥ 1 poorly controlled or end-stage disease with possible risk of death. Examples: unstable angina, symptomatic congestive heart failure, symptomatic chronic obstructive pulmonary disease, symptomatic atrial fibrillation
ASA 5: Moribund patients who are not expected to survive without an operation and/or other immediate intensive care. Patients are not expected to survive > 24 h without surgery. Patients are at imminent risk of death, have multiorgan failure, and/or are hemodynamically unstable. An example would be a sepsis patient who is anuric with kidney failure on dialysis, has liver failure, and is in respiratory distress for which he/she is intubated and on a ventilator
ASA 6: Declared brain-dead patient whose organs are being removed for donor purposes

Table 11.2 Factors associated with adverse events during procedural sedation

Comorbidity: especially patients with significant cardiovascular or respiratory or neurologic disorders
High ASA physical status
Extremes of age: elderly or very young – neonates and infants
Other *possible* factors: multiple sedatives and/or analgesics, high doses of medications, the procedure being done, the location

Table 11.3 Commonly used sedatives for procedural sedation

Sedative	CV effects	Use with caution if	Other side effects	Contraindications
Etomidate	Minimal CV depression	Adrenal insufficiency	Pain on injection, myoclonic movements, ↓adrenal steroid production	Adrenal insufficiency
Ketamine	CV stability Sympathomimetic Effects: ↑ HR, ↑BP	Hypertension Tachycardia	Emergence reactions, ↑ Intraocular pressure[a]	Thyrotoxicosis or other conditions with marked sympathetic activity, psychiatric conditions
Midazolam	Mild CV effects unless volume depleted	Hypotension shock	Paradoxical agitation vomiting, coughing, hiccups	Shock or significantly low BP (consider IVF bolus)
Barbiturates: methohexital, pentobarbital	Can cause CV depression	Hypotension shock, impaired cardiac function	Vomiting, coughing, hiccups; extravasation can cause tissue necrosis; intra-arterial injection can cause gangrene	Porphyria
Propofol	CV depression is dose/rate of administration dependent, negative inotrope (↓ CO) ↓ SVR (vasodilatation) ↑ Vagal tone (↑ risk of bradyarrhythmias when given with certain drugs)	Shock, low BP, impaired cardiac function	Respiratory depression, apnea	Egg, soybean, or EDTA allergy

CV cardiovascular, *HR* heart rate, *BP* blood pressure, *IVF* intravenous fluids, *CO* cardiac output, *SVR* systemic vascular resistance, ↑ increase, ↓ decrease

[a]↑ intracranial pressure was previously thought to be a contraindication, but recent reports suggest that ketamine may be safe and effective in patients with head injuries or CNS disorders

ASA 2. The patient with atrial fibrillation with good heart rate control and stable vital signs, who also has COPD or CHF, has well-controlled disease of two body systems and might be classified as ASA 3. The atrial fibrillation patient with CHF who is dyspneic, tachycardic with a heart rate of 160, and hypotensive with a blood pressure of 90 and with rales, peripheral edema, and jugular venous distention could be classified as ASA 4.

There are other variables that may lead to an increased incidence of complications during procedural sedation and analgesia. The evidence indicates that age may be a significant factor. Patients at the extremes of age have a higher incidence of adverse events during procedural sedation. Being an infant, e.g., age <1 year, is a predictor of increased risk of sedation adverse events in some pediatric studies [7]. The elderly have been reported to have significantly higher complication rates than their nongeriatric adult counterparts [8–10]. This difference for age remains even when other factors such as comorbidity or ASA class are taken into consideration [9]. The geriatric patient with atrial fibrillation is more likely to suffer an adverse event during procedural sedation and analgesia than their younger counterpart, an adult <65 years old (Table 11.2).

Procedural factors that may be associated with a higher incidence of adverse events in some studies include multiple sedatives and analgesics (e.g., opioids), high doses of sedatives and/or opioids, and specific sedatives and/or analgesics [10–14]. However, other studies have reported different conclusions. The location where the procedure is performed may affect the incidence of complications during procedural sedation and analgesia. The highest incidence of complications during procedural sedation has been in dental offices according to some reports [15] (Table 11.2).

Procedural Sedation and Analgesia in the Emergency Department

It is recommended that there be at least one practitioner skilled in airway management, venous access, and resuscitation present during the procedure in case any untoward events occur [15]. Emergency physicians as part of their training and certification are required to be competent in airway management, the use of critical care medications including advanced cardiac life support (ACLS) drugs, resuscitation, and procedural sedation. This knowledge and these skills are especially important in unstable or critically ill atrial fibrillation patients who may be in need of emergent cardioversion. There is abundant evidence-based literature to suggest that procedural sedation can be effectively and safely performed in the emergency department (ED) in patients of all ages and with all types of conditions and diseases [1, 16].

Cardioversion in the Emergency Department

Moreover, a recent study found that cardioversion in emergency department (ED) patients with dysrhythmias, most commonly atrial dysrhythmias, specifically atrial fibrillation, can be effective and safe even in critically ill patients with multiple comorbidities and high ASA physical status classifications (e.g., ASA 4 or 5) [17]. Another study found that four sedation regimens (propofol, etomidate, midazolam, and midazolam/flumazenil) were similarly effective in hemodynamically stable adults undergoing cardioversion in the ED [18].

Considerations in the Selection of Sedatives and Analgesics for Procedural Sedation: Sedatives

There is a wide range of procedures for which patients may undergo procedural sedation and analgesia including orthopedic procedures (reduction of fractures and dislocations), lumbar puncture, incision and drainage of abscesses, removal of foreign bodies, wound care including suturing, insertion of various tubes (chest tubes, feeding tubes, nasogastric tubes), and radiologic procedures. In addition to these procedures, patients with atrial fibrillation may need to undergo cardioversion to treat their underlying rhythm disorder.

Sedatives frequently used for procedural sedation and analgesia include etomidate, ketamine, midazolam, methohexital, pentobarbital, and propofol. There are advantages and disadvantages associated with every sedative. The ideal sedative would have no side effects, have a rapid onset and rapid offset, and be applicable for every patient and every procedure. Unfortunately, there is no ideal sedative. The commonly used sedatives have varying effects on cardiovascular function (Table 11.3).

Since patients with atrial fibrillation may have congestive heart failure, poor cardiac function with an impaired ejection fraction, an abnormal heart rate (more commonly tachycardia than bradycardia), or may be hypovolemic or in shock, the effects of the sedative on cardiovascular function must be considered prior to procedural sedation and analgesia.

Propofol can cause cardiovascular and respiratory depression. Therefore, it should be used with caution in any patient with shock, hypotension, or impaired cardiac function [8, 19, 20].

The barbital class of sedatives includes the commonly used sedatives methohexital and pentobarbital. Cardiovascular and respiratory depression can occur with both methohexital and pentobarbital [21].

The sedative, midazolam, belongs to the benzodiazepine class of sedative drugs. Midazolam has mild cardiovascular effects unless the patient is hypovolemic so midazolam could be used in patients who are euvolemic or volume overloaded. Midazolam decreases left ventricular (LV) filling pressure so it may be useful in patients with a high LV pressure, as seen in many heart failure patients. Respiratory depression and apnea can occur with midazolam, especially in high doses given as

a rapid intravenous (V) bolus [22]. In cardiac patients who also have coexisting respiratory disease, such as chronic obstructive pulmonary disease (COPD), and are at risk for loss of respiratory drive, midazolam may not be a good choice [2, 23].

Ketamine has remarkable cardiovascular stability and is known as the battlefield drug since it can be used in situations where hemodynamic monitoring is difficult or impossible. Ketamine does not lower blood pressure unlike propofol, methohexital, or pentobarbital. However, ketamine has sympathomimetic effects, which generally result in an increase in heart rate and blood pressure, so it would not be the ideal drug of choice in patients who are tachycardic and/or hypertensive [24].

Etomidate has minimal cardiovascular and respiratory depression so it can be used in patients who are hypotensive, hypertensive or normotensive, and/or tachycardic or bradycardic. Etomidate does, however, cause adrenal suppression and should be avoided in patients with adrenal insufficiency. However, the evidence suggests that a one-time dose of etomidate does not have any lasting untoward side effects as regards to adrenal suppression [25].

There are other relatively new agents that might be used for procedural sedation and analgesia including dexmedetomidine (sedative) [26–28], alfentanil (an opioid, an analogue of fentanyl) [29, 30], and remifentanil (synthetic opioid) [31, 32]. Dexmedetomidine is usually administered as a loading dose over 10 min followed by a maintenance infusion [26]. The need for a loading dose might be a disadvantage if there is a need for an emergent sedation. Dexmedetomidine has mainly been used as a sedative for radiology procedures with mixed results [27] although there is a case report from the ED [28]. In one ED study in which alfentanil was given, it was safe and effective; however, recovery rates were longer when alfentanil was coadministered with propofol, and the authors concluded that there was no benefit of alfentanil with propofol re-hypoventilation [29]. Remifentanil use in the ED has been also reported [31, 32]. Without any large studies or randomized controlled trials of these agents in an ED setting, it is problematic to make recommendations for their use in the ED.

In summary, for cardiac patients including those with atrial fibrillation, the cardiovascular (and other) side effects of the sedative must be considered. Etomidate has minimal cardiovascular depression. This is also true for midazolam unless the patient is hypovolemic. Replacement of volume with a bolus of a crystalloid, such as normal saline, could be considered when using midazolam in a volume-depleted patient. The barbitals methohexital and pentobarbital can cause cardiovascular (and respiratory) depression, so they would not be the drug of choice in patients with impaired cardiac function. Ketamine does not cause cardiovascular depression but instead raises the heart rate and blood pressure so it would not be ideal in a patient who is already hypertensive or tachycardic (Table 11.3).

Steps in Procedural Sedation

Successful procedural sedation and analgesia encompasses several elements: patient assessment, personnel and monitoring equipment, discharge criteria, and quality improvement. Preprocedural assessment involves an evaluation of the patient,

beginning with a detailed history and physical examination emphasizing the cardiovascular, airway, pulmonary, and neurologic systems in order to identify any problems or abnormalities that could make the patient high risk for complications. Cardiac medications needed to manage any dysrhythmias or other cardiac events, including ACLS resuscitation medications, defibrillators, respiratory supplies, oxygen, suction, and even intubation equipment should be immediately available [2, 4].

Qualified and trained individual(s) who are able to manage any potential cardiovascular or pulmonary problems or other adverse events that may occur should be present during the procedure [16]. Monitoring, at a minimum, entails obtaining and recording vital signs and the depth of sedation at regular intervals along with continuous monitoring of heart rate, pulse oximetry, and ideally capnography. In addition, for patients with any preexisting cardiovascular disease, including atrial fibrillation, continuous cardiac rhythm strip monitoring is recommended. Monitoring should be ongoing, even after the procedure has been completed, since the patient is still vulnerable, and adverse events are possible after the stimuli and pain of the procedure has ended. Only after patients are fully awake, have returned to their preprocedural mental and physical status baseline, and have normalized vital signs should they be discharged [2].

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Chapter 12
Risk Stratification in Atrial Fibrillation and Observation Unit Entry

Edgar Ordonez

Introduction

The most common complication of atrial fibrillation is arterial thromboembolism, which can lead to ischemic stroke [1–3]. Thus, it is important to determine which patients would benefit from antithrombotic therapy to help lower the risk of thromboembolic events. As detailed in the chapter on anticoagulation therapy, there are various Federal Drug Administration (FDA)-approved options for stroke prevention in atrial fibrillation that are endorsed by current guidelines [4]. These would include both oral and parenteral treatments, including low molecular weight heparin, unfractionated heparin, vitamin K antagonists, direct thrombin inhibitors, and factor Xa inhibitors [4]. The initiation of these medications may occur in the outpatient, emergency department (ED), observation unit (OU), or inpatient setting and will depend on the patient's presentation and risk for thromboembolic events, which will be discussed in this chapter. Furthermore, risk stratification for entry into the OU will be discussed as a means to identify a select number of patients who may be appropriate for a short hospital stay and continued evaluation and management of atrial fibrillation.

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Stroke Risk Stratification Scores

As previously discussed, anticoagulation decreases the risk of stroke and other embolic events. Several decision schemes have been validated to guide practitioners on what patients with non-valvular atrial fibrillation would benefit most from anticoagulant therapy. The decision to initiate anticoagulation therapy should be a shared decision between the patient and provider, and a discussion of the benefits of stroke prevention and the risks of bleeding, along with the patient's values and preferences, needs to be considered.

The two most commonly used stroke risk assessment tools are the $CHADS_2$ and the CHA_2DS_2-VASc [5, 6]. The acronym $CHADS_2$ represents congestive heart failure, hypertension, age ≥75 years, diabetes mellitus, and stroke/TIA/thromboembolism. Each risk factor is assigned a point of either 1 or 2 and added together for a maximum of 6 (Table 12.1). The listed risk factors have previously been shown to increase the risk of stroke in patients with atrial fibrillation [7–13]. In general, patients with a $CHADS_2$ score of ≥2 should be on oral anticoagulation with warfarin, dabigatran, rivaroxaban, or, apixaban.

The American College of Cardiology, American Heart Association, and Heart Rhythm Society Guidelines recently published that the CHA_2DS_2-VASc risk score is the preferred tool to assess stroke risk in patients with non-valvular atrial fibrillation, and it has widely become accepted by clinicians [4]. The scoring system is also detailed in Table 12.1. Evidence for the utilization of this tool for risk stratification has come from studies showing that several other factors are known to increase stroke risk, and this tool helps identify patients who are high risk for stroke who otherwise would have been considered low or moderate risk with other stratification schemas. Patients with non-valvular atrial fibrillation and a CHA_2DS_2-VASc score

Table 12.1 $CHADS_2$ and CHA_2DS_2-VASc scoring system

$CHADS_2$	**Score**
Congestive heart failure	1
Hypertension	1
Age ≥ 75	1
Diabetes mellitus	1
Stroke/TIA/thromboembolism	2
Total maximum score	6
CHA_2DS_2-VASc	**Score**
Congestive heart failure	1
Hypertension	1
Age ≥ 75	2
Diabetes mellitus	1
Stroke/TIA/thromboembolism	2
Vascular disease	1
Age 65 to 74 years	1
Sex **c**ategory (i.e., female sex)	1
Total maximum score	9

of 0 can be omitted from antithrombotic therapy. Patients with a CHA_2DS_2-VASc score of 1 may have consideration of no treatment or treatment with an oral anticoagulant or aspirin. Patients with a CHA_2DS_2-VASc score of ≥2 should be considered for oral anticoagulation with warfarin, dabigatran, rivaroxaban, or apixaban [4]. In this tool, vascular disease, such as prior myocardial infarction, peripheral artery disease, or aortic plaque, is included in the acronym. Vascular diseases have been shown to increase thromboembolic risk in atrial fibrillation [14–16]. Also, a second age category is part of the acronym, as patients with atrial fibrillation who are aged 65 or greater are known to have an increased stroke risk [17]. Additionally, as published in the Birmingham Atrial Fibrillation Treatment of the Aged Study (BAFTA) trial, it was shown that vitamin K antagonists were clearly superior to aspirin in stroke prevention in patients aged ≥75 years in the primary care setting, hence why this age group receives an extra point in the scoring tool [18]. Lastly, the scoring tool adds a sex category, as female gender is known to increase the risk of stroke and thromboembolism [19–21]. Table 12.2 outlines the adjusted stroke rates per year based on $CHADS_2$ and CHA_2DS_2-VASc scores.

While the $CHADS_2$ scoring system is simpler to use than the CHA_2DS_2-VASc, there are notable limitations due to it not including the previously stated common risk factors. It is noted that patients classified as low risk by $CHADS_2$ in its original validation study still had a stroke rate of 1.9 % per year, as listed in Table 12.2 [5]. A recent meta-analysis by Olesen et al. showed that many patients with a $CHADS_2$ score of 0 were not all at low risk for stroke [22]. By instead utilizing the CHA_2DS_2-VASc score, one would better classify the risk of stroke in atrial fibrillation patients and guide decision-making for anticoagulation therapy more appropriately.

Table 12.2 Adjusted stroke rate per year

$CHADS_2$ score	**Adjusted stroke rate (% per year) [5]**
0	1.9
1	2.8
2	4.0
3	5.9
4	8.5
5	12.5
6	18.2
CHA_2DS_2-VASc score	**Adjusted stroke rate (% per year) [6, 23, 24]**
0	0
1	1.3
2	2.2
3	3.2
4	4.0
5	6.7
6	9.8
7	9.6
8	6.7
9	15.20

Bleeding Risk Scores

Patients with atrial fibrillation also need evaluation for bleeding risk prior to the initiation of oral anticoagulation for stroke prevention. While identifying bleeding risk should not exclude physicians from initiating oral anticoagulation, it is important to correct any modifiable bleeding risk factors if possible. It is known that physicians overestimate bleeding risk, especially in the elderly, and this is a barrier to prescribing oral anticoagulants in patients with atrial fibrillation [25, 26]. This is important to note because as demonstrated in an analysis by van Walraven et al., oral anticoagulation was significantly protective against ischemic stroke regardless of the patient's age. Their study showed that while the protective benefit of antiplatelet therapy decreased significantly as the patient aged, the benefit for oral anticoagulation increased as they aged. Additionally, while there was an increased risk of serious hemorrhage as patient's aged, there were no significant differences between patients on aspirin versus those on warfarin [27].

While there are several bleeding scores available [28–30], the HAS-BLED score is a simple tool that allows for evaluation of bleeding risk in patients with atrial fibrillation [31]. HAS-BLED has several risk factors that are also known to be risk factors for stroke. The acronym stands for hypertension >160 systolic, abnormal liver or renal function (defined as dialysis, renal transplant, creatinine >2.6, cirrhosis, bilirubin >2 X's normal, alanine transaminase, aspartate aminotransferase, or alkaline phosphatase >3 X's normal), history of stroke, predisposition to or prior major bleeding, labile international normalized ratio (INR), elderly age >65, and, drugs, including medications that predispose to bleeding, along with excessive alcohol usage. The HAS-BLED score ranges from 0 to 9 with scores ≥3 indicate a high risk of bleeding. It is important to reiterate that HAS-BLED should not be used as a tool to exclude patients from receiving oral anticoagulation but rather to help physicians identify modifiable risk factors as they proceed cautiously and regularly review treatment plans. Table 12.3 illustrates the scoring system.

Table 12.3 HAS-BLED risk score

Risk factor	Description	Score
Hypertension	>160 systolic	1
Abnormal liver or renal function	Renal disease: dialysis, transplant, Cr >2.6	1
	Liver disease: cirrhosis, bilirubin >2 X's normal, alanine transaminase, aspartate aminotransferase, or alkaline phosphatase >3 X's normal	1
Stroke	History	1
Bleeding	Predisposition to or prior major bleeding	1
Labile INRs	Labile INRs	1
Elderly	>65	1
Drugs/alcohol use	Medications predisposing to bleeding	1
	Excessive alcohol use	1
Total maximum score		9

Observation Unit Inclusion and Exclusion Criteria

There are often scenarios where patients with atrial fibrillation may not require inpatient admission, but a short stay in the OU could be appropriate for stable patients that are not ready for discharge from the ED and warrant further management. As detailed in other chapters, the OU can serve to utilize pharmacologic treatments in attempts to achieve rate control or chemical cardioversion, electrical cardioversion if indicated, and initiation of parenteral and/or oral anticoagulation, provide patient education and consultation with cardiologists, and have appropriate discharge planning.

OU treatment protocols for atrial fibrillation have previously been described in the literature as a means to provide an alternative method to prolonged inpatient management of uncomplicated atrial fibrillation [32–35]. More recently, the Society for Cardiovascular Patient Care (SCPC) published a white paper on observation services, which included recommendations for the management of atrial fibrillation in the OU [36]. Included in those recommendations were the following inclusion and exclusion criteria:

Inclusion Criteria

- Hemodynamically stable patient
- Heart rate less than 110–120 after initial rate control
- Patient able to assist in follow-up and treatment plan

Exclusion Criteria

- Evidence of comorbidities requiring inpatient hospitalization
- Concerns for ischemia: positive initial troponins or presence of ischemic ECG changes
- Signs of CHF
- Ongoing significant tachycardia or hypoxia

The criteria above provide only framework for OU entry. There are several ways to treat atrial fibrillation, and an OU protocol may be best identified on an individual facility basis, as long as guideline and evidence-based medicine are practiced. This may depend on several factors including the availability of certain pharmacologic agents, diagnostic testing, and cardiology consultation. Either way, the OU is a viable destination for the uncomplicated patient with atrial fibrillation to receive further management to determine the need for inpatient admission or outpatient follow-up.

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Chapter 13
Cardiac Implantable Electronic Devices in the Short Stay Management of Atrial Fibrillation

Brian Hiestand and Andrew Wong

As discussed in previous chapters in this text, the incidence and prevalence of atrial fibrillation in the United States is substantial and expected to continue to increase [1]. As a result, we will continue to see increasing numbers of patients presenting to the emergency department (ED) primarily due to atrial fibrillation, due to a secondary complication of atrial fibrillation, or with atrial fibrillation as a comorbidity to a primary illness. In addition, atrial fibrillation frequently coexists with other cardiovascular disease states, such as chronic heart failure and ventricular tachydysrhythmias. Cardiac implantable electric devices (CIED), which include pacemakers and defibrillators, can serve multiple functions in patients with cardiac disease. In this chapter, we will discuss the primary indications for CIED in patients with atrial fibrillation or coexisting HF, as well as potential device-related complications. Finally, we will examine the data available from interrogation of CIED and how that data may be utilized to advance the management of the atrial fibrillation patient in the short stay setting.

Device Basics

The core components of a CIED include the canister, containing the battery, generator, and programming functions, and one to three leads, which run transvenously to implant in the myocardium. Leads may be implanted in the right atrium, right ventricle, or left ventricle. Left ventricular leads, if present, traverse the coronary sinus to an epicardial location to pace the left ventricle. Defibrillator capacity requires the presence of a shock coil, usually placed in the right ventricle.

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Table 13.1 The North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group generic code for pacemakers [2]

Position	I	II	III	IV	V
Category	Chamber paced	Chamber sensed	Response to sensing	Rate modulation	Multisite pacing
Nomenclature	*O* = None *A* = Atrium *V* = Ventricle *D* = Dual (A and V)	*O* = None *A* = Atrium *V* = Ventricle *D* = Dual (A and V)	*O* = None *T* = Triggered *I* = Inhibited *D* = Dual (T and I)	*O* = None *R* = Rate modulation	*O* = None *A* = Atrium *V* = Ventricle *D* = Dual (A and V)

CIED functionality is described by an alphabetic code system (Table 13.1) that describes the programmed function of the device [2]. The first letter describes which cardiac chamber is paced. The second letter refers to the chamber being sensed. The third letter describes the primary response of the device when an intrinsic cardiac depolarization is sensed. The device may be triggered to send a pacemaker spike, inhibited from sending a spike, or have a dual response (both triggered and inhibited). Dual response occurs in dual-chamber pacemakers, where an atrial impulse is sensed and conducted to the ventricle (triggered), but the pacemaker is inhibited from sending a spike to the atrium along the atrial lead. The fourth letter refers to how variable the pacing rate can be, whether output is fixed ("O" for no programmability) or variable depending on demand ("R"). Finally, the fifth position defines multisite pacing.

CIED Indications

Atrial Fibrillation

The primary indication for CIED placement in patients with atrial fibrillation is rate control. This may consist of providing a basal minimum rate in patients with sick sinus syndrome or tachy-brady syndrome. Using a ventricular sensing lead, the CIED can provide ventricular, atrial, or sequential pacing to ensure a minimum cardiac rate, although AV sequential pacing is preferred. Although pacing has not been shown to affect mortality, symptoms from inappropriate bradycardia can be controlled [3].

A frequent indication for CIED placement in atrial fibrillation is rate control by means of AV node ablation, thus theoretically eliminating the possibility of rapid ventricular response. This step may be indicated in patients who cannot tolerate pharmacologic rhythm or rate control agents, who have poorly controlled symptoms due to frequent episodes of rapid ventricular response. A recent meta-analysis compared ablation and pacing to medical therapy and demonstrated consistent improvements in quality of life and symptom control [4]. Unfortunately, after an AV ablation, the patient will typically be device dependent for the remainder of their life [5]. In

the case of device failure, this may be catastrophic, as a substantial portion (approximately 70%) of these patients will not be able to sustain a hemodynamically sufficient ventricular escape rhythm after ablation [6]. In these patients, device failure effectively results in profound bradycardia or asystole. It should be noted that the underlying atrial fibrillation continues unabated. Anticoagulation for stroke prophylaxis is still appropriate based on the risk benefit ratio for the individual patient [7].

The evidence base is not encouraging with regard to the utilization of atrial pacing, either using a continuous pacing rate to prevent atrial fibrillation or overdrive pacing for tachycardic episodes, as prophylaxis in paroxysmal atrial fibrillation. Multiple studies have failed to show benefit, or even suggest harm, with these strategies [8–13]. Oversensing can lead to inappropriate device activation, while undersensing can lead to a failure to pace and control the dysrhythmia. Therefore, in general CIED placement is not indicated in for prevention of atrial fibrillation in patients without other indications for a CIED.

Coexisting Heart Failure

Many patients with atrial fibrillation develop chronic heart failure and vice versa. There is evidence that increased atrial distension due to elevated diastolic pressures can lead to increased electrical irritability and atrial fibrillation [14]. Likewise, remodeling of the myocardial architecture will occur as a maladaptive response to the poor circulatory hemodynamics of atrial fibrillation [15]. Frequent episodes of tachycardia will exacerbate the remodeling, but evidence demonstrates that atrial fibrillation control can lead to reverse remodeling and improved contractility [16]. As there are multiple CIED indications for patients with heart failure, CIEDs may be present in atrial fibrillation patients as therapy for their concomitant heart failure, as opposed to primary treatment of atrial fibrillation.

Sudden Cardiac Death

Patients with heart failure are at increased risk for ventricular tachydysrhythmias. The annual estimated incidence of sudden cardiac death (SCD) in the general US population is estimated at approximately 2 per 1,000 people (0.2%) [17]. By way of comparison, in heart failure patients with inducible dysrhythmias (the group at highest risk), the annual incidence of SCD surpasses 30% [18]. In patients with reduced ejection fraction (<35%), SCD is the cause of approximately 50% of all deaths [19].

Primary prevention refers to the use of a CIED in a patient at risk for SCD but who has not yet sustained a ventricular tachycardia/ventricular fibrillation (VT/VF) event. Secondary prevention refers to the use of CIED with defibrillation capability in a patient who has already survived a VT/VF event. Given that the probability of

recurrence of VT/VF is high in these patients, regardless of ventricular function or heart failure etiology, CIED placement is recommended [20]. It should be noted, however, that device placement is not indicated in patients with terminal conditions in whom life-prolonging therapies are not appropriate or desired.

The indications for device implantation are modestly more nuanced for primary prevention. Multiple studies have demonstrated the superiority of CIED over medical therapy for the primary prevention of SCD in select populations. The benefit has been demonstrated in patients with reduced ejection fraction (<35 %) in both ischemic [21, 22] and nonischemic heart failure [23]. Patients with atrial fibrillation with reduced ejection fraction may be considered for referral for CIED placement once stabilized.

Cardiac Resynchronization Therapy

In addition to arrhythmia control, CIED may be indicated to manage ventricular dyssynchrony. Slowed and asynchronous ventricular contraction can worsen cardiac function, resulting in worsening disease and dysfunctional remodeling. Cardiac resynchronization therapy (CRT) uses pacing leads in both ventricles to induce synchronous depolarization of the ventricles. This modality has been demonstrated to reverse cardiac remodeling, decrease heart failure symptoms, and enhance quality of life [24]. However, the benefit to CRT has only been demonstrated in patients with electrical conduction impairment as evidenced by a QRS > 120 ms, and the majority of these trials have been conducted in patients with reduced ejection fraction [25–32]. To examine the potential benefit in patients without widened QRS intervals, the EchoCRT study randomized patients with narrow QRS intervals, but echocardiographic evidence of mechanical dyssynchrony to CRT vs. device implantation without CRT programming turned on [33]. Unfortunately, the trial had to be stopped early due to increased mortality in the CRT arm, suggesting that CRT is not beneficial and may actually be harmful in patients without QRS prolongation. Recent meta-analyses noted an increased benefit in patients with atrial fibrillation and CRT if AV nodal ablation was also performed [34, 35]. Gasparini et al. demonstrated that the clinical benefit of CRT in atrial fibrillation approached that of patients with sinus rhythm, if nodal ablation was performed [36].

Device-Related Complications

Device-related complications can occur either early, such as site hematoma, pneumothorax, or wound dehiscence, or in a delayed fashion after implantation. It is unlikely that patients with the primary complaint of a device-related issue will be slated for management in the short stay setting; however, a complication may be discovered during the evaluation in the short stay unit, either as a secondary issue or an unsuspected cause of the patient's complaint.

Electrical Complications

There are several common complications of lead programming and impulse delivery for CIEDs. "Failure to capture" describes the inability of a discharged pacing spike to depolarize non-refractory tissue. Failure to capture can be diagnosed on EKG as a pacer spike that results in no effective pacing stimulus. Common causes of failure to capture include battery failure, lead fracture or displacement, fibrosis at the lead placement site, cardiac perforation, electrolyte abnormalities, and the presence of antiarrhythmic drugs. Conversely, the term "failure to pace" refers to the failure of the pacemaker to deploy a pacemaker spike after an appropriate interval and is generally secondary to device, battery, or lead malfunction as opposed to patient physiology.

Oversensing, where the pacemaker output is suppressed due to a stimulus that is interpreted as an appropriate cardiac conduction, can cause suppression of the physiologically appropriate device function. The programming interprets the incoming signal as an indication that a pacemaker spike is not required and therefore does not discharge. Oversensing can be caused by misinterpreted skeletal muscle contraction, lithotripsy, lead fracture, and certain types of electromagnetic interference [37]. Undersensing occurs when device function is not inhibited by a normal QRS complex or p wave, and a pacemaker spike is deployed at an inappropriate interval. In additional to device-related issues, undersensing can be caused by myocardial fibrosis, antiarrhythmic medications, and electrolyte abnormalities.

Pacemaker-induced tachycardia is often caused by a premature atrial complex that triggers a reentry dysrhythmia in a dual-chamber pacemaker. Essentially, the pacemaker circuit itself acts as an accessory pathway transmitting the atrial beat to the ventricle, with retrograde conduction through the AV node to the atria, where it is sensed by the pacemaker and conducted back to the ventricle, creating an ongoing loop. Often the tachycardia is not pronounced due to programmed limit on the rate. Treatment modalities include placing a magnet to place the pacemaker into asynchronous mode or the use of adenosine to block the AV node. If a programming switch is induced using a magnet, interrogation and reprogramming of the device will need to occur. Most newer pacemakers have algorithms to prevent and stop pacemaker-induced tachycardia [37].

In the short stay setting, care of the patient with suspected CIED programming malfunction should focus on identifying and correcting underlying metabolic abnormalities while continuing to monitor the patient. Device interrogation should occur; although feasibly performed in the ED environment by ED personnel [38], this typically requires calling in trained personnel and therefore appropriately conducted in the observation setting in a patient not obviously requiring inpatient hospitalization.

Mechanical Complications

Lead fracture and lead dislocations are infrequent causes of device malfunction and are typically present with failure to sense or failure to pace. Lead dislodgement is generally an early complication of implantation but may occur late due to chronic

inflammation at the lead placement site. Lead migration may lead to phrenic nerve stimulation, resulting in chronic hiccups, or myocardial perforation, leading to pericardial effusion and potentially tamponade [37]. While tamponade is not appropriate to be managed in the observation setting, other presentations of lead migration may be more subtle and only discovered on device interrogation.

Upper-extremity venous stenosis and thrombosis may occur due to endothelial activation from mechanical trauma. The incidence varies substantially by reported series (from 14 to 64 %) and is affected by diagnostic modality (ultrasound vs. venography) [39–41]. Superior vena cava syndrome is much less common, at 0.2 % [42]. Venous stenosis and thrombosis is often asymptomatic. Depending on the extent of the thrombus, treatment may range from watchful waiting to anticoagulation to lead removal and mechanical thrombectomy. Observation management to obtain diagnostic studies and establish the appropriate treatment regimen may be appropriate.

Infection

Infections can occur at any time after implantation, from immediate postoperative wound infection to secondary infections years after implantation. The predominant organisms, causing approximately 70–95 % of infections, are *Staphylococcus aureus* and *S. epidermidis* [43]. Much like endocarditis, presentations can be acute and overt, with fever and frank sepsis, or indolent, with chronic low-grade fevers and weight loss. Lead infections can occur without overt evidence of pocket involvement and may be quite difficult to diagnose. Transesophageal echocardiography is necessary if lead infection is suspected, as endocarditis may result, and should be performed to detect concomitant lead infection if pacer pocket infection occurs [43]. Computed tomography may be required to determine the extent of infection. Given that the diagnosis of lead infection can be challenging on a clinical basis, it would not be unreasonable to manage a patient with suspected, but not overt, device infection in the short stay setting in order to obtain appropriate imaging studies. Patients with obvious pocket infection or sepsis should, of course, be managed in the inpatient setting, as either prolonged antibiotics or device removal will likely be required.

Device Data

For most functions, CIEDs must record and process the patient's intrinsic cardiac rhythms, so that they can perform their therapeutic function. Different devices record and encode data in different fashion, although there are certain consistent elements monitored between devices and manufacturers. In addition to cardiac rhythm, rate, and device response data, there are multiple devices that record advanced data, such as heart rate variability, patient activity level, and intrathoracic

impedance. These devices were targeted for use in patients with chronic heart failure; however, heart failure and atrial fibrillation frequently coexist, and patients managed acutely for atrial fibrillation may have a device capable of monitoring advanced parameters. Regardless, both advanced and conventional data may assist with the management of the patient with atrial fibrillation.

Cardiac Rhythm

As stated above, atrial fibrillation frequently occurs concomitantly with chronic heart failure. New-onset atrial fibrillation decreases expected long-term survival, and heart failure symptoms decompensate substantially with atrial fibrillation [44]. In addition, chronic fluid overload may lead to increased atrial fibrillation, possibly due to myocyte electric instability resulting from atrial distension [14]. Silent paroxysmal atrial fibrillation can occur even in patients presumed to be rhythm controlled [45]. Identification of higher than expected rates of atrial fibrillation, or atrial fibrillation as the index cause of decompensated heart failure episodes, could lead to therapeutic strategies that might otherwise not have been selected, such as altering pacemaker programming, adding anticoagulation for stroke prophylaxis, or modifying antiarrhythmic medications.

In addition, device-based studies have demonstrated episodes of cardiac decompensation associated with both sustained and non-sustained ventricular tachycardias, similar to atrial dysrhythmias, in patients with chronic heart disease [46–48]. A finding of a high burden of ventricular dysrhythmia during evaluation and stabilization of atrial fibrillation in the short stay setting should prompt a search for electrolyte abnormalities and cardiac ischemia as provocative events. As well, if the patient's CIED does not have defibrillator functionality, the presence of ventricular dysrhythmia should lead to consultation with the patient's electrophysiologist to discuss potential reprogramming or device replacement options.

Heart Rate Variability

In the patient with predominantly sinus rhythm, there is a natural degree of variability in the intrinsic cardiac rate, both due to response to physiologic demands and circadian patterns. When cardiac stress increases, however, variance diminishes as sympathetic drive increases and parasympathetic tone decreases [49]. Improving cardiac function, such as by CRT, has been demonstrated to increase heart rate variability [50]. Therefore, in a patient undergoing treatment for atrial fibrillation and concomitant heart failure, device interrogation may provide insight as to the onset of the cardiac decompensation.

In addition, heart rate variability can serve as a predictor of adverse outcomes. In a prospective longitudinal study of patients with CRT and symptomatic heart failure with reduced ejection fraction, reduced heart rate variability was noted in patients

experiencing repeat hospitalization or death during the study period [51]. The decline in heart rate variability was noted at a median 16 days before the index event occurred. Decreased variability in ventricular response in chronic atrial fibrillation has also been associated with cardiac mortality [52]. However, other illnesses that can result in increased sympathetic tone can manifest with decreased heart failure as well, such as systemic infection [53] and chronic obstructive pulmonary disease exacerbation [54].

Patient Activity

Several devices are able to use accelerometers within the device to measure the number of hours per day that a patient is active, although these metrics do not capture the actual degree of exertion associated with the mobility of the patient. Physical activity and exercise tolerance decrease with worsening heart failure [51]. Remote monitoring has demonstrated that decreased patient activity is predictive of impending (within 1 month) episodes of heart failure decompensation, when monitored concordantly with other device parameters [55].

Intrathoracic Impedance

Intrathoracic impedance measures the resistance to an electrical pulse conducted between a pulse generator (the pacemaker lead) and a sensor, usually the device canister itself. As the amount of tissue water increases, resistance decreases. Therefore, low resistance, or intrathoracic impedance, is a marker of pulmonary fluid congestion. Impedance correlates with fluid diuresis during hospitalization as well as wedge pressures and begins to drop several days prior to hospitalization for decompensated heart failure [56]. Intrathoracic impedance has been examined as a predictor of heart failure decompensation in several studies [48, 55, 57–59]; unfortunately, to date no prospective studies have been able to successfully use outpatient impedance monitoring to avoid hospitalization. However, given the relationship between heart failure decompensation and atrial fibrillation episodes, the presence of decreased impedance may suggest the need for aggressive co-management of heart failure while managing atrial fibrillation in the short stay setting.

Device Data in the Acute Care Setting

The literature on cardiac device data has been predominantly derived from the heart failure population and has focused on managing outpatient therapies to avoid complete decompensation and subsequent ED or hospital-based care. This conceptually remains relevant to the care of the patient with atrial fibrillation, as the two conditions

frequently coexist. Unfortunately, there is little focused, prospective data examining the additive benefit of acquiring and utilizing device data in the management of atrial fibrillation in the acute care setting. There is a need for quality research examining the additive value of basic and advanced device data for the evaluation and management of the patient with an acute episode of atrial fibrillation. Until such research is established, however, it is certainly reasonable to obtain this readily available data and consider the recorded information in the context of the patient's presentation.

Conclusion

Atrial fibrillation prevalence in the population continues to increase, and it is reasonable to assume that patients with atrial fibrillation will continue to present to the ED in substantial numbers. Many of these patients will have implantable cardiac devices, which contain untapped information that could potentially assist with the diagnosis and stabilization of these patients. Further research is needed to establish optimum diagnostic thresholds and treatment strategies based on device data in the acute setting.

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Chapter 14
Pitfalls in the Acute Management of Atrial Fibrillation

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The management of atrial fibrillation in the acute care setting can require complex medical decision-making, and inappropriate care can lead to patient harm. This chapter details potential errors that should be avoided in the acute management of atrial fibrillation (AF).

Incorrect Diagnosis

The first step in delivering appropriate care to a patient with an atrial arrhythmia is proper diagnosis. This seemingly simple step can be deceptively complex. AF is a supraventricular tachycardia without organized atrial activity and with the absence of distinct P-waves as well as irregular R-to-R intervals on ECG. While ECG diagnosis of AF is straightforward in the majority of cases, certain circumstances, such as a wide QRS complex, can obscure the diagnosis. One must also consider alternative atrial rhythm disturbances such as atrial flutter (AFL) and atrial tachycardia (AT) (Table 14.1).

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Table 14.1 Key findings of common arrhythmias

Rhythm	Key findings
Atrial fibrillation	Absent P-waves, irregular rhythm
Atrial flutter	Flutter waves, QRS complexes often regular or patterned
Atrial tachycardia	Abnormal P axis/morphology, often regular
Ventricular tachycardia	Fusion beats, capture beats, AV dissociation

Irregular, Wide Complex Tachycardia

A common difficulty arises when a patient presents with an irregular wide complex tachycardia. The differential diagnosis for such an arrhythmia includes AF with underlying bundle branch block, AF with aberrant conduction, or an irregular ventricular tachycardia (VT). Obtaining a patient's prior ECG can often assist in making the correct diagnosis.

AF with wide QRS complexes due to a preexisting bundle branch block will be readily apparent if a bundle branch block with the exact QRS morphology was present on a prior ECG while in atrial fibrillation at a slower rate (Fig. 14.1) or on a prior ECG during sinus rhythm (Fig. 14.2). Careful examination of the QRS morphology can also provide a clue as to the correct diagnosis. An irregular wide QRS tachycardia with a "typical" left or right bundle branch block morphology also suggests AF with underlying bundle branch block.

AF with aberrancy also results in an irregular wide QRS rhythm. Aberration refers to intermittent, reversible block of one of the bundle branches, typically the right bundle branch, which usually has a longer refractory period than the left bundle branch. This phenomenon can exist both at high heart rates (rate-dependent aberrancy) and after a "long-short" sequence of RR intervals (the so-called Ashman phenomenon). Aberration results in QRS complexes that are wider than the narrower QRS complexes at slower rates or before the "long-short" RR sequence. "Long-short" aberration occurs because the refractory period of the bundle branches is dependent on the preceding RR interval (i.e., the longer the preceding RR interval, the longer the bundle branch refractory period). Aberration can sometimes be identified on ambulatory monitoring by examining the initiation of the wide complex rhythm (Fig. 14.3) which will show QRS widening after a "long-short" RR sequence or upon acceleration of the heart rate. Of note, rate or acceleration-dependent aberrancy occurs at faster rates during acceleration but persists down to slower rates during deceleration due to mechanisms (concealed trans-septal conduction) that tend to preserve the aberrant conduction during deceleration. Likewise, once "long-short" aberrancy occurs, the bundle branch block tends to perpetuate during tachycardia for several beats prior to resolution due to a similar mechanism.

The most concerning alternative differential diagnosis of an irregular wide QRS tachycardia is an irregular ventricular tachycardia (VT) (Fig. 14.4). Failing to make the correct diagnosis could result in inappropriate therapies or omission

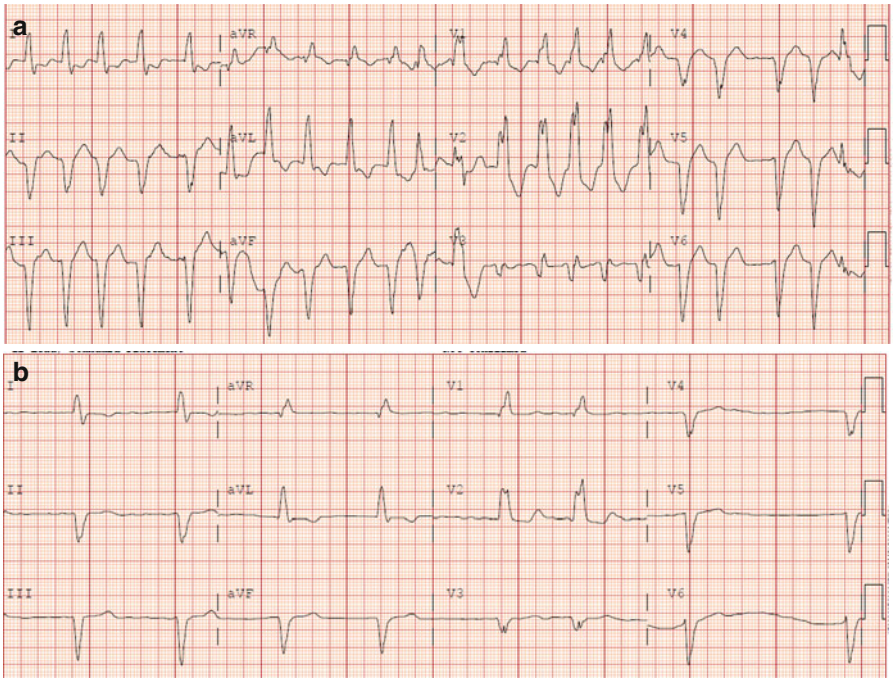

Fig. 14.1 (**a**) ECG showing atrial fibrillation with right bundle branch block (*RBBB*) and left anterior fascicular block (*LAFB*). (**b**) ECG showing atrial fibrillation with RBBB and LAFB on the same patient obtained 6 days later. AF is readily apparent when the rhythm is slowed but the bifascicular block persists, implying the conduction defect is fixed rather than due to rate-dependent or "long-short" aberrancy

of necessary therapies. VT often exhibits ECG features which should not be present in AF; these include capture beats where a sinus beat conducts to and captures the ventricle despite ongoing VT, fusion beats where a sinus beat's activation of the ventricle fuses with a VT complex, and AV dissociation where P-waves are visible and clearly independent or dissociated from the ventricular rhythm. QRS concordance across the precordium, extreme axis deviation ("northwest QRS axis"), and particularly wide QRS segments (>140 ms) are other ECG features that favor VT.

Other Supraventricular Tachycardias

Other atrial arrhythmias, such as AT and AFL, are important to distinguish from AF. While the acute management, including rate control and possibly cardioversion, may be similar, the long-term management strategies differ. As noted before, making the correct diagnosis is key to appropriate management. AT typically arises from a

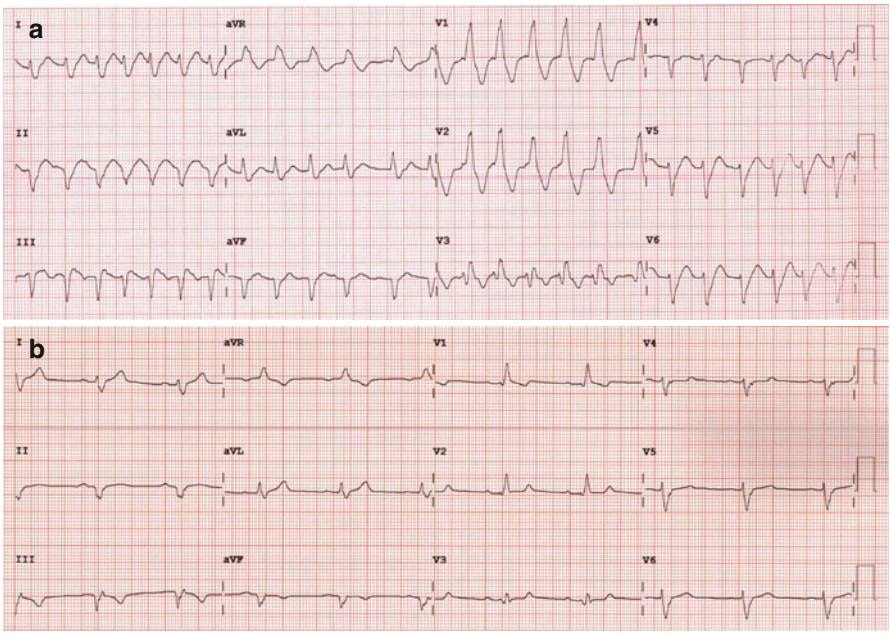

Fig. 14.2 (**a**) ECG showing atrial fibrillation with RBBB. (**b**) ECG from same patient showing normal sinus rhythm with RBBB. Again, the RBBB appears to be fixed rather than due to functional aberration

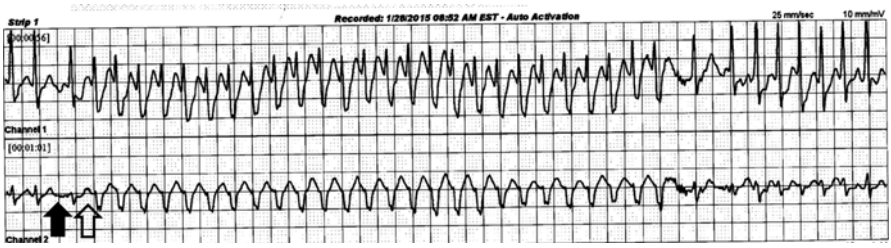

Fig. 14.3 Ambulatory event monitor tracing showing long-short initiation of aberrant ventricular conduction during atrial fibrillation. The preceding long R-to-R interval is indicated by the *black-filled arrow* and the short R-to-R interval is indicated by the *unfilled arrow*

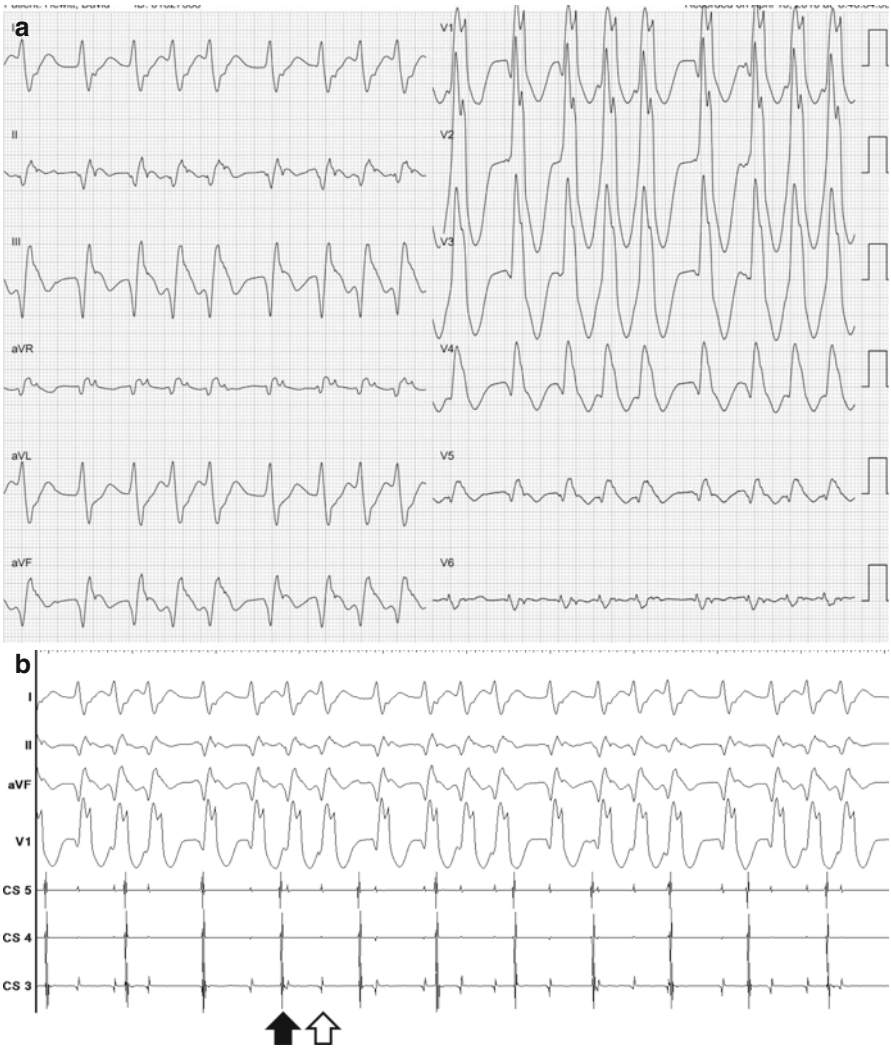

Fig. 14.4 (**a**) Irregular ventricular tachycardia. The regularly irregular pattern results from conduction (exit) block with Wenckebach periodicity in the ventricular tissue surrounding the VT focus. (**b**) Irregular VT, same patient. Intracardiac electrograms from coronary sinus catheter recordings with atrial electrograms (*black-filled arrow*) and ventricular electrograms (*unfilled arrow*) showing more ventricular depolarizations than atrial depolarizations (i.e., VA dissociation), thereby establishing the diagnosis of VT rather than AF/AFL with aberration

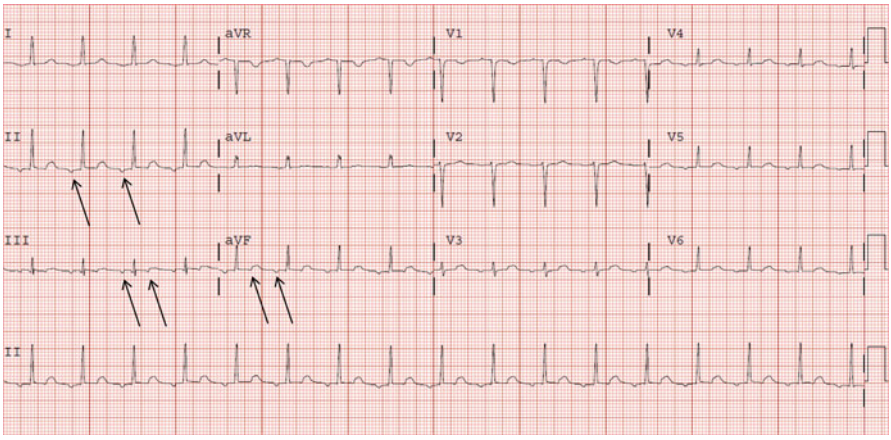

Fig. 14.5 Subtle atrial flutter with 2:1 AV conduction. The clue to the diagnosis is the inverted P-wave in the inferior leads (*black arrows*)

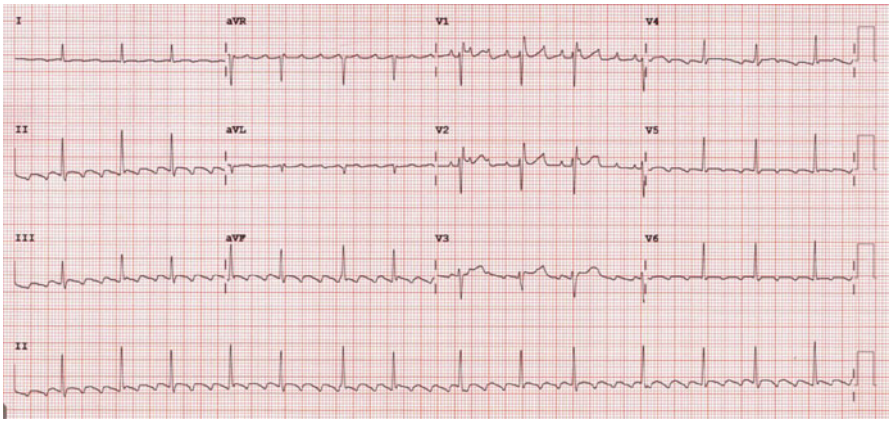

Fig. 14.6 Counterclockwise cavotricuspid isthmus-dependent "typical" atrial flutter with positive P-wave in V1 and negatively directed "sawtoothed" deflections in the inferior leads

non-sinus atrial site and has a faster rate than the sinus node, thereby suppressing normal sinus activity. As a clue to the diagnosis, the P-wave morphology is often different from the typical upright P-wave seen in lead II during sinus rhythm (Fig. 14.5).

AFL is usually a macro-reentrant rhythm (i.e., utilizing a large reentrant tachycardia circuit within the right or, less frequently, left atrium). "Typical" AFL, or more precisely counterclockwise cavotricuspid isthmus-dependent AFL, produces negative "sawtooth" flutter waves in leads II, III, and aVF and a positive deflection in lead V1 (Fig. 14.6). AFL can also arise from other locations producing flutter waves of different morphology, especially in patients after cardiac surgery or atrial ablation, where left AFL is not uncommon. The diagnosis of AFL is often straightforward if conduction to the ventricle is 3:1 or greater, allowing for easier

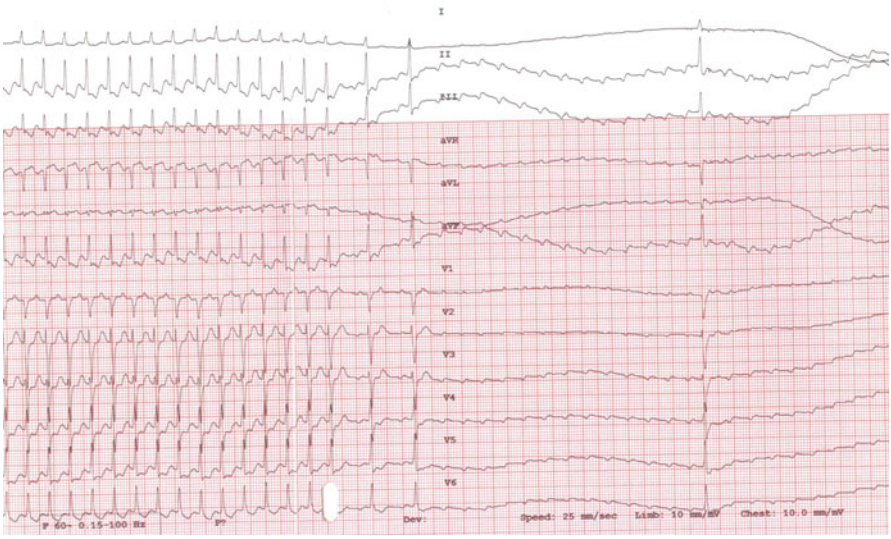

Fig. 14.7 IV adenosine administration "unmasking" the underlying rhythm of atrial flutter with 1:1 AV conduction. The 1:1 AV conduction subsequently returned a few seconds later after the adenosine was metabolized

examination of the underlying atrial activity. However, when 2:1 or 1:1 AV conduction is present, the diagnosis can be more difficult.

Diagnostic Maneuvers

Several diagnostic maneuvers can help to distinguish AF, AT, AFL, and VT. After auscultation of the carotid arteries to exclude a bruit, carotid sinus massage can produce transient slowing in AV node conduction via vagal reflex activation. The Valsalva maneuver can produce a similar effect. In the case of AF, AT, or AFL, the atrial arrhythmia will not terminate, but the ventricular response may slow and allow visualization of the underlying atrial rhythm. In most VTs, vagal activation has no effect. Adenosine (6–12 mg rapid IV bolus) has a similar effect and can be effective when sinus massage is not diagnostic (Fig. 14.7). In performing these maneuvers, obtaining a 12 lead ECG rhythm strip during the period of induced AV block is vital to making the diagnosis. After completion, written notations should be added to printed tracings indicating when the drug was administered or when carotid massage was performed so that the correlating change in the rhythm is properly documented.

Adenosine is metabolized by erythrocytes and vascular endothelial cells and has a plasma half-life of <10 s. AV block will be transient, lasting only seconds, and

non-adenosine-sensitive AT and atrial flutter will persist with a subsequent return of AV conduction to the pre-adenosine baseline state. On the other hand, reentrant supraventricular tachycardias that are dependent on AV node conduction (such as AV nodal reentry and AV reentry tachycardias) will terminate suddenly with adenosine administration. While adenosine is generally safe, the provider should have the patient lying supine on the bed and connected to an external defibrillator with continuous ECG monitoring. Providers should warn the patient to expect an unusual sensation (chest fullness and shortness of breath) and should be prepared to deliver external defibrillation and advanced cardiac life support in the rare cases where the underlying arrhythmia degenerates into a more unstable rhythm such as ventricular fibrillation (VF).

Failure to Appreciate the Importance of Duration of AF Episodes

Atrial fibrillation is commonly classified according to the duration of arrhythmia episode (Table 14.2). These classifications are relevant to management and affect treatment recommendations.

In patients with paroxysmal atrial fibrillation, each episode usually spontaneously converts to sinus rhythm within several hours. Electrical cardioversion is typically not necessary and should not be considered so long as the patient is stable. Conversion to sinus rhythm may be hastened by treating any arrhythmia triggers or exacerbating factors, such as infection or hypoxemia. Treatment with AV nodal blocking drugs to slow the ventricular rate is appropriate.

In patients with long-standing persistent atrial fibrillation, especially in the setting of left atrial enlargement or other significant underlying structural heart disease, rhythm control may be more difficult to achieve. In these instances cardioversion may fail to convert the patient to sinus rhythm, or the patient may convert to sinus rhythm for only a short period of time (occasionally only a few beats) before having return of atrial fibrillation. Elective cardioversion and attempts to maintain sinus rhythm should be undertaken in consultation with cardiology or clinical cardiac electrophysiology. This is especially true for recommendations regarding initiation of appropriate antiarrhythmic drug therapy.

Table 14.2 Atrial fibrillation classification

Recently diagnosed AF	Not yet clear into which category AF will be classified
Paroxysmal AF	Terminates spontaneously or with intervention within 7 days of onset
Persistent AF	AF that is sustained >7 days
Long-standing persistent AF	AF>12 months in duration
Permanent AF	AF with a decision by patient and clinician to make no further attempts to restore or maintain sinus rhythm

AF atrial fibrillation

Selecting Rate Versus Rhythm Control

In the urgent setting, any patient who is unstable (hemodynamically, respiratory, or otherwise) and it is felt that their clinical status will be improved by restoration of sinus rhythm should be urgently or emergently cardioverted. For all clinically stable patients, the decision to pursue rate vs. rhythm control is more nuanced. Making an appropriate decision is critical to delivering optimal care to the patient with AF.

The AFFIRM trial was a multicenter trial of over 4000 patients with AF that randomized patients over age 65 or with other risk factors for stroke or death to either a rate control or rhythm control strategy [1]. This study showed no survival advantage to a rhythm control strategy over a rate-control strategy. As such, a rate-control strategy is often the primary strategy in patients with AF who are asymptomatic and whose heart rate is well controlled without drug therapy or on AV nodal blocking drug therapy. Cardioversion in asymptomatic patients with normal heart rates is often not recommended. Application of data from the AFFIRM trial is limited to patients over age 65 or who had other risk factors for stroke or death and in whom the investigators felt AF was likely to be recurrent. In addition, a majority of patient's in the trial had left atrial enlargement and a significant proportion had left ventricular systolic dysfunction, and patients with highly symptomatic AF were excluded. As such, younger patients, regardless of symptoms or heart rate control, and especially those with newly diagnosed atrial fibrillation and without structural heart disease, are appropriate candidates for consideration of a rhythm control strategy.

Target Heart Rate (HR)

Aggressive attempts to control heart rate in patients with AF can result in adverse effects. With the exception of digoxin, the commonly used AV nodal blocking drugs (beta-blockers and the non-dihydropyridine calcium blockers) lower the blood pressure and can result in medication-related hypotension. This is especially common in the elderly and can place patients at risk for adverse events. Lenient rate control (target HR < 110 bpm) is not associated with increased mortality or morbidity over a relatively short follow-up in patients with AF and is more easily achieved than strict rate control (target HR < 80 bpm) [2]. Current guidelines suggest that a lenient rate-control strategy with a resting HR < 110 bpm may be reasonable as long as the patient is asymptomatic and LV systolic function is preserved [3].

Appropriate Medication for HR Control

IV agents are often useful for initial rate slowing of AF with rapid ventricular rates. Bolus IV drugs (such as diltiazem or metoprolol) have a rapid onset of action, but occasionally the rapid offset can lead to some difficulty in rate-control stability.

While continuous infusions (e.g., diltiazem or esmolol) can be helpful, one should also consider initiating oral rate-control medication concurrent with IV therapy as blood pressure allows.

In patients with known LV systolic dysfunction, beta-blockers should be used preferentially for attempts at HR control in AF. Due to negative inotropic effects, non-dihydropyridine calcium channel blocking drugs should not be prescribed to patients with decompensated heart failure. When patients present with decompensated LV dysfunction, rapid AF, and borderline hypotension, management may be especially challenging, and cardiology consultation is recommended.

Compared with beta-blockers or calcium channel blockers, digoxin has the benefit of having no hypotensive or negative inotropic effects. Digoxin can be used in combination with beta-blockers or calcium channel blockers. The use of IV digoxin is limited by an onset of action > 1 h, and it does not reach peak effect until approximately 6 h. Importantly, digoxin should be avoided in patients who either have or are at high risk for developing renal insufficiency. Caution is advised when starting digoxin in elderly patients, especially elderly female patients, and patients already receiving amiodarone. Of note, there have been recent studies indicating the use of digoxin is associated with increased mortality [4–7].

Systemic Anticoagulation

AF increases the risk of thromboembolic stroke [8–11]. The severity of functional deficits associated with AF-related cardio-embolic stroke is greater than for non-AF stroke [12–16]. Incorrect management of systemic anticoagulation is also a well-recognized area of potential medical-legal risk [17, 18]. For these reasons it is imperative that the acute care provider have an understanding of the appropriate use of anticoagulation in AF. Improper management can be associated with potential patient harm, and the following pitfalls should be avoided.

Cardioversion Without Appropriate Prior Systemic Anticoagulation

When considering non-emergent DC cardioversion (DCCV), one needs to assess the duration of the ongoing AF episode and prior anticoagulation. If the duration of AF or AFL is unquestionably under 48 h, DCCV can be safely performed. If the duration is at least 48 h or unknown, non-emergent DCCV should only be performed if the patient has been adequately anticoagulated for at least 3 weeks, or if transesophageal echocardiography (TEE) has excluded intracardiac thrombus prior to DCCV [3, 19].

Failure to Confirm Therapeutic Systemic Anticoagulation for the Prior 3 Weeks

If a patient has been on warfarin, INR values should be reviewed prior to proceeding with DCCV. The number of documented therapeutic INR values necessary to proceed with DCCV is a clinical decision left to the evaluating practitioner, but a patient's present INR value as well as his or her history of stable or labile INR values and warfarin dosing can help to guide this decision. If a patient is taking one of the new target-specific oral anticoagulants (OAC) (dabigatran, rivaroxaban, apixaban, or edoxaban), then the practitioner should confirm that the patient has been taking the medication without interruption or missed doses in the preceding 3 weeks and should document the conversation in the medical record prior to proceeding with DCCV.

Failure to Consider Post-procedure Anticoagulation Prior to TEE/DCCV

When AF or AFL duration is >48 h, structural changes occur at the cellular level resulting in atrial stunning and weakened atrial contraction despite sinus rhythm post-cardioversion [20]. For this reason it is recommended that the patient with AF or AFL >48 h duration prior to TEE/DCCV, even in the absence of thrombus at TEE, be anticoagulated for a minimum of 4 weeks post-cardioversion [3, 19]. In fact, it is recommended that the patient be therapeutically anticoagulated (not subtherapeutic) at the time of the TEE/DCCV procedure and receive bridging anticoagulation (e.g., subcutaneous enoxaparin until INR is therapeutic on warfarin), if necessary, to ensure uninterrupted systemic anticoagulation for the recommended minimum 4 weeks. The need for systemic anticoagulation post-cardioversion should be considered prior to TEE/DCCV; if the patient has a contraindication to systemic anticoagulation, then the increased risk of thromboembolic stroke post-cardioversion should be discussed with the patient and that discussion documented in the medical record.

Incorrect Assumption that Symptom Onset Is Equivalent to Arrhythmia Onset

Documentation of the time of onset of AF or AFL is critical to determine the need for 3 weeks of systemic anticoagulation or TEE prior to DCCV, as well as the need for systemic anticoagulation post-cardioversion. In patients with a pacemaker, ICD, implantable loop recorder or in whom telemetry monitoring or ambulatory ECG monitoring was ongoing at the time of reported arrhythmia onset, the timing can

usually be established with some degree of certainty. However, the subjective report of patient symptoms as a surrogate for timing of arrhythmia onset should be taken with caution, especially in the patient with vague symptoms such as fatigue, mild palpitations, or mild dyspnea. Although the practitioner must use clinical judgment, it is often best to err on the side of assuming arrhythmia duration of >48 h unless clear documentation of onset is available.

Proceeding Immediately to TEE/DCCV for Minimally Symptomatic Rate-Controlled AF

In the AF patient with minimal symptoms attributable to arrhythmia and in whom ventricular rates are well controlled, a strategy of initiating systemic anticoagulation and then performing DCCV after 3 weeks of anticoagulation should be considered. This strategy will obviate the need for and any procedural risks associated with TEE. In addition, placing the patient on systemic anticoagulation prior to DCCV can serve the purpose of ensuring that he or she tolerates anticoagulation, which needs to be continued at minimum 4 weeks post-cardioversion.

Failure to Risk Stratify Patient for Thromboembolic Stroke and Hold Appropriate Discussion Regarding Initiation of Systemic Anticoagulation

For long-term anticoagulation decisions in all patients with AF or AFL, regardless of AF duration, AF burden, or decision regarding rate vs. rhythm control strategy, risk stratification for thromboembolic stroke should be performed. The estimated risk should be discussed with the patient and documented. Decisions regarding antithrombotic therapy should be individualized and take into account absolute risks, relative risks of stroke and bleeding, and the patient's values and preferences [3, 21]. For patients with nonvalvular AF, the CHA_2DS_2VASc score (Table 14.3)

Table 14.3 CHA_2DS_2-VASc score

Stroke risk factors	Score
*C*ongestive heart failure/LV dysfunction	1
*H*ypertension	1
*A*ge ≥ 75 years	2
*D*iabetes mellitus	1
*S*troke/TIA/thromboembolism	2
*V*ascular disease (CAD/MI, PAD)	1
*A*ge 65–74 years	1
*S*ex category (female gender)	1

should be used to estimate stroke risk [3, 21–24]. In men with a CHA_2DS_2VASc score of 0 or women with a score of 1 (female gender is the lone risk factor), it is recommended to omit long-term antithrombotic therapy. In patients with a CHA_2DS_2VASc score of ≥ 2, systemic anticoagulation is recommended unless a contraindication exists [3, 21]. In men with a CHA_2DS_2VASc score of 1, treatment with aspirin alone, oral anticoagulant therapy, or no antithrombotic therapy are all reasonable options [3].

Failure to Start Systemic Anticoagulation Because of AF Duration or AF Burden

In the patient with clinically apparent AF, risk stratification for thromboembolic stroke, and initiation of systemic anticoagulation if appropriate, is recommended irrespective of AF duration or AF burden. Analysis of patients treated with aspirin in the Stroke Prevention in Atrial Fibrillation (SPAF) studies demonstrated that those with intermittent AF had stroke rates similar to patients with sustained AF and similar stroke risk factors [25]. Studies and current clinical guidelines support that patients with paroxysmal or persistent AF should be treated the same in regard to systemic anticoagulation. Anticoagulation should not be withheld in the patient with clinical AF of only short durations or patients with low AF burden.

Failure to Start Systemic Anticoagulation Because of an Elevated Estimated Bleeding Risk

Bleeding risk is an important variable to consider when weighing the risks and benefits of systemic anticoagulation, and the HAS-BLED score (Table 14.4) may help define patients at elevated bleeding risk [26]. A score of >3 may identify patients at increased risk of bleeding on systemic anticoagulation and such

Table 14.4 HAS-BLED score

Bleeding risk factor	Score
*H*ypertension	1
*A*bnormal renal/liver function (1 point each)	1 or 2
*S*troke	1
*B*leeding tendency or predisposition	1
*L*abile INRs (only applies if on warfarin)	1
*E*lderly (age >65)	1
*D*rugs (aspirin, NSAIDs, etc.) or alcohol abuse (1 point each)	1 or 2

patients may benefit from closer observation for bleeding complications, and in patients on warfarin, closer monitoring of INR levels [3]. Bleeding risk score estimates should be part of shared decision-making and an informed discussion of the potential risks and benefits of systemic anticoagulation therapy. This scoring system can also be used to identify and address modifiable risk factors (e.g., NSAID therapy).

In comparing thromboembolic risk estimates per the CHA_2DS_2VASc score and bleeding risks per the HAS-BLED score, the practitioner will notice that there is overlap. For instance, advanced age, HTN, and stroke are risk factors for both scoring systems. Importantly, a high bleeding risk score is not always grounds to forego anticoagulation. Not all bleeds are equivalent. Intracranial bleeding is rare but devastating. Gastrointestinal bleeding is more common than intracranial bleeding, transiently disabling and uncomfortable, but death is uncommon. In contrast, death and serious permanent disability are common results of cardio-embolic stroke. AF-associated thromboembolic stroke is associated with increased morbidity, diminished residual functional status, and increased mortality compared to non-AF stroke [12–16]. Although current bleeding risk scores do not account for these differences in complication severity, such differences should be considered by the astute practitioner when weighing the risks and benefits of systemic anticoagulation.

Not Initiating Anticoagulation Because a Patient Is Elderly or Is a "Fall Risk"

Advanced age is a prominent risk factor for AF-associated thromboembolic events [27], hence its inclusion in stroke risk score estimates [22, 27]. As such, elderly patients are one of the groups that stand to benefit most from systemic anticoagulation [28]. Paradoxically, literature suggests that the elderly are less likely to be started on systemic anticoagulation than their younger counterparts [29]. Given the high risk of thromboembolic events in the elderly, withholding systemic anticoagulation simply because of age is inappropriate. Like others, elderly patients should be risk stratified for thromboembolic stroke, and the decision regarding antithrombotic therapy should be made based on individual risks and benefits and taking into account patient and family values and preferences.

A history of prior falls or perception of elevated fall risk often results in a practitioner withholding systemic anticoagulation. In many instances, this may be inappropriate. Even though a history of falls is associated with an elevated risk of bleeding, these elderly patients also have an increased risk of stroke/thromboembolism and all-cause mortality [30, 31]. Given their elevated stroke risk, patients with AF at high risk of falls may benefit from anticoagulant therapy if they have multiple stroke risk factors [30, 31].

Failure to Consider Warfarin Versus Target-Specific Oral Anticoagulants (OAC)

Warfarin, a vitamin K antagonist, has decades of strong evidence supporting decreased thromboembolic risk in patients with nonvalvular AF [32]. However, warfarin dosage is often very difficult to stabilize in individual patients, requiring frequent INR monitoring. Warfarin has interactions with multiple foods (such as green vegetables) and drugs (such as amiodarone and many antibiotics). For sufficient stroke protection, time in therapeutic INR range on warfarin needs to be >70 % [33], and achieving this target can be difficult [34]. The thrombin inhibitor dabigatran and the factor Xa inhibitors rivaroxaban, apixaban, and edoxaban have all been shown to be non-inferior to warfarin for thromboembolic prophylaxis, have acceptable bleeding risk profiles, and are now FDA approved for thromboembolic stroke prophylaxis in nonvalvular AF [35–38]. The decision between a vitamin K antagonist and target-specific OAC should be shared with the patient, including a review of the benefits and drawbacks for each agent. The patient and provider can then make a decision based on patient risk combined with patient values and preferences.

Generally speaking, factors that favor target-specific OAC use over warfarin include difficulty achieving sufficient time in therapeutic INR range on warfarin, patient-specific drug-drug interactions on warfarin, patient preference for decreased food-drug interactions on target-specific OAC agents, and decreased need for follow-up visits. While the frequency of follow-up visits for anticoagulation monitoring is significantly reduced with target-specific OAC agents vs. warfarin, outpatient follow-up to ensure drug tolerance and patient safety is still required, especially during the initiation period. Complete patient inability or willingness to follow up for any subsequent visits is a contraindication to target-specific OAC therapy, as it is for warfarin.

Treatment with a target-specific OAC drug is not indicated for valvular atrial fibrillation, defined as AF in the presence of mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair [3]. Patients in whom warfarin may be preferred over target-specific OAC therapy include those with significantly reduced renal function and patients with an inability to afford the more expensive target-specific OAC medications. A FDA-approved reversal agent idarucizumab (Praxbind®) for dabigatran is now available. The lack of reversal agent at present for other target-specific OAC medications should be taken into account when considering the risks of anticoagulation with these agents, especially in patients with elevated risk of bleeding, and until antidotes are available, this drawback should be discussed explicitly with patients and documented in the patient record. Once reversal agents for target-specific OAC drugs are readily available, prescribing them to the AF patient with an elevated risk of bleeding may become less of a concern. The individual medications should not be used interchangeably. The four available target-specific OAC medications have subtle differences with which providers should become familiar before routinely using them in clinical practice (Table 14.5).

Table 14.5 Target-specific oral anticoagulant medications

Drug	Dabigatran	Rivaroxaban	Apixaban	Edoxaban
Target	IIa (thrombin)	Xa	Xa	Xa
Dose	150 mg, 75 mg	20 mg, 15 mg	5 mg, 2.5 mg	60 mg, 30 mg
Dosing regimen	Twice daily	Once daily	Twice daily	Once daily
Renal dosing	CrCl 15–30 mL/min: 75 mg BID	CrCl 15–30 mL/min: 15 mg daily	If ≥ 2: Cr > 1.5 mg/dL, > 80 years old, or < 60 kg, then 2.5 mg BID	CrCl > 95 mL/min: contraindicated CrCl 15–50 mL/min: 30 mg daily
Renal clearance	80 %	33 %	27 %	50 %

Failure to Recognize Special Populations of Valvular AF and Hypertrophic Obstructive Cardiomyopathy (HOCM)

The CHA_2DS_2VASc scoring system discussed previously only applies to patients with nonvalvular AF. Valvular AF is associated with a significantly increased risk of thromboembolic events. Patients with AF along with rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair should receive systemic anticoagulation regardless of other stroke risk factors. In addition, current guidelines recognize patients with HOCM as a unique group with elevated thromboembolic stroke risk and recommend that, in the absence of contraindication, all patients with HOCM and AF receive systemic anticoagulation regardless of the presence or absence of other stroke risk factors [3].

Failure to Consider Systemic Anticoagulation in the Patient Status Post Left Atrial Appendectomy, Left Atrial Appendage (LAA) Occlusion, or LAA Ligation

Methods of decreasing thromboembolic risk in AF are rapidly evolving beyond anticoagulant medications. Surgical left atrial appendectomy has been available for some time, and endocardial LAA occlusion with the Watchman™ device, thoracoscopic placement of the Atriclip™ LAA occlusion device, and epicardial LAA ligation with the Lariat™ device are all now available. Providers will likely encounter an increasing number of patients that have had either surgical or device-associated LAA occlusion, ligation, or appendectomy. These devices and interventions, however, do not preclude the possibility of ischemic stroke. Patients who received the Watchman™ device within the PREVAIL study still had a 1.9 % risk of ischemic stroke [39]. Several possible reasons for persistent stroke risk have been postulated.

TEE performed after surgical left atrial appendectomy has demonstrated incomplete LAA ligation in roughly one-third of patients [40], which could be a mechanical nidus for thromboembolism. Thrombus formation may occur in areas of the heart outside of the LAA [41]. Separate from mechanical effects of the LAA, inflammatory factors and/or abnormal hemostatic and endothelial function may contribute to thromboembolism in AF [42]. While mechanical LAA occlusion or ligation lowers the risk of AF-associated thromboembolism and likely swings the balance in favor of no systemic anticoagulation, they do not abolish thromboembolic risk completely. Future studies will hopefully help guide clinical decision-making in these populations.

Failure to Arrange for Adequate Follow-Up

Any patient treated for AF in the acute care setting needs close outpatient follow-up after discharge. Stable, rate-controlled, asymptomatic, or minimally symptomatic patients may be managed with close outpatient follow-up, at which time anticoagulation can be initiated by the clinic provider. If a target-specific OAC drug is chosen, it can be initiated immediately. With warfarin, it may be preferable to have the patient establish with the anticoagulation clinic that will follow the patient prior to initiation of anticoagulation.

The error to avoid is not documenting the discussion and decision to start systemic anticoagulation or not ensuring that the patient is given a prompt appointment in a primary care/anticoagulation clinic. Upon initiation of systemic anticoagulation with warfarin such that INR measurement and dose titration are necessary, follow-up should ideally be within 72 h of discharge. If anticoagulated patients are treated for non-AF conditions (such as infections), they should also have prompt follow-up with their anticoagulation provider because of drug-drug interactions that may arise. Separate from anticoagulation, the AF patient in whom a rhythm control strategy is being pursued should have close follow-up arranged with a cardiology or electrophysiology provider. The patient with asymptomatic rate-controlled AF in whom anticoagulation is sufficiently addressed may follow-up with their primary care provider at an appropriate interval.

Inappropriate Discontinuation of Systemic Anticoagulation

In the patient already receiving systemic anticoagulation for AF who is seen in an acute care setting for reasons other than arrhythmia, systemic anticoagulation should be continued unless a specific contraindication (planned procedure, active bleeding) exists. For necessary procedures, in patients without prior CVA and with an acceptably low CHA_2DS_2VASc risk score, systemic anticoagulation can usually

be safely held for a short period and then restarted once safe from a post-procedural standpoint. Patients with DCCV out of persistent AF within the past 4 weeks who are in sinus rhythm and patients within 2 months of recent AF ablation should not have interruption of systemic anticoagulation without first weighing the increased risk of thromboembolic CVA in this patient population.

After AF ablation, guidelines recommend anticoagulation for a minimum of 2 months. Thereafter, long-term anticoagulation should be based on the patient's CHA_2DS_2VASc score, even if the patient is in sinus rhythm and doing well.

Pharmacologic Cardioversion

Initiation of antiarrhythmic drug (AAD) therapy should be viewed as an acute intervention with the intent to achieve rhythm control. Failure to recognize the risks associated with initiation of AAD therapy can potentially result in patient harm.

Failure to Consider Systemic Anticoagulation Prior to Initiating AAD Therapy

Thromboembolic risk associated with pharmacologic cardioversion from AAD therapy is comparable to that with DCCV. As such, the provider initiating AAD therapy is responsible for ensuring appropriate systemic anticoagulation, as detailed above, before and after conversion to sinus rhythm in patients with AF of over 48 h duration.

Choosing the Inappropriate AAD

When starting a patient with AF on AAD therapy, it is important to choose the appropriate drug. In a non-elderly patient without structural heart disease, conduction system disease, or coronary artery disease (CAD), a class IC agent (flecainide or propafenone) is often the first drug of choice. Class IC AAD therapy is contraindicated in the presence of left ventricular dysfunction or CAD with past MI [43], and many providers avoid this drug class in the presence of any degree of coronary obstruction, any structural heart disease, and in the elderly. In addition, class IC AAD agents can potentially slow atrial flutter and promote 1:1 conduction, resulting in rapid ventricular heart rates. As such, these agents should be prescribed along with an AV nodal blocking drug (beta-blocker or calcium channel blocker).

The class III AAD therapy agents (sotalol and dofetilide) prolong the action potential duration and are commonly used by cardiology and electrophysiology practitioners to treat atrial fibrillation. In a small subset of patients, these medications can be pro-arrhythmic (most commonly a polymorphic VT termed torsades de pointes), and monitoring of the QT interval is required during initiation. Inpatient admission for ECG monitoring is required for dofetilide initiation and recommended by the FDA for sotalol initiation as well. These drugs prolong the QT interval and, especially if combined with other drugs that have the same effect, can potentially lead to dangerous arrhythmias. Therefore, drug-drug interactions must be carefully considered when prescribing these medications. The decision to treat with dofetilide or sotalol should be done in conjunction with a consulting cardiologist or cardiac electrophysiologist.

Amiodarone is a powerful and effective AAD that exerts its effect by prolonging action potential duration, slowing conduction, blocking beta and calcium receptors, and a variety of other actions. Amiodarone is associated with multiple adverse effects, including but not limited to pulmonary, thyroid, hepatic, ocular, skin, and neurotoxicity. The adverse effects of amiodarone are usually associated with chronic therapy, after months to years of drug administration; however, acute toxicity does occur. Alternative AAD therapies should be considered, if available, in patients with underlying lung, liver, or thyroid disease. Consultation of cardiology or clinical cardiac electrophysiology prior to initiation of amiodarone is at the discretion of the acute care provider.

Close outpatient follow-up needs to be arranged following AAD therapy initiation. Ongoing surveillance for the development of structural heart disease is needed in patients receiving class IC AAD therapy. Patients receiving dofetilide or sotalol require periodic monitoring of the QT interval, renal function, electrolytes, and medications for surveillance of drug-drug interactions. Patients receiving amiodarone require liver and thyroid profiles every 6 months and a chest X-ray at least yearly. Pulmonary function tests should be performed upon drug initiation and repeated if pulmonary symptoms arise, and routine ophthalmologic exams should be performed.

Attempting Acute Pharmacologic Cardioversion with Ibutilide Without Appropriate Monitoring

In select cases, intravenous ibutilide can be given for acute pharmacologic cardioversion of AF. Ibutilide prolongs the QT interval and can cause torsades de pointes, and thus continuous ECG monitoring is required during drug administration and for a minimum of 4 h thereafter. Ibutilide should not be given to patients already receiving drugs that prolong the QT interval. Electrolytes should be checked and potassium repleted to >4 mmol/L and magnesium to >2 mg/dL prior to administering the drug. Some providers give 1–2 g of IV magnesium sulfate empirically prior to

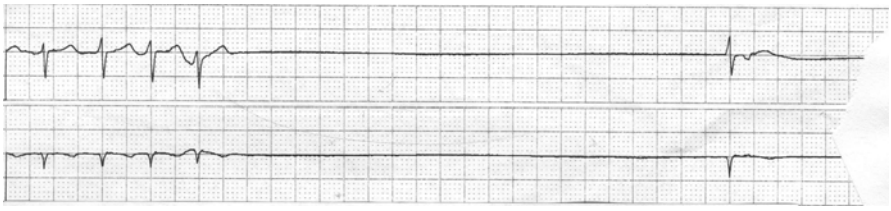

Fig. 14.8 Conversion pause at the termination of atrial fibrillation indicates sinus node dysfunction (the so-called tachy-brady syndrome)

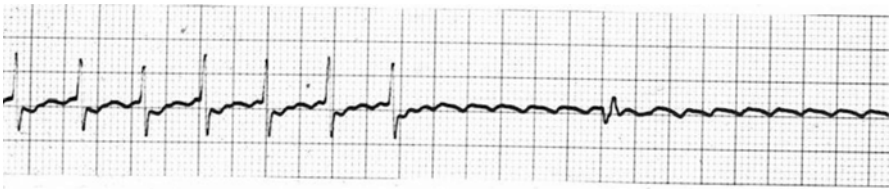

Fig. 14.9 Pause during AF indicating AV node dysfunction

ibutilide administration regardless of the baseline magnesium level. A code cart with an external defibrillator should be readily available due to the risk of VF.

Electrical Direct Current Cardioversion (DCCV)

Failure of Pre-procedure Preparations

Once an accurate diagnosis of AF or AFL has been made, therapeutic systemic anticoagulation has been initiated for the requisite period of time, and the decision is made to proceed with DCCV, the following considerations can help to ensure a safe and successful procedure.

First, the practitioner considering DCCV should anticipate possible complications that may arise and be prepared to care for the patient. For instance, if the practitioner is not aware of the patient's underlying sinus node function, he or she should be prepared for possible bradycardia post-cardioversion. Conversion pauses on termination of atrial fibrillation are not rare (Fig. 14.8) and are indicative of sinus node dysfunction. Separately, a pause during AF or AFL indicates AV nodal disease (Fig. 14.9). Importantly, if AF is associated with perfectly regular QRS complexes and a slow rate, the patient should be suspected of having AF with complete AV block and a junctional escape rhythm (Fig. 14.10). Post-cardioversion bradycardia is not uncommon, especially in patients receiving high doses of AV nodal blocking agents, and practitioners need to be familiar with their external defibrillator device and know how to rapidly switch the device to a transthoracic pacing mode should

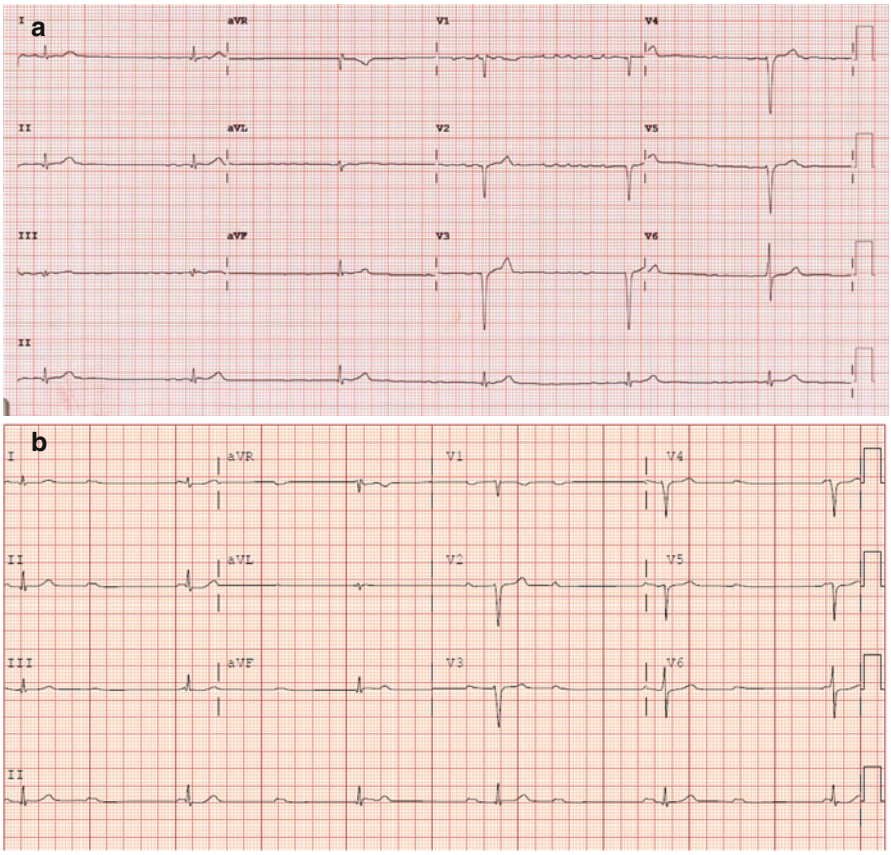

Fig. 14.10 (**a**) Atrial fibrillation with a regular, bradycardic ventricular rhythm suggesting complete AV conduction block and a junctional escape rhythm. (**b**) Normal sinus rhythm with high-grade AV block in the same patient, now post-DCCV

that become necessary. If a patient presents for elective cardioversion with a supratherapeutic INR and post-cardioversion bradycardia is a concern, then it may be appropriate to postpone the procedure so that if urgent or emergent invasive (temporary or permanent) pacing is required, it can be done safely and without prohibitive bleeding risks.

Next, it is requisite to have the appropriate equipment for patient monitoring, as well as emergency equipment should complications occur. Intravenous access for medication administration should be established pre-procedure. Monitoring equipment including sphygmomanometry, continuous pulse oximetry, and cardiac telemetry should be present. Airway management tools including supplemental oxygen, a suction device, and intubation supplies should be in the room and available. Finally, an external defibrillator is needed and a code cart with resuscitation medications should be available.

Consideration should be given as to whom will provide sedation for the procedure. At minimum, moderate sedation should be administered to ensure patient comfort, and every effort should be made to not electrically cardiovert an awake patient. The specific regulations regarding which medications can be safely administered by a cardiology practitioner or an emergency medicine practitioner and which medications are restricted to anesthesia providers are typically dictated by the rules and privileges of individual facilities.

If TEE is necessary prior to the procedure and is planned to be performed in the procedure room at the time of cardioversion, then the room will need to be arranged such that the TEE machine can be placed in an appropriate location. Most commonly an echocardiographer will position himself or herself at the head of the left side of the bed with the patient in the left lateral decubitus position for the procedure. Importantly the patient bed, TEE machine, and external defibrillator/code cart need to be positioned such that access to the patient's airway and IV sites is maintained, and providers can access the patient to deliver advanced cardiac life support measures should they be required.

Intraprocedural Error: Failure of Appropriate Synchronization

For cardioversion purposes, the external defibrillator shock should be synchronized to the QRS complex to avoid VF due to a shock on a T wave. When properly set to synchronized cardioversion, the device should indicate such with markers synchronized to the QRS complexes. The provider performing the cardioversion procedure needs to review the ECG tracing on the external defibrillator to ensure that the device is appropriately identifying and synchronizing on the QRS complex prior to charging and delivering cardioversion energy. If the synchronization markers are absent or not appropriately located on the QRS complexes, then device synchronization can be improved by selecting a different lead. Rarely, the external defibrillator device fails to appropriately synchronize to the QRS complex and the shock results in VF. Prompt evaluation of post-DCCV rhythm is crucial. If VF occurs, immediate non-synchronized defibrillation should be performed. Providers should be aware as to the default setting of their device after a shock is delivered; in some cases, the device must be "unsynced" in order to shock VF after a synchronized DCCV.

Failure to Distinguish Failure to Cardiovert Versus Early Return of Atrial Fibrillation (ERAF)

After cardioversion, AF may recur after as few as 1–2 sinus beats. ERAF must be distinguished from failure to cardiovert, when no sinus beats are observed. Failure to cardiovert (without sinus rhythm for even 1–2 beats) implies a failure to deliver sufficient energy to overcome the defibrillation threshold of the atria. This can be

addressed by maneuvers to deliver increased energy during cardioversion, including taking a towel and pressing down on the anterior defibrillation patch (to decrease thoracic diameter, increase electrode contact with the skin, and decrease transthoracic impedance). In addition, if cardioversion is initially attempted with electrode patches in the anterior-lateral position, then electrodes should be repositioned to the anteroposterior position. The anteroposterior electrode position provides a superior vector of energy delivery and higher cardioversion success rates [44–46]. If these methods of increased energy delivery fail, then consideration can be given to administration of ibutilide, which decreases the atrial defibrillation threshold, followed by repeat cardioversion if necessary [47]. Particularly refractory cases may require intracardiac catheter cardioversion.

If there is successful cardioversion followed by ERAF, then sufficient energy was delivered to overcome the atrial defibrillation threshold and terminate AF. In this instance, a repeat cardioversion may be tried once, but multiple cardioversions and/or higher energies are unlikely to be effective. This patient may benefit from antiarrhythmic drug therapy to aid in maintaining sinus rhythm. After AAD initiation, repeat cardioversion should be performed if AF/AFL persists. ECG strips from cardioversion attempts should be saved to the patient chart so that, if consulted, an advanced cardiology practitioner can review the strips, make the distinction between failure to cardiovert and ERAF, and make appropriate recommendations for further care.

Incorrect Management of Pacemaker or Internal Cardioverter-Defibrillator (ICD) Devices During Cardioversion

If cardioversion of a patient with an ICD is planned, external transthoracic cardioversion is preferred over internal cardioversion utilizing the ICD. This is because the RV coil of an ICD is positioned in the ventricle, not across the atrium, and the shocking vector obtained with external anterior-posterior electrode placement is often superior to vectors that can be obtained with cardioversion via the patient's ICD. Secondly, shocks from the ICD can deplete its battery.

In a patient with a permanent pacemaker or ICD, external cardioversion patches should not be placed directly over the device generator. The device generator should be kept out of the external cardioversion shock vector. The device should be checked before and after cardioversion to ensure stable lead impedances, sensing, pacing thresholds, and battery status.

Inadequate Post-procedure Monitoring

Multiple factors influence the appropriate length of post-procedure monitoring after DCCV. Most notably, these factors include the level of sedation administered and any requisite ECG or telemetry monitoring required if potentially pro-arrhythmic

drugs are administered or initiated. The patient needs to be able to ambulate and ingest food safely prior to discharge and should be instructed not to drive or operate machinery until the next day. Furthermore, appropriate anticoagulation needs to be ensured and appropriate anticoagulation follow-up established prior to patient discharge.

Special Circumstances

Failure to Recognize and Appropriately Treat Pre-excited Atrial Fibrillation

AF/AFL occurring in the presence of an anterogradely conducting accessory pathway (AP) (pre-excitation syndrome, Wolff-Parkinson-White syndrome) is a particularly dangerous arrhythmia that can be extremely rapid and has the potential to degenerate into VF. It is important that the acute care practitioner recognizes ECG and telemetry tracings of pre-excited AF: irregular rhythm with wide QRS complexes having varying widths and morphologies (Fig. 14.11). Importantly, the QRS axis remains stable in pre-excited AF using a single AP for anterograde AV conduction, which helps differentiate it from catecholaminergic polymorphic ventricular tachycardia (CPVT), characterized by constantly varying QRS axes more similar to torsades de pointes. The presence of a delta wave on prior ECG or on ECG after conversion to sinus rhythm helps confirm the diagnosis.

If a patient with pre-excited AF is hemodynamically stable, drug therapy is an acceptable treatment, but the practitioner must be prepared to deal with the potential of hemodynamic deterioration at any time. The drugs of choice for pharmacologic treatment are intravenous procainamide or ibutilide. Medications that preferentially block the AV node, especially if they also decrease blood pressure, may promote rapid conduction over the bypass tract resulting in VF and should be avoided in the patient with pre-excited AF. For this reason, administration of intravenous adenosine, intravenous amiodarone, or oral or intravenous digoxin, beta-blocker, or calcium channel blocker is not recommended in the patient with pre-excited AF [3, 48–51].

The hemodynamically unstable patient with pre-excited AF should undergo emergent synchronized DCCV. Due to the varying QRS morphology and prominent repolarization changes resulting in large T waves, appropriate synchronization of the external defibrillator on the QRS complex can sometimes be problematic in patients with pre-excited AF. The rhythm strip on the external defibrillator should be examined prior to DCCV to ensure that the synchronization markers are correctly overlying the QRS complexes and not the T waves. If the external defibrillator device is inappropriately synchronizing to the T waves, a different lead that gives improved discrimination of the QRS complexes and appropriate synchronization should be chosen. Inappropriate synchronization and shock on the T wave can result in VF [52]. If this occurs, the synchronization feature of the external defibrillator should be disabled and immediate defibrillation performed.

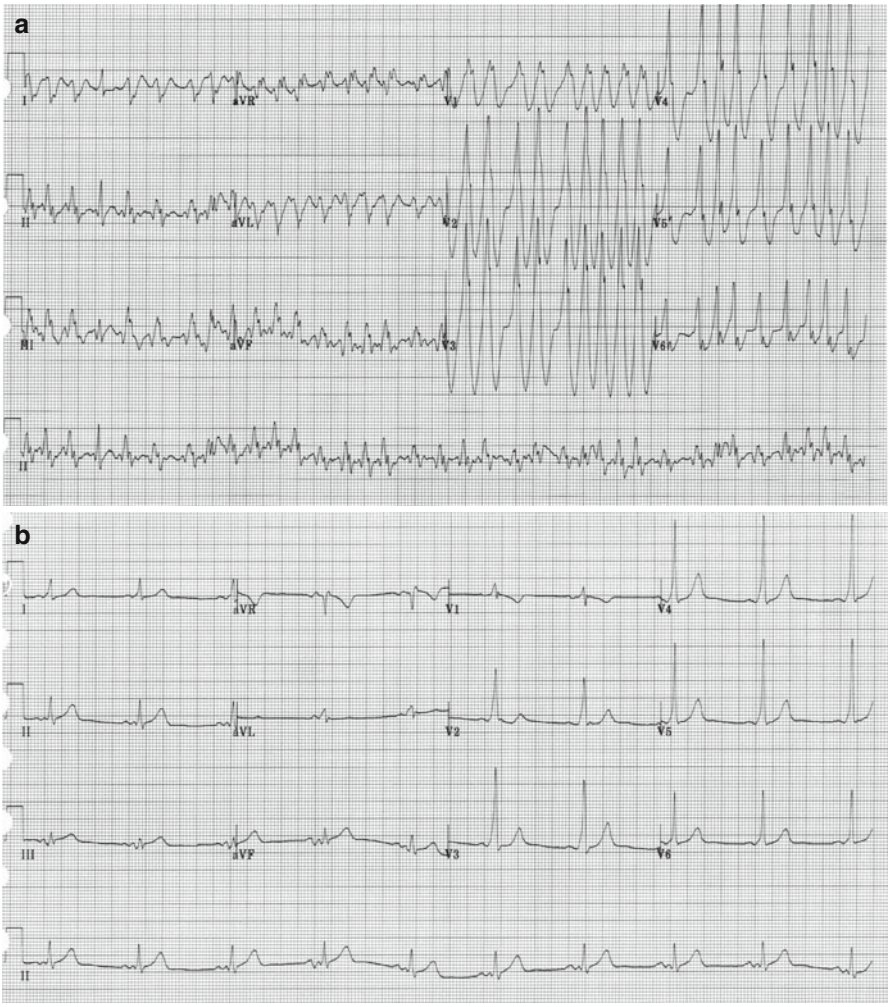

Fig. 14.11 (**a**) Pre-excited AF. Note the irregular, rapid, bizarre (not typical RBBB morphology) wide QRS complexes representing conduction to the ventricles via the accessory pathway, and the occasional narrow QRS complexes and fusion beats (e.g., 4th beat from the *left*), representing intermittent AV nodal conduction. (**b**) Sinus rhythm ECG from the same patient post-DCCV showing overt pre-excitation with an evident delta wave

Digoxin

Initiation of Digoxin in the Inappropriate Patient

Digoxin may be used either alone or, more commonly, in combination with other AV nodal blocking drugs for ventricular rate control in atrial fibrillation. Digoxin is usually less effective than other AV nodal blocking drugs at controlling ventricular rates during

exercise. Importantly, the prescribing practitioner needs to be aware that studies have associated an increased risk of mortality in patients taking digoxin [4–7]. If digoxin is prescribed, renal function should be checked prior to initiation. Impaired renal function can result in elevated serum drug levels and place patients at an increased risk of toxicity. In addition, serum electrolytes should be monitored because hypokalemia or hypomagnesemia can potentiate digoxin-associated arrhythmias. Loop diuretics that can cause hypokalemia should be prescribed with caution in the patient taking digoxin.

Inappropriate Interpretation of Digoxin Levels

Digoxin blood levels can serve to confirm whether or not a patient is taking the medication. The usefulness of the "therapeutic range" for digoxin levels is limited, must be interpreted in context of the timing of the last dose of the medication, and is ideally measured at least 6 h after the last dosing. Most importantly, digoxin toxicity is a clinical diagnosis, and digoxin-related cardiac arrhythmias and extracardiac manifestations can occur at therapeutic or even subtherapeutic levels.

Failure to Recognize Digoxin Toxicity

Findings of digoxin toxicity can be divided into cardiac and extracardiac manifestations. The most common arrhythmia manifestation is premature ventricular contractions [53]. Bradycardia and varying degrees of AV block, as well as atrial or ventricular tachycardias due to increased automaticity, are associated with digoxin toxicity. Bidirectional VT is also a classic association with digoxin toxicity. Importantly, ECG findings of digoxin "effect" (Fig. 14.12) include "scooped" ST segments with ST depression most commonly in the lateral leads, QT shortening, and increased u-wave

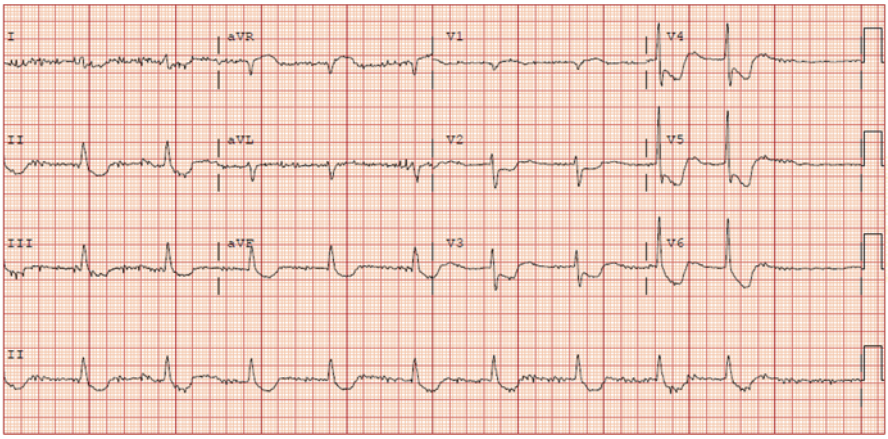

Fig. 14.12 Atrial fibrillation with "scooped" ST segments demonstrating "digoxin effect"

amplitude, and these findings correlate with chronic digoxin use, not digoxin toxicity [54]. Extracardiac manifestations most commonly involve the gastrointestinal or neurologic systems. GI manifestations may include nausea, vomiting, abdominal discomfort, or anorexia. Neurologic manifestations range from fatigue and lethargy to confusion and delirium. Varied visual changes are also frequently associated with digoxin toxicity with alterations in color vision being a common association.

Inappropriate Management of the Patient with Digoxin Toxicity

The hemodynamically and electrically stable patient with digoxin toxicity can be managed conservatively with supportive measures alone. Close monitoring of the patient's respiratory status, hemodynamic status, and neurologic status and continuous telemetry monitoring should be performed. In the patient with a hemodynamically unstable arrhythmia, end-organ dysfunction from hypoperfusion, or hyperkalemia [55], digoxin-specific antibody (Fab) fragments should be administered. Life-threatening ventricular arrhythmias should be treated with emergent cardioversion, if necessary, per ACLS guidelines.

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Part III
Transition to the Outpatient Environment

Chapter 15
Education of the Atrial Fibrillation Patient in the Observation Unit

Michelle Mead-Salley

We see these people in the emergency department every day: the young mother who was at the playground with her children when she becomes dizzy and her heart starts flipping around her chest, or the middle-aged man whose heart rate is 180 beats per minute even though he just started his workout, or the man who wakes up out of a sound sleep with his heart pounding. Sounds familiar? These are typical stories of atrial fibrillation patients that we see all the time. It used to be that uncomplicated atrial fibrillation was an automatic hospital admission, even if it was paroxysmal. With all of the changes in healthcare, the observation unit is becoming a more attractive location to treat these patients during an overnight stay. Along with medical observation comes the daunting task of taking the time to do patient education.

The challenges in the observation unit are different than that in the outpatient setting. In the Atrial Fibrillation Center, we have the luxury of appointments, developing relationships and following up with patients and their regimens that we assign to them. In the observation unit, there is a short amount of time to diagnose, treat, and educate patients. The practitioner must also develop therapeutic communication and a relationship that is at the least transient. In this chapter we will tackle patient education, how best to approach it, and how to be successful.

My first encounter with educating a new-onset atrial fibrillation in the observation unit was a young, healthy man who had a lot of appointments and obligations. He had no interest in his new diagnosis and told me he just wanted to be discharged so he could go to work and get on with his life. I called him the next day for a follow-up appointment which was supposed to be in 2 days. He could squeeze in his follow-up with his new cardiologist in a month when he could find an opening in his busy schedule. He eventually found his way to the Atrial Fibrillation Center.

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Many of the patients we encounter are not ready to hear about treatment options initially, but need time to digest their diagnosis. People feel betrayed by their body and cannot understand why this has happened to them, and it is not just an "old persons' diagnosis." This patient had already started making lifestyle changes before we saw him in the office. The above patient was shocked at his diagnosis and never wanted to feel like that again. He had read up on atrial fibrillation and had some questions that he wanted to ask. The encounter with this gentleman was very different than the initial contact. After his consultation with his new cardiologist, he felt much better than he did initially. He had done some "self-education" and had read the booklets that were given to him. Even though he was not ready to hear about anything during our first meeting, he was very receptive to education during his office visit. We came up with a treatment plan for the next time he had an episode. He was satisfied with his visit and was ready to participate in his care.

Francis Bacon said it best, "Knowledge is power." After a thorough literature search, there are limited publications on education of atrial fibrillation in the observation unit so we are breaking new ground. There are multiple resources for medical professionals developing long-term relationships with their patients but nothing for the staff in the observation unit that are trying to educate patients on their atrial fibrillation. So how do we approach these patients that need education to understand their disease process in a setting that can be rather stressful?

It is well documented that patients have a difficult time recalling the information that has been told to them. Patients all learn at a different rates and levels. Some people are visual learners and some are auditory. There are different educational hurdles to overcome, as well. Sometimes a whole family would like education and have a million questions. Education is important because having knowledge of their arrhythmia gives the patient a sense of control. Patients may have some preconceived notions of the diagnosis of atrial fibrillation. They arrive in the emergency department scared and feeling helpless. The diagnosis of atrial fibrillation only increases their anxiety as they start to recall family members that have pacemakers or have been on warfarin. They think of commercials that they see on a regular basis for blood thinners and all of the described side effects, not to mention the legal commercials for people on non-vitamin K oral anticoagulants that are meant to be inflammatory by the producers. Patients' anxiety may be relieved with good information.

Let's review some of the basics of communication. As we know, information is readily available at the click of a button. The information that we can acquire compared to 20 years ago is astounding. So as healthcare professionals, we have to step up our game. Our patients no longer just take the medications they are prescribed. They "Google" their side effects, some patients own *Physicians' Desk References*, and some knowledge, albeit not always the most accurate, is gathered from someone they know that has the same diagnosis. No longer are vague answers good enough for most patients. While sitting in the emergency center, patients have had time to look up their diagnosis and medications they are being given and start inquiring about what they have read.

It was thought in the past that a patient could not comprehend the complexities of medical diagnoses. Gone is the thought of "take two of these and call me in the morning." The "Patient's Bill of Rights" was created in 1975 by the American Hospital Association. This changed the way that the medical community approached patients about their diagnoses. It was thought that treatment should be centered around the patient rather than the institution of medicine. It is stated that the patient's individual needs should be considered when exploring options with his or her diagnosis [4].

So therapeutic communication ideology was developed. The premise behind therapeutic communication is that by using different communication techniques, you can help a patient understand their diagnosis and begin to accept it. It takes years to develop effective communication as a healthcare provider [7]. There are many things we do each day as we communicate with patients that we learned in our education. We use such as using humor, touch, and decision-making on how much information to give at a certain time.

When talking about education of patients, we often overlook the education of the observation unit staff. What is the observation staff's understanding of atrial fibrillation? When working in the emergency department, nursing staff is told during orientation that the role of the nurse in emergency is to know a little about a lot. Think about a typical day in an emergency department. The staff deals with such a wide variety of people and diseases that walk through the door. It would be impossible for the staff to understand all the intricacies of all disease processes. There was a study done that discussed how the correlation of nursing knowledge directly affected patient's knowledge. The conclusion to the study was that there are greater deficits in the knowledge of the patient if the nurse has knowledge deficits in the disease process. The study focused on how better communication between nursing staff and physicians leads to better patient understanding [3].

In another study, a group tested a patient's knowledge base of their atrial fibrillation over a 3-month period after their stay in the emergency department. The study concluded that the patient had moderate knowledge after discharge and in 3 months their knowledge did not improve very much. The group studied had a good grasp on the effects on their everyday life, but lacked knowledge in anticoagulation, symptom detection, and when to consult with a physician [5].

In the observation unit, you have to be able to get a lot of information across to the patient in a short amount of time. What is so appealing about the observational unit is also what makes patient education difficult. In an observation unit, a plan is in action, and the patients will be discharged in a timely manner if they improve. If they do not improve or worsen, they will then be fully admitted. The obvious shortcoming with this model is the lack of follow-up that the observation unit staff have with the patients. You need to be able to educate them about their disease state and medications within a short amount of time. The patient's ability and willingness to follow up is important in their education. Every avenue to make follow-up easier should be undertaken. It may be helpful to introduce the patient to the cardiology attending during the patient's observation admission. This can be of great help in the education process.

Let's first talk about new-onset atrial fibrillation. When a patient arrives to the observation unit, they have had their workup completed and are probably a little stressed and confused about this new finding. Maybe they have had a direct current synchronized cardioversion (DCCV). In the clinic we have the luxury of seeing a patient and sitting down with them and explaining the process of a cardioversion. The staff helps the patient understand that it is not without risk but we alleviate the patient's anxiety by having a 20-min discussion about the process. By the time the patient walks out of the office, they are confident in this plan and most actually look forward to it.

In the emergency department, there is no ability for a 20-min discussion in a relaxed atmosphere, but rather a decision is made to undergo a procedure that the patient consents to having, but has only seen used on people that are dying in medical television dramas. While the patient more than likely feels better after being cardioverted, they are still a little shocked how their day at the office has now turned into a more serious day in the emergency department.

So when talking with someone about their new-onset atrial fibrillation, it is important that they recognize the symptoms that brought them here in the first place. Did they have palpitations, dizziness, shortness of breath, or felt their heart racing? If they can identify these symptoms with their episode, they need to know what to look out for the next time they have another episode. Were they drinking alcohol at the time of onset? Alcohol is a known trigger for atrial fibrillation [6]. Giving patients the knowledge that there can be triggers will help them identify these in the future.

Was the patient started on anticoagulation? Another big subject that requires quite a bit of discussion. What kind of anticoagulation did the patient start? Is it good old fashioned warfarin? If so the patient needs to be aware of the effects of diet and alcohol on their warfarin levels. Has warfarin management been set up prior to discharge? This is a very important step. The newly diagnosed patient will not know the first thing about setting this up. Make sure before they are discharged that they know who is doing the monitoring as well as when they have their first lab draw scheduled. They need to know about the issue of increased bleeding times and what that could mean for them.

If the patient is going home on warfarin, do they also require enoxaparin injections as well? Teaching a patient to give himself/herself an injection can be time consuming. Sometimes you can enlist the help of pharmacy staff or a diabetic nurse specialist who can help in this education. Evaluating what is available within your own institution and what kind of resources you can use is important. Be creative! Don't be afraid to enlist the help of the patient's primary care physician who may be willing to see the patient in the office for some early teaching.

Now onto the non-vitamin K oral anticoagulants. Using these medications means no enoxaparin teaching, so no need to teach injections. The dietary restrictions are also limited again shortening educational time. There is, however, another conversation about reversal agents or rather lack thereof. This can be a scary but necessary subject to discuss with the patient. Patients on any anticoagulants should be encouraged to invest in a medical alert bracelet of some sort. Tell the patient to put a bright piece of paper in their wallet that has the anticoagulant name and dosage on it. The first place

the Emergency Medical Services (EMS) looks for information is in a wallet or purse. If the patient cannot speak for themselves at the time of emergency, it is important for emergency personnel to know that they are on a blood thinner. This little bit of knowledge could save a life. If the patient is on a twice-daily pill, make sure and emphasize the importance of taking it twice a day approximately twelve hours apart. If the patient is on a once-daily pill such as rivaroxaban (Xarelto), you need to stress the importance of taking it *with an evening meal* which is the recommendation on the package insert.

Patients also need education on bleeding complications. Dark, tarry stools are a big discussion. There is nothing worse than a gastrointestinal bleed that has been going on for days because the patient just thought that they had a little gastrointestinal distress and now the hemoglobin is down to five. A preventative conversation can alleviate a lot of risk in the future. Also mention the importance of seeking medical attention if the patient should hit their head while on any anticoagulants. A recent study in the Journal of Emergency Medicine did a study over a 2-year period of patients taking warfarin with minimal and minor head injuries. Of 176 patients enrolled, 157 had a head CT. Of those 157 patients, 28 (15.9%) had intracranial bleeding [1]. Patients need to understand that it is important to take anticoagulation, but also understand that it is not without risk. As always, make sure and document your education.

Antiarrhythmic medications are a tricky category. More than likely you will not be selecting an antiarrhythmic medication for your patient to start, but you may be starting a patient on a beta-blocker, calcium channel blocker, or Lanoxin. Informing a patient about the side effects of these medications is very important. One of the side effects is bradycardia. An important teaching point may be to show a patient how to take a radial pulse, or you could develop a handout on how to check your pulse. Home blood pressure kits are readily available and rather affordable. Encourage your patient to purchase one of these devices so they can also check for hypotension. Some insurance companies may even cover it.

One of the most important aspects of the patient's observation stay is the plan for discharge. Early follow-up is helpful at reducing early recurrent visits to the emergency department(ED) as discussed in the American Heart Journal, June 2013 article. It stated that of the 12,772 patients with atrial fibrillation seen in the ED, 67.8% had no follow-up within 14 days of their initial presentation, and there were 1310 repeat ED visits made by 1146 patients. The conclusion of this study was that early follow-up was key to decreasing repeat visits to the ED [2].

How do you assist with early follow-up? Try to make a patient's follow-up appointment prior to them leaving your area. Either make contact with their primary care physician or introduce them to a cardiologist/electrophysiologist. Give them their appointment time with their discharge instructions. When you consult with their follow-up physician, make sure they have access to the emergency department records. If they do not, give them copies of the electrocardiogram showing the atrial fibrillation and any testing that was done during his/her observation stay. Make sure and tell them to bring those with them to their physician visit. Good communication will increase patient and physician satisfaction.

As we know from countless articles, atrial fibrillation is here to stay. The projections of people affected by atrial fibrillation in the future are inconsistent as is the

estimate of people currently diagnosed with this arrhythmia. What, however, is consistent is that millions of people are affected, and projections have shown this number is only increasing.

The treatment options are good but not great. Antiarrhythmic medications are good but carry with them side effects and may not be effective in all patient populations. Anticoagulation has definitely improved over the years with the arrival of the novel agents and newfound freedom from strict dietary regimens and blood draws. Even though the medications are improved, however, it is not a carefree lifestyle. Ablation procedures offer type of atrial fibrillation hope, but they vary in success depending on the patient's anatomy of their left atrium, and the skill of the operator. One thing that can keep improving with all of medicine is our relationships that we develop with our patients. These relationships can become a partnership over time. While atrial fibrillation can be frustrating, that does not mean that it is hopeless.

In the observation unit, sitting down with a patient and just talking to them about their diagnosis may mean a lot to them and may open up their minds to learn, even just a little, about atrial fibrillation. The encounter with the young man previously mentioned just reinforced that education in the emergency department may not be all that well received, but it is necessary. He had heard enough of the conversation to realize that he needed to follow up.

The future of medicine seems to point more and more to outpatient treatment, and what happens in the observation unit may set the tone for a patient's treatment plan. Encourage as much education as the patient is ready to hear, and get the patient early follow-up to keep repeat visits to the emergency department at a minimum and increase patient and physician satisfaction. A multidisciplinary collaborative effort may be the best approach going forward for these patients to make sure that they understand their diagnosis and the treatment options and to hopefully prevent multiple trips to the emergency department.

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Chapter 16
Discharge Criteria

Deborah B. Diercks and George McLeod

Perhaps the most controversial decision in the management of atrial fibrillation resides around the patient's disposition. The incidence of atrial fibrillation is on the rise as life expectancy increases, the incidence of coronary artery disease increases, and the tools used to diagnose atrial fibrillation improve. The USA spends approximately 26 billion dollars annually on the management of atrial fibrillation, the majority of which is spent on hospitalizations for management. Approximately 80 % of evaluations for atrial fibrillation are hospitalized in the USA, whereas 60 and 25 % of cases are hospitalized in Australia and Canada, respectively, without any significant difference in outcomes. Despite such a high hospitalization rate for atrial fibrillation, there is, in most patients, a very low 30-day adverse event rate [1]. This staggering difference highlights the need for standardized criteria to aid emergency department physicians in deciding whether patients presenting with atrial fibrillation should be discharged home and admitted to the hospital or to an observation unit. Furthermore, criteria that can be used to discharge subjects home from an observation unit or emergency department would substantially decrease healthcare costs.

There is little data regarding discharge criteria from an observation unit; therefore, extrapolation from like populations is required. The AFFORD trial was a prospective cohort trial which studied 30-day adverse event rates for patients presenting to the emergency department with symptomatic atrial fibrillation. The goal for the trial was to identify patients who were appropriate to discharge from the emergency department. Data were collected from patients presenting to the emergency

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department with a primary complaint of symptomatic atrial fibrillation in the first 2 h from initial evaluation. Regardless of disposition, patients were followed for 30 days to record adverse events, which were graded from 1 (none) to 10 (atrial fibrillation-related death). A decision rule was created using covariates such as age; initial vitals including systolic blood pressure, pulse rate, respiratory rate, oxygen saturation, chest X-ray results, hemoglobin, and creatinine; and comorbidities including congestive heart failure, coronary artery disease, and history of cardioversion. Overall, those patients with the least burden of comorbidities had the lowest rate of adverse events and therefore identified a patient population that was suitable for discharge (Table 16.1) [1].

Although not explicitly defined, most trials discharged patients with absence of compelling indication requiring admission such as suspicion of or confirmed sepsis, acute coronary syndrome, cerebrovascular accident, and acute congestive heart failure [1]. Patients who were successfully rate controlled in the first 2 h from initial emergency department physician evaluation were considered low risk for adverse events and were determined to be safe for discharge if tolerating oral medication. In most studies that looked at discharge from the emergency department or observation

Table 16.1 Components of the Clinical Decision rule described in the AFFORD trial [1]

Hemodynamic instability	5
Hypoxia	5
NYHA III/IV CHF	5
ACS	5
Sepsis	5
Thyroid storm	5
CVA	5
Respiratory distress	5
Acute kidney injury	5
Complications from cardioversion	5
Structural heart disease	2
Poorly controlled DM	2
Poorly controlled HTN	1
Chronic kidney disease	1
Age >70	2
Peripheral artery disease	1
History of prior DCCV	1
Inability to derive history	1
Further testing warranted	1
Inability to rate/rhythm control	1
Thyroid dysregulation	1
Score $\geq 5 \rightarrow$ admit	
Score 2–4 $\rightarrow$ observation unit (if from two separate characteristics)	

unit, patients were managed on an outpatient basis with primary care or cardiology follow-up clinic visits.

The subset of patients that is at the greatest risk of adverse events and should therefore be given careful consideration prior to discharge after treatment in the emergency department or observation unit for atrial fibrillation were patients with significant comorbidities [1]. It is therefore essential that prior to discharge from an acute treatment area, all comorbid conditions have been addressed. Patients who are rate controlled, can tolerate oral medications, and have follow-up may still require further hospital management for their other medical problems. These patients with conditions that require continued medical interventions may warrant transition to an inpatient unit to address their additional acute conditions.

A recent investigation performed at a university hospital was designed to evaluate whether an observation unit substantially reduced admission rates for management of symptomatic atrial fibrillation. In the retrospective evaluation, cases of atrial fibrillation were reviewed over three time periods: one without an observation unit or outpatient clinic follow-up, one with an observation unit, and one with both an observation unit and outpatient clinic for close follow-up of cases determined to be appropriate for discharge from the emergency department. In this study, the authors noted that conditions that prevented early discharge from the diagnostic unit included lack of rhythm control, advanced age, and frailty and numerous significant comorbidities.

In patients without structural heart disease where rhythm control is determined to be the primary strategy, the majority can be converted into sinus rhythm prior to discharge. In the absence of significant comorbidity requiring admission, those patients who were able to be converted to sinus rhythm had a very low rate of subsequent adverse events and are safe for discharge after 3–6 h of observation [1–3]. However, in the same patient subset, if the patient has a history of known coronary artery disease and presents with symptomatic atrial fibrillation, it is reasonable to obtain serial cardiac markers to exclude a myocardial infarction prior to discharge. Those patients who are unable to provide a reliable medical history or require further analysis with echocardiogram may also warrant the completion of testing prior to discharge.

Patients without NYHA III/IV heart failure or other acute illness presenting with symptomatic atrial fibrillation for less than 48 h duration also may undergo pharmacological intervention to convert to sinus rhythm. Prior to discharge on antiarrhythmic medications, and an anticoagulant as indicated, these patients should be observed for maintenance of sinus rhythm for a period of time. Those patients who have age > 70, poorly controlled diabetes mellitus, lack of rhythm control, and significant comorbidities including structural heart disease, thyroid dysfunction, respiratory disease, and life expectancy less than 6 months are known to be high risk for adverse events, and, therefore, careful consideration should be taken with regard to discharge planning.

While no definitive criteria exist to identify whether patients presenting to emergency departments should be discharged, observed, or admitted, the preponderance of evidence suggests a lower risk category can be safely discharged with outpatient

Table 16.2 Criteria for discharge

Criteria for discharge
1. Rate or rhythm control
2. Completion of diagnostic evaluation
(a) Echocardiogram if warranted
(b) Evaluation for ischemia/infarction if warranted
3. Anticoagulation started as indicated
4. Outpatient follow-up arranged
5. Outpatient therapy defined

follow-up. Although there are no specific trials that define discharge criteria, it is reasonable based on available data to discharge patients after rate or rhythm control has been obtained, diagnostic evaluation is complete, anticoagulation is started as indicated, and follow-up arranged (Table 16.2).

In the absence of other acute medical conditions, this approach allows patients, such as a middle-aged male with well-controlled hypertension and diabetes presenting with stable vitals other than tachycardia who is successfully rate controlled with IV metoprolol and then observed after taking PO metoprolol, to be discharged home with outpatient clinical follow-up regardless of the modality of conversion. Pairing this approach with those patients identified as low risk for adverse events should result in efficient and safe medical care.

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Chapter 17
Discharge Planning

Ginger Conway and Barbara Bell

Discharge planning is the process of evaluating and planning for the patient's needs post-discharge. The process begins at the time of presentation and must be reevaluated throughout the treatment period [1, 2]. A well-executed discharge plan minimizes the impact of delays and complications that may impede the patient's ability to progress to the next locus of care [3]. This is especially beneficial for the elderly who, due to multiple comorbidities coupled with cognitive and mobility decline, tend to have complex discharge plans [4, 5].

The emergency department is often the location of the initial diagnosis of atrial fibrillation [6]. Management of atrial fibrillation in the emergency department may include stabilization of rate and/or rhythm as well as discharge to home or another facility or admission to an observation or inpatient unit [6–9]. If a patient is to be discharged from the emergency department for outpatient follow-up, the plan of care must include a comprehensive individualized discharge plan.

Importance of Discharge Planning

The Agency for Healthcare Research and Quality (AHRQ) reports that undesirable events occur within 30 days of discharge in nearly 20 % of all patients. They further emphasize that 75 % of poor outcomes are potentially preventable or can be minimized by a quality discharge plan [3, 10]. In addition, individualized comprehensive

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discharge plans may reduce hospital readmissions resulting in cost savings for healthcare systems [3, 10].

Atrial fibrillation, present in 3.3–10% of admissions, is the most common cardiac dysrhythmia seen in the emergency department [6, 11]. Prevalence continues to increase as the population ages [6–8, 12–16]. It is well documented that individuals with atrial fibrillation, especially those 60 years of age and older, are at an increased risk for ischemic stroke [9, 15]. In addition, a diagnosis of atrial fibrillation is frequently associated with multiple comorbid conditions, including structural heart disease, advanced age, heart failure, chronic obstructive pulmonary disease, hypertension, thyroid disorders, renal dysfunction, sleep apnea, alcohol abuse, dyslipidemia, obesity, and cardiomyopathies [6, 8, 9, 14]. Dizziness and light-headedness, common symptoms associated with atrial fibrillation, are often precursors to falls in a population where the consequences are of increased significance due to anticoagulation and/or age.

Causes for poor outcomes are multifactorial and include multiple prescribers, polypharmacy, poor adherence, inadequate education, and/or poor discharge preparation [2, 17, 18]. Failure to adhere to the medical plan increases the risk of poor outcomes including bleeding, stroke, poorly controlled ventricular response rate, toxic drug levels, and worsening symptomatology [15]. It has been demonstrated that effective discharge planning can improve adherence to the medical plan [9, 15].

Effective Discharge Planning

Patients are typically discharged from the emergency department with an ongoing need for medical care [2, 19]. Patients and their caregivers are often unprepared to care for themselves in the next care setting [20]. Literature supports that those with chronic atrial fibrillation do not have an adequate understanding of their disease, symptoms, medication management, stroke risks, and when to seek emergency care [15, 21].

Despite the chaos in the emergency department, the effective discharge plan must begin with the first encounter regardless of the final disposition of the patient [19]. All members of the healthcare team including physicians, nurses, pharmacists, social workers, as well as patients, their family, and any other post-discharge caregivers must be involved in assessing the needs of the patient and their ability to meet them [3, 19]. Frequent collaboration provides opportunities for the development of the discharge and post-discharge plan [2, 4, 5, 22]. It improves the patient's ability to achieve more sustainable skills, resulting in improved outcomes and increased patient satisfaction [3, 4, 20].

The discharge plan and the manner in which it is communicated must be individualized for each patient [21, 22]. Individualized discharge plans improve adherence and outcomes by empowering patients to manage their health problems [3, 15, 19]. It is essential that the patient and caregivers understand and have the ability to implement the discharge plan [3, 19, 21]. It has been documented that patients and

caregivers who have confidence in their ability to care for themselves, and believe that their actions will result in the desired outcomes, are more likely to follow the medical plan [15, 19, 22].

Patients and their caregivers must be assessed for their knowledge and ability to learn. The ability to learn may be limited early in their care due to anxiety, symptom severity, and fear [3, 19]. This is especially true in the stress-filled environment of an emergency department [18]. Health literacy, the ability to read and understand prescription labels, instructions, appointment cards, and health-related materials, should also be assessed [21, 23, 24]. It has been reported that as many as 80 million Americans have poor health literacy [24].

Discharge instructions must be legible, provided in a patient-friendly format, printed in a font appropriate for the patient's vision, and be at a reading level appropriate for patients and their families/caregivers [19, 21, 25]. Most are written at a 9th–10th grade level, when they ideally should be at the 6th grade reading level [19, 23]. Verbal review of the discharge instructions may result in improved comprehension by the patient [21].

Contents of the Effective Discharge Plan

The AHRQ has published a document providing guidance on effective discharge planning. The IDEAL discharge plan includes elements addressing the needs of the patient, family, and healthcare providers in the discharge process [3]. This document has been developed for an inpatient setting but provides guidelines that can be applied in the emergency department.

The key elements in the IDEAL discharge plan include the following:

I = inclusion of patient and family in the process
D = discussion of key areas to prevent problems in the home setting
E = education in patient-appropriate language concerning the health condition and steps that will be taken to ensure a quality transition to the next locus of care
A = assessment of patients' understanding of information provided by healthcare workers using the teach-back method
L = listening and respecting the patient and family regarding their goals and concerns and an assessment of their needs and concerns [3]

The discharge plan should include information for the patient and family on what to expect when discharged back to the community. It should be tailored for that particular patient [3]. Additional items to be included are information regarding when to return to the emergency department, symptom monitoring, medication instructions, dietary restrictions if any, further diagnostic evaluations needed, follow-up appointments, and contact information if there are questions [2, 3, 15, 19]. It is essential that key information be conveyed in the discharge process while remembering that too much information can actually decrease retention. Less text, more pictures, and thoughtful word choices are key [21, 25].

Medications

Post-discharge management of medications is an important part of discharge planning. Medication reconciliation is crucial and should include over-the-counter medications [3]. Patients seldom reveal what over-the-counter medications and herbal therapies they are taking, not realizing that there are possible drug-to-drug interactions. The discharge plan should include dosages, actions, side effects, and any dietary restrictions or drug-to-drug interactions [3, 15]. Patients are encouraged to discuss and maintain a written record of all active medications and supplements.

The primary approaches to the management of atrial fibrillation are rate versus rhythm control [6, 8, 9]. It is beneficial if patients know which medications are prescribed to control rate or rhythm. If the patient underwent cardioversion in the emergency department, discharge instructions should include information about the procedure and the need for follow-up. If the patient is prescribed anticoagulation therapy, they will require strict follow-up for INR testing and dose adjustments [3, 8, 9, 14, 26]. Newer non-vitamin K antagonist oral anticoagulants may also be prescribed for patient with non-valvular atrial fibrillation [8, 9, 14]. It is important to discuss bleeding risks with any patients prescribed any anticoagulant therapy [3, 8, 9, 14].

Symptom Assessment

The heterogeneity of the patient's symptoms, clinical presentation, and comorbid conditions makes discharge education as it relates to symptom assessment challenging [18]. Those with atrial fibrillation are at a greater risk for stroke; however, many do not even know the signs and symptoms of a stroke [15]. It is estimated that 20 % of strokes are due to atrial fibrillation [8, 9, 14]. Patients with atrial fibrillation may be asymptomatic, or they may describe a cacophony of symptoms including palpitations, dizziness or light-headedness, fatigue, weakness, angina, shortness of breath, or syncope [9, 18]. A comprehensive discharge plan for symptom assessment will include signs and symptoms of stroke and the need to seek medical care immediately [15, 21]. It should also include a discussion of what atrial fibrillation feels like for that individual and when to contact a healthcare provider for a change in symptoms.

Follow-Up and Additional Testing

Patients and their caregivers/family are often the only common thread as they navigate the healthcare system [20]. They need an explanation of their diagnostic evaluation and results [3]. This information along with instructions on follow-up

appointments, pending diagnostic results, and future evaluations should be provided in written format including names, addresses, directions, and contact information [3, 19, 27]. If at all possible, the appointments should be made prior to discharge. If this is not possible, the patient and their family/caregivers will need adequate information so that they can follow through with the follow-up plan [3, 21].

The decision between referral to primary care or cardiology is based on patient or provider preference, level of difficulty managing rate or symptoms, and concomitant structural heart disease [8, 9, 14]. It is essential that patients are provided with adequate information that ensures that the team in the next setting will have a complete understanding of the patient's emergency care and the discharge plan [3]. The written discharge summary should include the patient's functional status, medical history, baseline information, learning needs, plan of care, and services provided while in the emergency department [3]. The transfer of information between the hospital team to the outpatient team is essential for continuity and transition of care. Documentation of the discharge plan in the patient's medical record will facilitate this communication [4]. Poor outcomes can be a result of the lack of appropriate communication between the pre- and post-discharge healthcare team [4, 28]. Follow-up phone calls can be used to assess understanding and patient's progress. This may be especially helpful for vulnerable populations [2, 4, 19].

How to Get It All Done

The emergency department is a unique high-acuity fast-paced, and at times chaotic, environment which creates significant challenges for quality communication [19]. There are time constraints, unpredictable interruptions, overcrowding, frequent staff changes, and life-threatening medical emergencies [16], all of which can result in discharge planning being a low priority [2]. Being in the emergency department is also a time of increased stress for patients and their families which may diminish their ability to assimilate new information [19]. It is essential for the emergency department staff to address the patient's concerns and maintain open, high-quality communication [3, 21]. The excellent observation skills of nurses in the emergency department coupled with their frequent patient interactions enhance the discharge planning process; however, many teachable moments are overlooked or missed. Discharge planning is often left to the nursing staff only, but all members of the healthcare team should look for and capture these teachable moments. Repetition and reinforcement of the information improve patient understanding [3].

Incorporation of the discharge planning process into the acute assessment tools, standardized order sets, checklists, and ongoing documentation will help all members of the busy healthcare team remain engaged in the discharge process, ultimately avoiding last-minute hurried discharge plans [2]. Utilization of prepared discharge materials regarding medications, lifestyle modifications and symptom assessment can facilitate more efficient comprehensive discharge instructions [2, 29]. Posters, videos, preprinted materials, and the use of a systematic approach,

such as the IDEAL discharge planning process, can guide staff in a busy emergency department through all the elements of discharge planning [2, 3]. Using the teach-back method will assist in assessing the patient's and their families' understanding [19, 21]. Checklists can be used to simplify responsibilities and help the patient and family feel prepared to assume responsibility of their care after discharge [3, 25].

Conclusions

Discharge planning is a complex process that begins with the first encounter in the emergency department and continues throughout the entire stay. Effective discharge planning involves the patient and their caregivers as well as all members of the healthcare team. It is patient focused, practical, individualized, and aimed at improving adherence to the medical plan. The ultimate goal is to improve outcomes. It is provided in written and verbal format that the patients and their caregivers can understand and shared with all members of the healthcare team ideally through a shared medical record. The majority of published data on discharge planning is from research focused in the inpatient setting. There is limited evidence-based data on appropriate discharge processes from the emergency department as well as limited published information regarding discharge plans specific for individuals with atrial fibrillation. In an era that increasingly challenges institutions and healthcare providers to own responsibility for patient and financial outcomes, each institution will need to assess the needs of the population they serve and the services provided by their institution and community. More research is needed to ensure that discharge plans result in improved outcomes.

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Chapter 18
Outpatient Medications in Atrial Fibrillation

L. Kristin Newby and Sean D. Pokorney

Managing outpatients with atrial fibrillation involves managing three areas: anticoagulation, rhythm control, and rate control. Randomized clinical trials have not demonstrated a mortality or stroke benefit with a rhythm control strategy versus a rate control strategy [1–4]. While a substudy from the Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) study found comparable quality of life scores among patients randomized to rate or rhythm control strategies, maintenance of sinus rhythm in clinical trials was associated with improved quality of life and increased exercise capacity in some patients [5–7]. Evaluating patients outside of clinical trials, a quality of life study from the Registry on Cardiac Rhythm Disorders Assessing the Control of Atrial Fibrillation (RECORD-AF) found that rhythm control was associated with higher quality of life scores [8]. These data have led the atrial fibrillation (AF) guidelines to recommend antiarrhythmic medications to relieve symptoms in patients with symptomatic AF [9, 10]. The 2014 American Heart Association/American College of Cardiology (AHA/ACC) guidelines recommend antiarrhythmic medications as first-line therapy for patients with symptomatic AF, but rate control alone remains a cornerstone of management in asymptomatic patients [10].

Rate Control

Rapid ventricular response during AF can contribute to symptoms. One aspect of this is that faster heart rates result in a smaller proportion of time in diastole, which can lead to heart failure in patients with diastolic dysfunction. Without any

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atrioventricular nodal blocking agents, patients in AF have mean resting heart rates in the 110–120 beats per minute [11, 12]. Long periods of tachycardia due to AF with rapid ventricular response can result in a tachycardia-induced cardiomyopathy with a decline in ejection fraction [13]. The target heart rate to be considered sufficient rate control is an ongoing debate. Rate control in the AFFIRM trial was defined as (1) a ventricular heart rate of 80 beats per minute or less at rest, (2) a heart rate of less than 110 beats per minute during a 6-minute walk on flat ground, and (3) a mean heart rate of less than 100 beats per minute over a 24-h period with no heart rates greater than 110 % of the age-adjusted maximum predicted heart rate [1].

The Rate Control Efficacy in Permanent Atrial Fibrillation: a Comparison between Lenient versus Strict Rate Control II (RACE II) trial then randomized AF patients to strict rate control strategy (a similar definition to that from AFFIRM) versus a lenient rate control strategy (resting heart rate less than 110 beats per minute). The rates of cardiovascular death, heart failure hospitalizations, stroke or systemic embolism, bleeding, or life-threatening arrhythmias were similar in the lenient and strict rate control arms [14].

There are multiple medications that can be used to slow conduction in the AV node and facilitate rate control in AF patients (Table 18.1).

Beta Blockers

Beta blockers work by blocking the effects of epinephrine, and beta blockers can be beta-receptor selective or nonselective. Data from the AFFIRM trial showed that beta blockers were the most commonly used rate control medication with 50 % of AF patients receiving a beta blocker alone or in combination with digoxin or a calcium channel blocker [28]. Similarly, beta blockers were the most commonly used rate control agents in contemporary clinical trials and registries, in which 53–65 % of AF patients were taking a beta blocker (Table 18.2) [29–38]. Compared with calcium channel blockers and digoxin, beta blockers were the most effective drugs at controlling ventricular rates both at rest and with exercise [28, 39, 40]. The characteristics of many of the beta blockers are described in Table 18.1.

Patients with heart failure and AF are an important subpopulation of patients. Since carvedilol and metoprolol XL are both recommended for patients with heart failure with reduced ejection fraction, these are the two agents that should be considered for rate control in AF patients with a concomitant cardiomyopathy. A small randomized, double-blind, placebo-controlled study of 47 patients with AF and heart failure found similar efficacy in heart rate lowering between carvedilol and digoxin, and improved heart rate control and symptom scores were observed with the combination of carvedilol and digoxin, compared with a single agent [41]. A subanalysis of the US Carvedilol Heart Failure Trial evaluated carvedilol versus placebo in patients with heart failure and AF at the time of trial enrollment. Among the 136 patients evaluated, patients treated with carvedilol versus placebo had improvement in their ejection fraction and physician global assessment (71 % vs.

Table 18.1 Rate control agents

Generic drug name	Oral dose range	Renal dose adjustments	Primary metabolism	Excretion	Half-life	Mechanism
Beta blockers						
Atenolol [15]	25–200 mg daily	CrCl 15–35: max dose of 50 mg daily. CrCl < 15: max dose of 25 mg daily	N/A	Urine: 50 %	6–7 h	Beta-1 receptor selective inhibitor
Bisoprolol [16]	2.5–20 mg daily	CrCl < 40: start at 2.5 mg	Liver	Urine: 50 % (unchanged)	9–12 h	Beta-1 receptor selective inhibitor
Carvedilol [17]	3.125–25 mg twice daily	No dose adjustment but plasma concentrations increased in renal impairment	Liver	Urine: <2 % (unchanged)	7–10 h	Nonselective beta-receptor inhibitor with alpha-1 receptor inhibition activity
Carvedilol phosphate [18]	10–80 mg daily	No dose adjustment but plasma concentrations increased in renal impairment	Liver	Urine: <2 % (unchanged)	7–10 h	Nonselective beta-receptor inhibitor with alpha-1 receptor inhibition activity
Metoprolol tartrate [19]	25–200 mg twice daily	No dose adjustment	Liver	Urine: 95 % (<5 % unchanged)	3–7 h	Beta-1 receptor selective inhibitor
Metoprolol succinate [20]	12.5–400 mg daily	No dose adjustment	Liver	Urine: 95 % (<5 % unchanged)	3–7 h	Beta-1 receptor selective inhibitor
Nadolol [21]	20–320 mg daily	CrCl 31–50: dosage interval 24–36 h. CrCl 10–30: dosage interval 24–48 h. CrCl <10: dosage interval 40–60 h	N/A	Principally unchanged in urine	20–24 h	Nonselective beta-receptor inhibitor

(continued)

Table 18.1 (continued)

Generic drug name	Oral dose range	Renal dose adjustments	Primary metabolism	Excretion	Half-life	Mechanism
Propranolol [22]	10–30 mg 3–4 times daily	No dose adjustment	Liver	Principally metabolized and excreted in urine	3–6 h	Nonselective beta-receptor inhibitor
Non-dihydropyridine calcium channel blockers						
Verapamil [23]	80 mg 3 times daily–120 mg 4 times daily ER dosing 120 mg daily to 240 mg twice daily	No dose adjustment. Closely monitor patients with impaired renal function	Liver	Urine: 70 % (3–4 % unchanged)	5–12 h	Calcium ion influx inhibitor
Diltiazem [24, 25]	30–90 mg 4 times daily ER dosing 120–360 mg daily	No dose adjustment	Liver	Urine: 2–4 % (unchanged)	3–5 h	Calcium ion influx inhibitor
Others						
Digoxin [26]	0.125–0.5 mg daily, titrate to lowest dose with ventricular rate control	Use with caution, as primarily excreted in urine. Starting dose 0.0625 mg	Liver	Urine: 50–70 % (unchanged)	2 days (4–5 days in anuric patient)	Cardiac glycoside parasympathetic properties affecting the AV node
Amiodarone [27]	100–200 mg daily	No dose adjustment	Liver	Urine: minimal	Mean of 58 days (range 15–142 days)	Noncompetitive alpha- and beta-receptor inhibitor

N/A not available, *ER* extended release, *CrCl* creatinine clearance by Cockroft-Gault calculation, *AV* atrioventricular

Table 18.2 Rates of use of medications in atrial fibrillation clinical trials and registries

Generic drug name	ACTIVE-I [29]	RE-LY [30]	ROCKET AF [31]	ARISTOTLE [32]	ENGAGE [33]	ORBIT AF [34, 35]	TREAT AF [36]	ATRIA-CVRN [37]	Medicare Part D 2007 [38]
Beta blocker	4913 (54%)	11,375 (63%)	9212 (65%)	11,482 (63%)	N/A	64%	71,311 (58%)	15,926 (58%)	20,420 (53%)
Calcium channel blocker	2445 (27%)	N/A	3954 (28%)	5567 (31%)	N/A	30%	N/A	7920 (29%)	N/A
Non-dihydropyridine calcium channel blocker	N/A	N/A	N/A	N/A	N/A	17%	37,082 (30%)	N/A	6756 (17%)
Digoxin	3148 (35%)	N/A	5239 (37%)	5828 (32%)	6327 (30%)	24%	28,679 (23%)	4858 (18%)	11,453 (30%)
Antiarrhythmic	2066 (23%)	N/A	1434 (10%)	N/A	N/A	29%	18,445 (15%)	N/A	6823 (18%)
Amiodarone	N/A	N/A	N/A	2051 (11%)	2492 (12%)	10%	11,655 (10%)	N/A	3547 (9%)

N/A = not available

48 %, $p = 0.025$), as well as a trend toward lower rates of heart failure hospitalization or death (RR 0.35, 95 % CI 0.12–1.02, $p = 0.055$) [42]. However, in a meta-analysis of heart failure clinical trials, the association between beta blocker use and mortality in a population of 3066 AF patients with heart failure, treatment with beta blockers was not associated with lower mortality compared with placebo (HR 0.97, 0.83–1.14; $p = 0.73$) [43].

Calcium Channel Blockers

Dihydropyridine calcium channel blockers (including amlodipine, felodipine, nicardipine, and nifedipine) are selective for vascular calcium channels and are used for blood pressure control due to their vasodilatory properties. Non-dihydropyridine calcium channel blockers (including diltiazem and verapamil) are myocardial selective and have atrioventricular nodal blocking properties. Unlike beta blockers, non-dihydropyridine calcium channel blockers should not be used in patients with heart failure with reduced ejection fraction due to their negative inotropic effects; however, these medications may be used in patients with heart failure with preserved ejection fraction. Non-dihydropyridine calcium channel blockers should not be used in patients with AF and preexcitation, as verapamil has been shown to decrease the refractoriness of the accessory pathway, which can then result in ventricular fibrillation in the setting of AF [44].

The degree of heart rate control is dose dependent, and at higher doses of 360 mg per day of diltiazem, there was also better heart rate control at rest among AF patients [45]. The use of diltiazem has been shown to result in similar heart rate control to digoxin at rest, but improved rate control with diltiazem, relative to digoxin, was noted with exercise [46]. Among 9 patients with permanent atrial fibrillation who underwent maximal treadmill testing on and off diltiazem, maximum heart rate was reduced by 17 % with diltiazem ($p < 0.01$) [47]. A study of 18 patients with permanent AF found that relative to digoxin (167 ± 12 beats per minute), diltiazem (142 ± 24 beats per minute, $p < 0.001$) and verapamil (137 ± 30 beats per minute, $p < 0.001$) both significantly lowered the heart rate during a 6-min walk test, and the peak heart rates were similar between diltiazem and verapamil [48]. The heart rate reduction with non-dihydropyridine calcium channel blockers was associated with greater exercise capacity among AF patients [49].

Digoxin

Digoxin affects heart rate in AF patients through its parasympathetic properties on the atrioventricular node. Because of the vagolytic properties of digoxin, the medication is not effective at controlling heart rate with exercise [46]. Digoxin is one of the oldest cardiac medications that is still in use today, but there is a meaningful

amount of controversy around the use of digoxin in AF patients, and digoxin is no longer a first-line agent for AF [10]. Digoxin remains frequently used in AF patients in clinical practice (Table 18.2). Data from the RACE II trial showed that patients treated with digoxin alone or in combination with other agents were less likely to achieve their rate control targets relative to patients not treated with digoxin (79 % vs. 87 %, $p = 0.007$) and patients on digoxin were more likely to require a combination of rate control medications and had higher rates of hospitalizations for rate control [50].

Digoxin has been studied in a large randomized clinical trial of heart failure patients, in which reduced heart failure hospitalizations but no change in mortality was demonstrated for patients treated with digoxin [51]. The trial had AF as an exclusion, and occurrence of AF was the second most common reason for digoxin discontinuation in the trial (approximately 3 % of patients over the course of the trial). There have been no randomized clinical trials of digoxin in patients with AF stratified by heart failure or not. Retrospective, observational data are conflicting regarding the safety of digoxin in AF patients, although data has been mounting about the association between digoxin and higher mortality among AF patients. The confusion around digoxin began with secondary analyses of digoxin from AFFIRM, which were conflicting within the same dataset, depending on the statistical methodology used. The Cox proportional hazards model found that digoxin was associated with higher all-cause mortality (HR 1.41, 95 % CI 1.19–1.67, $p = 0.001$) and higher arrhythmic mortality (HR 1.61, 95 % CI 1.12–2.30, $p = 0.009$), and these associations were consistent across patients with and without heart failure at baseline [52]. However, methodology using propensity score matching within the AFFIRM dataset found that there was no association between digoxin and all-cause mortality or arrhythmic death [53].

There was no association between digoxin use and higher cardiovascular morbidity or all-cause mortality in RACE II, although this analysis was limited to 284 patients and was underpowered to detect these outcomes, and 11 % of patients had an ejection fraction of 40 % or less, while 35 % of patients had NYHA class II or greater [50]. Multiple larger analyses have supported the concerns regarding the association between digoxin use and higher mortality among AF patients. In ROCKET, digoxin was more frequently used among patients with heart failure (73 % vs. 56 %, $p < 0.001$) and was associated with higher all-cause mortality (HR 1.17, 95 % CI 1.04–1.32, $p = 0.009$) and higher rates of sudden cardiac death (HR 1.36, 95 % CI 1.08–1.70, $p = 0.008$) [31]. In ORBIT AF, 24 % of patients were on digoxin at baseline and 31 % of patients were exposed to digoxin over the duration of follow-up. Incident digoxin use among patients without heart failure was associated with higher all-cause mortality (HR 1.99, 95 % CI 1.12–3.56) and higher rates of cardiovascular hospitalization (HR 1.58, 95 % CI 1.12–2.23), but the use of digoxin was neutral with respect to all outcomes for prevalent digoxin use, as well as incident digoxin use among patients with heart failure [34]. Data from the SPORTIF III and V trials also found an association between digoxin and higher all-cause mortality (HR 1.53, 95 % CI 1.22–1.92, $p < 0.001$) [54]. The TREAT AF study is a US Veterans Affairs retrospective analysis of 122,465 AF patients. A

multivariable-adjusted model (HR 1.26, 95 % CI 1.23–1.29, $p < 0.001$) and a propensity-matched analysis (HR 1.21, 95 % CI 1.17–1.25, $p < 0.001$) both identified an association between digoxin treatment and higher all-cause mortality [36]. Finally, a study of incident digoxin treatment among AF patients within the Kaiser system found that after propensity score matching, incident digoxin use was associated with a 71 % higher risk of all-cause mortality (HR 1.71, 95 % CI 1.52–1.93).

Amiodarone

There is limited data available on the efficacy of amiodarone for rate control, although amiodarone does have atrioventricular nodal blocking properties. There are some data that amiodarone may be as effective as digoxin in controlling heart rate [55]. Given the poor side effect profile of amiodarone, which is discussed in greater detail below, this should not be a frequently used medication solely for rate control in AF.

Practical Guidance for Rate Control

Beta blockers and dihydropyridine calcium channel blockers can safely be started in the outpatient setting, including in the emergency department. If an assessment of the ejection fraction is not feasible prior to discharge from the emergency department, it may be better to use beta blockers rather than non-dihydropyridine calcium channel blockers, as there is less concern regarding the use of these medications in the setting of a reduced ejection fraction. Beta blockers may be less well tolerated in younger and more physically active patients, who may develop fatigue with beta blocker initiation. Beta blockers can also cause erectile dysfunction in sexually active men, which may be a greater concern among younger men. Non-dihydropyridine calcium channel blockers can cause constipation, which should be considered in older patients and patients with constipation.

Digoxin may be a less favorable medication to prescribe to patients being discharged from the emergency department based on the requirement for close follow-up, its narrow therapeutic window, and the emerging association between digoxin and mortality. Amiodarone is not a first-line rate control medication. Rather than discharging AF patients from the emergency department on amiodarone as a rate control strategy, AF patients should be evaluated by specialists for consideration of other strategies such as an atrioventricular nodal ablation with a pacemaker.

Rate control agents should be titrated to target heart rate control in the outpatient setting, where the patient is in their typical environment. It is reasonable to start at lower doses of rate control agents and titrate up as an outpatient, since tachycardia-induced cardiomyopathy generally takes weeks to develop and overdosing the rate

control agents could result in symptomatic bradycardia. Patients can also have tachycardia-bradycardia syndrome, in which their heart rates are fast in AF but are slow, causing symptoms of bradycardia, when driven by a sinus mechanism. Patients should be educated that increased fatigue, decreased exercise tolerance, dyspnea on exertion, light-headedness, or syncope may all be symptoms consistent with too much rate control medication. Patients initiated on rate control agents for AF in the ED should have outpatient physician follow-up within 2 weeks. Patients will require regular monitoring during the initiation of rate control medications in order to achieve the target heart rate goals, and once this has been accomplished, monitoring of rate control may only be needed every 6–12 months.

Rhythm Control

Flecainide

Flecainide is a use-dependent antiarrhythmic medication. This means that the antiarrhythmic properties of flecainide are stronger at faster heart rates, and this is part of the reason that all patients should be on an atrioventricular nodal blocking medication in addition to flecainide in order to prevent 1:1 atrial flutter. It is important to remember that all Vaughan Williams class IC medications (flecainide and propafenone) have a class III recommendation (evidence of harm) for use in patients with structural heart disease and coronary artery disease due to increased mortality in the CAST trials [56, 57]. Flecainide should be used at lower doses in patients with low creatinine clearance (Table 18.3). When flecainide is being used to reduce the frequency of AF relapses, it can be started as an outpatient. Periodic ECG assessment for QRS widening and renal function monitoring are necessary.

Flecainide is effective in cardioverting AF patients, and the oral formulation is as effective as the intravenous formulation. One randomized trial of oral versus intravenous flecainide for cardioversion in atrial fibrillation found that approximately 75 % of patients had restoration of sinus rhythm in both groups, but oral flecainide had a longer time to cardioversion (mean 110 min) than the intravenous flecainide (52 min, $p = 0.002$) [66].

Flecainide is also effective at maintaining sinus rhythm. Patients with paroxysmal AF were studied in a double-blind randomized crossover comparison of flecainide and placebo. Flecainide, compared with placebo, was effective at delaying the time to first AF recurrence (15 days vs. 3 days, $p < 0.001$), delaying between AF episodes (27 days vs. 6 days, $p < 0.001$), and preventing any AF episodes (31 % vs. 9 %, $p = 0.013$) [67]. The efficacy of flecainide in maintaining sinus rhythm in AF patients has been shown to be similar [68], although there was a trend toward a higher probability of persistence at 1 year with flecainide (0.62) versus propafenone (0.47, $p = 0.08$), which was driven by the gastrointestinal side effects with propafenone [69].

Table 18.3 Rhythm control agents

Generic drug name	Oral dose range	Renal dose adjustments	Primary metabolism	Excretion	Half-life	Mechanism
Vaughan Williams class Ia						
Disopyramide [58]	IR: 100–200 mg 4 times daily CR: 200–400 mg twice daily	CrCl of 40–80: 100 mg 4 times daily or 200 mg CR twice daily. CrCl 30–40: 100 mg 3 times daily. CrCl 15–30: 100 mg twice daily. CrCl <15: 100 mg daily. Avoid CR formulation for CrCl <40	Liver: CYP450 3A4	Urine: 80 % (50 % unchanged)	Mean of 6.7 h (range 4–10 h). 8–18 h with creatinine clearance <40 mL/min	Sodium channel blocker, reduction of action potential upstroke velocity (phase 0)
Quinidine gluconate extended release [59]	324–648 mg 3 times daily	CrCl < 10: dose reduce by 25 %	Liver: CYP450 3A4	Urine: 5–20 % (unchanged)	6–8 h	Sodium channel blocker, reduction of action potential upstroke velocity (phase 0)
Quinidine sulfate [60]	300–600 mg 3 times daily	CrCl < 10: dose reduce by 25 %	Liver: CYP450 3A4	Urine: 5–20 % (unchanged)	6–8 h	Sodium channel blocker, reduction of action potential upstroke velocity (phase 0)
Vaughan Williams class Ic						
Flecainide [61]	50–150 mg twice daily	CrCl of 35 or less: starting dose of 50 mg twice daily	Liver: CYP450 2D6	Urine: 30 % (unchanged)	Mean of 20 h (range 12–27 h)	Sodium channel blocker, reduction of action potential upstroke velocity (phase 0)

Generic drug name	Oral dose range	Renal dose adjustments	Primary metabolism	Excretion	Half-life	Mechanism
Propafenone [62]	IR: 150–300 mg 3 times daily ER: 225–425 mg twice daily	No dose adjustment	Liver: CYP450 2D6 CYP450 3A4	Urine: 50%	2–10 h, 10–30 h in 10% of patients (5-hydroxy metabolite deficiency)	Sodium channel blocker, reduction of action potential upstroke velocity (phase 0)
Vaughan Williams class III						
Dofetilide [63]	500 mcg twice daily	CrCl 40–60: 250 mcg twice daily. CrCl 20–40: 125 mcg twice daily	Liver: CYP450 3A4	Urine: 80% (80% unchanged)	10 h	Potassium channel blocker, blocks delayed rectifier potassium current (IKr), prolongs phase 3 of action potential
Dronedarone [64]	400 mg twice daily	No dose adjustment	Liver: CYP450 3A CYP450 2D6	Urine: 6%	13–19 h	Mechanism of action is unknown, antiarrhythmic properties from all 4 Vaughan Williams classes
Sotalol [65]	80–320 mg twice daily	CrCl 30–59: dose daily. CrCl 10–29: dose every 1.5–2 days. CrCl < 10: individualize	N/A	Urine: 66–75% (unchanged)	12 h	Nonselective antagonist of beta-receptors, prolongs phase 3 of action potential
Amiodarone [27]	100–200 mg daily	No dose adjustment	Liver	Urine: minimal	Mean of 58 days (range 15–142 days)	Noncompetitive alpha- and beta-receptor inhibitor, prolongs phase 3 of action potential

IR immediate release, *CR* controlled release, *ER* extended release, *CrCl* creatinine clearance by Cockroft-Gault in mL/min

Propafenone

Similar to flecainide, propafenone is also a use-dependent sodium channel blocker antiarrhythmic medication, and a rate control medication should be used in combination with it. Propafenone is also contraindicated in structural heart disease patients. There is no dose adjustment necessary for patients with renal dysfunction (Table 18.3). When propafenone is being used to reduce the frequency of AF relapses, it can be started as an outpatient. Monitoring is recommended every 6 months with ECG for QRS widening and blood work for electrolytes (potassium and magnesium), complete blood count (CBC), and liver function tests [62].

The Rythmol Atrial Fibrillation Trial (RAFT) was a randomized, double-blind, placebo-controlled trial of propafenone SR with three different doses (225 mg, 325 mg, and 425 mg twice daily) versus placebo for up to 39 weeks in 523 patients [70]. Patients randomized to placebo had median time to recurrence of 41 days in 69 % of the patients, while propafenone SR reduced the recurrence rates of AF to 30 % in the 425 mg arm (median time to AF of >300 days, HR 0.35, 95 % CI 0.24–0.51), 42 % in the 325 mg arm (median time to AF of 291 days, HR 0.43, 95 % CI 0.31–0.61), and 52 % in the 225 mg arm (median time to AF of 112 days, HR 0.67, 95 % CI 0.49–0.93) [70]. Similar findings were seen in the European Rythmol Atrial Fibrillation Trial (ERAFT) with fewer patients (293 patients) [71]. In an additional randomized double-blind, placebo-controlled trial, 63 % of patients randomized to propafenone were in sinus rhythm at 1 year, which was significantly higher than the 35 % in the placebo group ($p = 0.001$) [72].

As discussed in the above section on flecainide, propafenone has been demonstrated to have similar safety and efficacy to flecainide, although there was a trend in one study toward a higher rate of adverse events with propafenone, which was driven by gastrointestinal side effects [68, 69]. The data are mixed on whether propafenone has similar efficacy or greater efficacy in maintaining sinus rhythm, relative to sotalol [72, 73]. In a series of controlled trials, amiodarone was more effective than propafenone at preventing the recurrence of AF [73–75].

Pill in the Pocket

Flecainide and propafenone have been prospectively evaluated and shown to be safe and effective for use as an as-needed medication for paroxysmal AF [76]. This medication strategy is known as "pill in the pocket" because patients can carry their medication with them and take a dose, when they develop symptomatic palpitations in order to pharmacologically cardiovert them from AF to sinus rhythm, as outpatients. The prospective study evaluated the "pill-in-the-pocket" strategy with flecainide or propafenone in 210 patients, finding that 165 patients (79 %) had 618 outpatient recurrences of AF over a mean follow-up time of 15 months. The "pill-in-the-pocket" strategy successfully restored sinus rhythm in 94 % of the treated

episodes, while only 1 patient (0.6 %) reported a cardiac adverse event of atrial flutter with 1:1 conduction after taking the antiarrhythmic medications [76].

It is important to understand that this was a selective patient population, as patients needed to have recent onset (<48 h) and symptomatic AF, as well as an absence of preexcitation, QRS >120 ms, structural heart disease, or long QTc interval. Patients had to have a safe and successful pharmacologic cardioversion with flecainide or propafenone that was observed in the emergency department (51 % of patients) or the inpatient setting before being able to use the "pill-in-the-pocket" strategy. Among patients that received a monitored dose of flecainide or propafenone, 15 % did not have restoration of sinus rhythm, and 5 % had adverse events, including hypotension, conversion to atrial flutter, and bradycardia [76].

The "pill-in-the-pocket" dosing for flecainide is 300 mg for patients that weigh 70 kg or more and 200 mg for patients that weigh less than 70 kg, and the dose for propafenone is 600 mg for patients that weigh 70 kg or more and 450 mg for patients that weigh less than 70 kg.

Disopyramide

Disopyramide is best known for its reduction in symptoms and gradient in patients with hypertrophic obstructive cardiomyopathy, as it has a negative inotropic effect [77]. Although disopyramide can be started in the outpatient setting, ECGs should be monitored periodically for QRS widening of greater than 25 % from baseline [58]. In addition to being a sodium channel blocker, disopyramide also has vagolytic properties. Disopyramide is not a first-line agent for AF, although it has been demonstrated to be more effective than placebo at maintaining sinus rhythm in a randomized double-blind trial comparing disopyramide versus placebo in AF patients after electrical cardioversion. In this trial, patients randomized to disopyramide versus placebo were more likely to be in sinus rhythm at 1 month (70 % vs. 39 %, $p<0.01$) and 12 months (54 % vs. 30 %, $p<0.01$) [78]. Disopyramide was also compared with propafenone after cardioversion of persistent AF patients, and, in the disopyramide arm, 71 % of patients were in sinus rhythm at 3 months and 67 % were in sinus rhythm at 6 months, which was statistically similar to the propafenone arm [79].

Quinidine

A meta-analysis of randomized controlled trials identified six trials between 1970 and 1984 that included quinidine and placebo arms and a total of 808 patients. The meta-analysis found that quinidine was more successful at maintaining sinus rhythm compared with placebo at 3 months (69 % vs. 45 %, $p<0.001$), 6 months (58 % vs. 33 %, $p<0.001$), and 12 months (50 % vs. 25 %, $p<0.001$); however, there was an association between quinidine and higher mortality (HR 2.98, $p<0.05$) [80].

An open-label randomized trial of quinidine versus sotalol in Swedish AF patients after cardioversion found that quinidine and sotalol had similar rates of AF recurrence (22 % and 34 %, respectively, p = nonsignificant) and a similar proportion of patients in sinus rhythm at 6 months (48 % and 52 %, respectively, p = nonsignificant), but a higher proportion of patients had to stop quinidine due to adverse effects (26 % vs. 11 %, $p < 0.05$) [81]. Quinidine should be started in the hospital with monitoring for QRS widening greater than 130 % of the baseline value. In chronic therapy with quinidine, periodic monitoring of CBC, liver function tests, and kidney function is necessary [59].

Dofetilide

Dofetilide is a reverse use-dependent antiarrhythmic medication, meaning that its antiarrhythmic properties are more pronounced at slower heart rates. Given the fact that dofetilide prolongs the QTc interval, caution is necessary when using dofetilide in bradycardic patients, as stronger antiarrhythmic properties at these lower heart rates can lead to even greater QTc prolongation. Patients being started on dofetilide must be monitored in the hospital for the first 5 doses of the medication, and only certified prescribers can use dofetilide [63]. Dofetilide can be used safely, but it is important to have a program in place to ensure its proper use by monitoring the QTc interval after every dose, potassium and magnesium daily, and contraindicated concomitant medications (such as verapamil, hydrochlorothiazide, cimetidine, etc.) [82]. After dofetilide initiation, patients chronically on dofetilide should have an ECG for QTc monitoring and a chemistry for renal function and electrolyte assessment performed every 3 months [63].

Dofetilide is the second most effective antiarrhythmic medication behind amiodarone. The Symptomatic Atrial Fibrillation Investigative Research on Dofetilide (SAFIRE-D) trial was a randomized, double-blind, placebo-controlled trial that found that dofetilide was superior to placebo at maintaining sinus rhythm at 1 year after cardioversion with 25 % of the placebo arm patients maintaining sinus rhythm, compared with 58 %, 37 %, and 40 % of patients on dofetilide 500 mcg, 250 mcg, and 125 mcg, respectively ($p = 0.001$) [83].

Dofetilide has been well studied in patients with heart failure, and it and amiodarone are the only first-line agents for patients with heart failure with reduced ejection fraction. This came out of the Danish Investigations of Arrhythmia and Mortality on Dofetilide-Congestive Heart Failure (DIAMOND-CHF) trial, which was a double-blinded, placebo-controlled trial evaluating the safety and efficacy of dofetilide in 1518 patients with severe left ventricular dysfunction during a median follow-up of 18 months [84]. The rates of all-cause mortality were similar in the dofetilide (41 %) and placebo (42 %) arms, while patients in the dofetilide arm were more likely to convert from AF to sinus rhythm and were more likely to remain in sinus rhythm [84]. A similar study was done by the same study group in 1510 patients with myocardial infarction within the last 7 days (DIAMOND-MI trial),

and, again, the dofetilide arm had similar mortality but higher rates of sinus rhythm relative to the placebo arm [85]. A pooled analysis of the two DIAMOND trials found that dofetilide was more effective than placebo at maintaining sinus rhythm at 1 year (79 % vs. 42 %, $p < 0.001$) [86].

Amiodarone

Amiodarone is the most effective antiarrhythmic in AF, and 65 % of AF patients were without recurrence of AF at 16 months on amiodarone [74]. Similarly, from the AFFIRM study, 62 % of patients on amiodarone were in sinus rhythm at 1 year, which was higher than class I agents or sotalol [87]. Based on mixed treatment comparisons of randomized controlled trials, amiodarone was the most effective antiarrhythmic at reducing AF recurrence (OR 0.22, 95 % CI 0.16–0.29), but amiodarone was the antiarrhythmic with the highest rate of medication discontinuation due to adverse events (OF 2.91, 95 % CI 1.66–5.11) [88]. Amiodarone has been shown to be more effective than sotalol and dronedarone in direct comparison randomized controlled trials [5, 89]. Amiodarone can be started as an outpatient. Although QTc prolongation may occur, it infrequently leads to torsade de pointes. Amiodarone use is plagued by multiple drug-drug interactions of which the prescribing physician must be aware, including with commonly used drugs in the AF population (e.g., digoxin, warfarin, and multiple statins, among many others), for which proper dose adjustments are necessary. A useful tool to investigate amiodarone drug-drug interactions is available at the Drugs.com website (http://www.drugs.com/drug-interactions/amiodarone.html).

Given the concerns about adverse events with amiodarone, it is a second-line agent for AF patients without structural heart disease and with coronary artery disease, and it is a first-line agent in patients with heart failure [10]. A meta-analysis of 1465 patients on low-dose amiodarone with a mean dose of 152–330 mg daily for at least 12 months found that relative to placebo, amiodarone was associated with higher rates of thyroid (OR 4.2, 95 % CI 2.0–8.7), neurologic (OR 2.0, 95 % CI 1.1–3.7), skin (OR 2.5, 95 % CI 1.1–6.2), and ocular (OR 3.4, 95 % CI 1.2–9.6) adverse events. There was a trend toward higher pulmonary toxicity with amiodarone (OR 2.0, 95 % CI 0.9–5.3). Patients should have liver and thyroid function tests performed every 6 months, and a baseline pulmonary function test with diffusion capacity (D_LCO) prior to starting amiodarone is important to have as a comparison to future tests, which should be ordered in the event of pulmonary symptoms, for diagnosing pulmonary toxicity [90, 91].

Beyond the side effect profile of amiodarone, there are some concerns about the safety of amiodarone on outcomes of AF patients. Based on data from the Apixaban for Reduction in Stroke and Other Thromboembolic Events in Atrial Fibrillation (ARISTOTLE) trial, patients on amiodarone and warfarin had a lower percentage of time in the therapeutic range (57 % vs. 63 %, $p < 0.0001$), and amiodarone was associated with higher rates of stroke or systemic embolism (HR1.47, 95 % CI 1.03–2.10, $p = 0.032$) [92].

Dronedarone

Dronedarone was developed from amiodarone without the iodine moieties in an effort to improve the side effect profile while preserving the superior efficacy of amiodarone. Like amiodarone, dronedarone can also be started as an outpatient, and monitoring should be done every 3 months with an ECG, electrolytes, and creatinine, as well as periodically for liver function tests [64]. Dronedarone was shown in European and non-European double-blind, randomized trials to be superior to placebo with a longer median time to AF recurrence and lower mean heart rate when in AF. Further, the rates of pulmonary, thyroid, and liver toxicity were similar in the placebo and dronedarone arms [93]. A Placebo-Controlled, Double-Blind, Parallel Arm Trial to Assess the Efficacy of Dronedarone 400 mg BID for the Prevention of Cardiovascular Hospitalization or Death from any Cause in Patients with Atrial Fibrillation or Flutter (ATHENA) trial was a larger double-blinded randomized controlled trial comparing dronedarone with placebo in 4628 patients with AF over a mean follow-up of 21 months. The primary outcome was the first of cardiovascular hospitalization or death [94]. Dronedarone reduced the primary outcome with an HR of 0.76 (95% CI 0.69–0.84, $p<0.001$), and thyroid and pulmonary adverse events were similar in the dronedarone and placebo arms of the trial [94].

Dronedarone does have a black box warning for class IV heart failure or class II or III heart failure with a recent decompensation based on data from the Antiarrhythmic Trial with Dronedarone in Moderate to Severe CHF Evaluating Morbidity Decrease (ANDROMEDA) trial, in which patients with severe heart failure and left ventricular dysfunction who were treated with dronedarone had increased early mortality due to worsening heart failure [95]. Dronedarone is also contraindicated in patients with permanent AF based on the findings from the Permanent Atrial Fibrillation Outcome Study Using Dronedarone on Top of Standard Therapy (PALLAS) trial, which identified a higher risk of heart failure, stroke, and cardiovascular death in permanent AF patients treated with dronedarone versus placebo [96]. Although there are fewer side effects with dronedarone than with amiodarone, dronedarone also has inferior efficacy for the maintenance of sinus rhythm, relative to amiodarone [89, 97].

Sotalol

Sotalol is another reverse use-dependent antiarrhythmic medication. Sotalol has significant beta-blocking properties at oral doses as low as 25 mg, and its class III effects are stronger at doses above 160 mg daily [98]. Regardless, it is still reasonable to prescribe traditional beta blockers in addition to sotalol. Sotalol is a first-line agent for patients without structural heart disease and for patients with coronary artery disease [10]. Similar to dofetilide, patients should be hospitalized for the initiation of sotalol, and ECGs should be performed 2–4 h after each of the first 5 doses to

monitor for QTc prolongation [65]. After initiation, patients should have an ECG done to evaluate QTc and a chemistry for renal function assessment every 6 months.

Sotalol was studied against placebo in a double-blind, randomized controlled trial to assess for the maintenance of sinus rhythm. Patients on placebo had AF recurrence at a median of 27 days, while sotalol patients had AF recurrence at medians of 106, 229, and 175 days for the 80 mg, 120 mg, and 160 mg arms, respectively [99]. As discussed in the sections on propafenone and quinidine, sotalol has similar efficacy to propafenone and quinidine [72, 75, 100]. Sotalol was inferior to amiodarone in a double-blind, randomized trial evaluating time to AF recurrence in 665 patients; time to recurrence of AF was longer in the amiodarone arm (median of 487 days), compared with 74 days in the sotalol arm ($p < 0.001$) [5].

Conclusion

There are many options for outpatient rate control and rhythm control in patients with AF. All AF patients need to have their heart rate controlled, and antiarrhythmic medications are recommended in patients with symptomatic AF, despite rate control. It is important to consider patients' comorbidities such as the presence of structural heart disease, coronary artery disease, or heart failure, when selecting an antiarrhythmic medication. Renal function is also important for the dosing of many antiarrhythmic medications.

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Part IV
Systems Management

Chapter 19
Atrial Fibrillation Accreditation

Philip Beckley and Donna Hunn

Introduction

Atrial fibrillation (AF) has been described as a worldwide epidemic with a huge clinical and economic burden to patients and hospitals [1–3]. In the United States alone, the prevalence of this condition was determined to be 5.2 million patients in 2010 and is expected to rise to 12.1 million patients by 2030 [4]. Since AF incidence increases with age, this rise in prevalence is due, in part, to the aging population and the increasing coincidence of risk factors such as hypertension, obesity, diabetes, and sleep apnea. Not surprisingly, AF hospitalizations, readmissions, and costs have risen sharply. It has been determined that emergency department (ED) visits for AF-related complaints increased by 88 % between 1993 and 2003, and more than 65 % of these ED visits resulted in a hospital admission [5]. At the American College of Emergency Physicians' 2014 Scientific Assembly, Dr. Barrett was quoted as saying that "admitting nearly seven of ten patients with acute atrial fibrillation is not sustainable, as the prevalence of atrial fibrillation in the United States continues to rise each year" [7]. One study reported that hospital admissions and mean costs have increased by 23 % and 24 %, respectively, from 2000 to 2010 [8]. Extending that time period, Pant et al. found that the cost of AF hospitalizations will rise another 55 % from 2010 to 2020 [9]. It is generally agreed that our current processes to deliver care to the AF patient must change in order to reduce the clinical and economic burden on the healthcare system. It has been suggested that practices must be put in place that incorporate clinical practice guidelines, timely risk assessment, expedited cardioversion, and effective use of observation and outpatient medicine to limit AF hospitalizations and readmissions.

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In response to this AF epidemic and the resulting clinical and economic burden, the Society of Cardiovascular Patient Care (SCPC) has developed Atrial Fibrillation Accreditation. The SCPC is a nonprofit organization committed to leading the fight to eliminate heart disease as the number one cause of death worldwide. Its primary mission is to develop and share quality practices that optimize the care and outcomes of patients with acute cardiovascular disease through innovative cross-disciplinary processes and education that brings science to the bedside. The SCPC has been developing and offering accreditation services to hospitals since 2003 and currently also offers Chest Pain Center (CPC) Accreditation and Heart Failure Accreditation services. SCPC accreditation has been shown to improve performance on both acute myocardial infarction and heart failure quality measures [10, 11]. Peacock et al. studied participating ACTION Registry-GWTG hospitals to determine if achieving SCPC CPC Accreditation changed outcomes over the course of a year following accreditation [12]. It was found that median door-to-PCI time, in-hospital mortality, and median hospital length of stay were all lower 1 year following accreditation.

Through AF accreditation, hospitals are able to (1) incorporate recognized recommendations and best practices into the assessment, treatment, and management of AF patients, (2) improve risk assessment and provide structure to disposition decisions to avoid unnecessary admissions, (3) engage an integrated team to effectively and efficiently manage AF patients across all transitions of care, and (4) align processes with evidence-based science to improve quality of care which also impacts patient satisfaction and cost efficiencies. In short, SCPC accreditation serves to drive the implementation of evidence-based guideline-driven clinical practice, process improvement, and up-to-date performance measurements, across the full continuum of care for AF patients – from the community through the ED-observation-inpatient experience and back out into the community.

Development of Atrial Fibrillation Certification

In 2010, the SCPC recognized the need to develop a service line for AF. At that time, Chest Pain Accreditation was in its third 3-year cycle and Heart Failure Accreditation was in the first year of its first 3-year cycle. Both had been shown to effectively provide the tools needed by hospitals to improve processes and quality of care and it was assumed that similar tools would benefit those seeking to improve processes for AF patient care. An atrial fibrillation panel of experts, represented by a variety of clinical disciplines, was assembled to determine what recognized guideline recommendations and best practices were to be considered by hospitals seeking to improve processes for patient care.

Atrial Fibrillation Certification, Cycle 1, was launched in July 1, 2011. This set of tools was released as a certification rather than accreditation since AF was viewed at that time as a condition related to both heart failure and chest pain. AF Certification differed from the Chest Pain and Heart Failure Accreditations in only

two ways – (1) the hospital was first required to have achieved Chest Pain Accreditation and (2) the award was based on a desk review of the application and documentation without a site visit. The AF Certification tools consisted of 41 Item Statements from guideline recommendations and recognized best practices and an instrument which was used to collect data pertaining to annual patient and procedure volumes, treatment strategies, length of stay, and performance measures. Upon the first and second anniversaries of the 3-year cycle of the accreditation award, the AF Coordinator was required to resubmit data so that the Accreditation Review Specialist (ARS) could follow trends in data and measures and advise the hospital on further process improvement that may be needed.

The Item Statements in the tool were arranged into ten Key Elements which represented logical categories for process improvement:

1. Emergency Department Integration with the Emergency Medical Services
2. Emergency Assessment of Patients with Symptoms
3. Risk Stratification
4. Treatment for Patients Presenting to the Emergency Department
5. Discharge Criteria from the Emergency Department and/or Observation Stay
6. Education in the Emergency Department and Observation Unit
7. Personnel, Competencies, and Training
8. Process Improvement
9. Organizational Structure and Commitment
10. Community Outreach

Subsets of the Key Elements, called Essential Items, further divided the Key Elements into logical subcategories of guideline recommendations and best practices. Under the guidance of ARS, the AF Coordinator and hospital care teams performed a "gap analysis" of each Item Statement contained in each of the Essential Items to determine which of the recommendations or best practices were already in place and which would require some process improvement to put into place. The teams were instructed that, in order to achieve accreditation, compliance with a specific quantity of Item Statements would need to be met under each of the Essential Items. The ARS would use documentation provided by the AF Coordinator to determine if compliance with an Item Statement was indeed met.

During the first 3-year cycle (July 1, 2011 to June 30, 2014), 45 hospitals from 17 states achieved AF Certification. Florida had the most hospitals which earned certification (13), followed by Nebraska (5) and California (4). It would not seem to be a surprise that Florida led the way due to its large elderly population and the recognition that improvement in processes would improve care. Thirty-seven of the 45 hospitals (82%) offered electrophysiology services (EPS) which included EP studies, catheter-based ablations, surgical ablations, and left atrial appendage exclusion and/or occlusion. However, AF Certification did not distinguish between EPS hospitals and non-EPS hospitals with respect to the certification award. EPS hospitals were significantly different from non-EPS hospitals in the number of licensed beds, average daily census, and the number of AF patients (both primary and secondary) discharged from an inpatient unit (Table 19.1). EPS hospitals were not significantly

Table 19.1 Selected average data from EPS and non-EPS hospitals

	EPS hospitals	Non-EPS hospitals	
Licensed beds	402	236	$p<0.01$
Average daily census	232	85	$p<0.01$
AF patients (1° and 2°) discharged from the ED (most recent full year)	363	204	N.S.
AF patients (1° and 2°) discharged from an inpatient unit (most recent full year)	1863	467	$p<0.01$
ED length of stay (hours)	4.6	3.7	N.S.
Observation length of stay (hours)	26.4	23.5	N.S.
Inpatient length of stay (hours)	95	79.5	N.S.
% ED patients who experienced a spontaneous cardioversion	8.1	6.8	N.S.
% ED patients who received an emergent cardioversion	10.6	3.3	N.S.
% ED patients initially assigned to a rate-control strategy	76.6	88.3	N.S.
% ED patients initially assigned to a rhythm-control strategy	20.4	9.8	N.S.

1° primary AF, *2°* secondary AF, *N.S.* not significantly different

different from non-EPS hospitals with regard to the number of AF patients discharged from the ED, length of stay (ED, observation unit, and inpatient unit), and percent of patients who experienced a spontaneous cardioversion, received an emergent cardioversion, or were initially assigned to a rate- or rhythm-control strategy.

Hospitals that achieved AF Certification reported recognized improvements in a variety of clinical and process areas derived from guideline recommendations and best practices:

- Integration of facility goals with the local Emergency Medical Services (EMS)
- Processes for an immediate ECG and early assessment
- Processes for safe DC cardioversion based on established guidelines
- Thromboembolic risk assessment and proper prescription of oral anticoagulants
- Clinical pathways for rate/rhythm control and disposition
- Well-documented and individualized discharge processes
- Patient education related to lifestyle modification and strategies for AF recurrence
- Medical staff education on all aspects of AF assessment and management
- Organizational structure supportive of process evaluation and improvement
- Community and healthcare provider education

The hospitals also reported, in follow-up surveys, what benefits were realized as a result of AF Certification:

- Improved coordination of AF patients along the continuum of care
- Improved operational efficiencies in the care of AF patients

- Improved patient throughput
- Has stimulated process improvement activities
- Patients risk stratified more appropriately to prevent stroke
- Increased community awareness of AF symptoms and treatment

Development of Atrial Fibrillation v2 Accreditation

The next release (July 1, 2014, to June 30, 2017) was launched as Atrial Fibrillation v2 Accreditation. A complete online platform had been developed for Heart Failure Accreditation a year before, and it was decided that Chest Pain Accreditation would adopt this new platform and AF Certification would be promoted to an accreditation and adopt this new platform as well. AF Accreditation would not require a pre-awarded Chest Pain Accreditation and would include a site visit at the conclusion of the application process.

The online platform for Atrial Fibrillation v2 Accreditation has numerous features:

- Specifically developed to be identical with the chest pain and heart failure platforms so that individual hospitals and hospital systems would be comfortable in switching between multiple accreditation applications.
- The ten Key Elements of Cycle 1 were restructured into seven Essential Components which are also common to the chest pain and heart failure disciplines.
- Item Statements, still derived from recognized guideline recommendations and best practices, are arranged into mandatory, recommended, and innovative categories. It was determined, under the guidance of the Writing Committee, that certain recommendations and best practices are absolutely required in order to achieve quality in patient care. Hospitals striving to earn AF Accreditation must achieve compliance with all mandatory items. Recommended and Innovative Items are provided to allow the hospital to perform above the standard.
- Item drop-down menus have accompanying guidance which explains the intent of the statement and provides definitions, reference links to the science behind the item, and shared practice examples that can be edited and adopted by the hospital.
- Documentation which provides support for compliance can be uploaded to accompany Item Statements, and an associated text box is provided to allow the AF Coordinator to write comments which may be of help to the ARS.
- The data collection instrument which was a companion to the Cycle 1 tool is replaced with a patient database called the Accreditation Conformance Database (ACD). Hospitals enter data for patients with a primary discharge diagnosis code of AF (currently ICD-9 427.31). Data fields are provided to capture information related to patient demographics, past medical history, early stabilization, acute care, diagnostics, evaluations, and transitions.

- Selected data fields in the ACD are used to automatically calculate various performance and outcome measures. These calculated measures are updated each time new patient data is entered so that the hospital can quickly see trends and institute corrective actions if needed. Data can be viewed in a real-time format as well as retrospective in monthly and year-to-date reports. A drill down feature allows the user to pull the patient records used to calculate the measure so that other relationships in data can be discovered. The ACD helps to measure current performance and outcome processes and target those that are not meeting goals and thresholds. Subsequent changes in processes are guided by the content found in the accreditation tool. It is the combination of the ACD and the tool that brings the ultimate value to accreditation.

Seven Essential Components define the key areas of consideration for Atrial Fibrillation v2 Accreditation (listed with examples of content):

1. Governance – develop educational AF programs to meet the needs of physicians, providers, and staff, ensure competencies and skills, and monitor compliance with accreditation goals.
2. Community Outreach – raise awareness and educate the public and businesses, educate primary and specialty care providers and staff, and integrate your hospital with the community.
3. Prehospital Care – integrate your hospital with first responders and healthcare providers, identify measures for process improvement, and educate the EMS to better care for AF patients.
4. Early Stabilization – develop protocols for triage and initial treatment, develop risk assessment strategies, and implement evidence-based guideline-driven care for the early management of AF patients.
5. Acute Care – develop protocols for ED and inpatient management and guide patient care through rate control, rhythm control, and prevention of thromboembolism.
6. Transitions of Care – develop discharge processes that ensure effective transition of care, educate, build patient compliance, and provide follow-up.
7. Clinical Quality Measures – develop processes to capture performance and outcome data and visualize numerous calculated measures that compare actual performance with your thresholds and goals.

Eight categories of information define the data fields included in the ACD (listed with examples of content):

1. Patient – demographics, insurance, arrival information, and previous hospitalizations
2. Past Medical History – comorbidities and risk factors, social history, and meds from home
3. Prehospital Care – chief complaint, vital signs, ECG, medications administered, and interventions
4. Early Stabilization – ED arrival, chief complaint, ECG, AF classification, vital signs, ED medications, and interventions

5. Acute Care – admit to and by, in-hospital conditions, and procedures
6. Diagnostics – lab tests and results, x-ray, TTE, and TEE
7. Evaluations – consults, risk assessments, and scores
8. Transitions – discharge from and to, vital signs, medications, post-discharge education, and follow-up

Accreditation Process

Following the purchase of AF v2 Accreditation Services, a hospital moves through three distinct phases. An ARS is assigned to the hospital following the purchase to provide guidance and answer questions related to the mechanics of using the online tool, the intent and objective of each Item Statement, the appropriate documentation to be attached to the Item Statements, and the trending of calculated measures.

Baseline Gap Analysis Phase This first phase extends from purchase to the submission of a baseline gap analysis and baseline patient data. This phase has a 30-day deadline from the first log-in to the tool and ACD. A gap analysis is performed on all Item Statements in the tool to demonstrate to the hospital and the ARS which processes are fully in place at the time of purchase and, obviously, which are not and will require some improvement. This baseline analysis serves as a guide to organizing the work that needs to be done to achieve AF Accreditation. Basic facility information and a full list of contacts are also entered into the online tool. Thirty recent patients with an AF discharge diagnosis code are entered in the ACD to provide a baseline calculation of the performance and outcome measures. This set of calculated measures serves as a starting point for the demonstration of improvement as the accreditation process continues.

Application Phase The second phase extends from the Baseline Submission to the granting of accreditation. During this phase, the hospital hardwires all of the processes needed to meet the intent and objective of each mandatory Item Statement. Recommended and innovative Item Statements are considered for possible improvements. Supporting documentation for all Item Statements is collected and uploaded as an attachment to each Item Statement that is marked Yes. An ACD data submission plan is chosen at the start of this phase which allows monthly entry of all patient data or a monthly minimum sampling of data. The performance and outcome measures are recalculated daily so that positive or negative improvement trends can be documented. The final steps in this phase are the submission of the accreditation application, the review of the application by the ARS, the site visit, and the approval and granting of accreditation by the Accreditation Review Committee. The hospital has 1 year from the first log-in to the tool and ACD to submit the application for consideration.

Accreditation Phase The third phase extends from the granting of accreditation to the anniversary date. Following the site visit, the ARS sends a full report to the hospital which provides comment on each of the Item Statements and well as an Executive Summary which lists noteworthy observations and opportunities for each of the Essential Components. The stated opportunities serve as a guide for continued process evaluation and improvement. Monthly patient data is entered into the ACD during the Accreditation Phase to provide continued calculations of performance and outcome measures. The ARS remains assigned to the hospital to provide continuous feedback and support.

Why Pursue Atrial Fibrillation v2 Accreditation

AF v2 Accreditation provides the tools to identify a guideline recommendation or best practice, determine the gap between your current and ideal clinical practice, improve the processes that are necessary to achieve the ideal practice, and monitor your progress in meeting that goal. For example:

- Mandatory Item Statement – The facility utilizes a validated tool to assess the risk of thromboembolism (CHA_2DS_2-VASc score).
- Your care team determines that this guideline recommendation is only partially being met. Some of your physicians and providers are using the assessment tool but you have no idea how many. Others are unaware of it, not using it, or not using it correctly.
- For your Baseline Submission gap analysis, you check that you are *not* compliant with this recommendation.
- For your Baseline ACD Submission, you respond to the inquiry in the database as best as you can with the information found in your 30 most recent patient discharge records:
  - ACD inquiry – CHA_2DS_2-VASc stroke risk assessment calculation completed
- When your baseline calculated measures report is shown in the tool, you find that the "percentage of patients with nonvalvular AF in whom assessment of thromboembolic risk factors using the CHA_2DS_2-VASc risk criteria has been documented" shows a value of 20 %. Now you have a reasonable estimate of how often your physicians and providers are using this assessment tool.
- As AF coordinator, you break down this problem into its component parts and assign tasks to various members of your care team:
  - Research the correct use of the assessment tool, the information it provides, and how to use the results.
  - Incorporate the assessment tool into the AF order sets or the electronic medical records system.

  - Begin a campaign to educate physicians and providers on the importance of the assessment and how to use the tool as found in the AF order sets.
  - Ensure that documentation of the use of this tool is included in the discharge record.

- Your care team spends the next weeks and months educating and documenting.
- As time passes, the monthly report of this calculated measure gradually increases in value. By the time you are ready to submit your accreditation application, you can check that your facility is compliant with this mandatory Item Statement, and you also find that 93 % of your 165 ACD patient data entries have had the risk score performed at some point prior to discharge.
- Congratulations!

As the Society works with hospitals in the accreditation journey, it has become quite evident that important processes are not in place at the time of the Baseline Submission. At the time of this writing, 14 hospitals have either been fully accredited or are in the course of pursuing accreditation. The Baseline Submission from each of these hospitals revealed that 70 % or more of them did *not* have the following in place:

- Protocols for the initial assessment and evaluation of patients with suspected AF
- Protocols to determine the etiology and/or factors predisposing patients to AF
- Protocols that direct treatment along a rate-control or rhythm-control strategy for AF
- Standardized order sets for the initial assessment, evaluation, and management of AF patients
- Protocols for disposition decisions, including specific criteria to identify patients appropriate for admission and observation as well as those who may be discharged directly home
- Processes to conduct AF case reviews with the medical staff for purposes of continuing education and process improvement
- Processes to provide AF education to the nursing staff on signs and symptoms and the process to follow when an inpatient develops AF
- Processes to provide education and/or training related to treatment protocols, precipitating factors, and signs and symptoms of AF to all nurses caring for AF patients
- Processes to provide specialty-specific AF care education and training to ancillary healthcare providers and staff
- Processes to provide formal training to triage staff on protocols and/or policies associated with AF

Accreditation Services for AF Accreditation provides the educational and organizational support to be successful:

- Assigned Accreditation Review Specialists who offer personalized support
- Workshops to learn how to navigate the tools, build your care teams, perform the gap analysis, and manage your accreditation initiative

- Instructional videos to take you step by step through the mechanics of using the tools
- Guidance with each of the Item Statements to provide the background and intent of the statement
- References with each of the Item Statements to help search out the science and clinical practice related to the statement
- Educational opportunities on current science, process, and management including an annual Congress for continuing education and networking
- Weekly Ask-the-Expert call-in sessions to review Item Statement and ACD data field objectives and answer questions
- The opportunity to see shared practice documents from other hospitals that may be edited to include your specifics and adapted to your processes
- Consultative site reviews
- Recommendations for ongoing improvement after accreditation

With the incidence and prevalence of AF reaching staggering proportions in the next two decades and beyond, AF v2 Accreditation, offered by the Society of Cardiovascular Care, positions the hospital to meet the needs of this unique population. The tools have been designed to help you:

- Critically examine the continuum of processes from prehospital care through the hospital stay and back out into the community.
- Identify gaps between present practice and excellence in care and improve processes to achieve goals.
- Apply recognized evidence-based guideline recommendations and best practices to the assessment, treatment, and management of AF patients.
- Redesign services to maximize effectiveness.
- Ensure that your local EMS has the education, training, and support that they need for routine and emergent care of patients in your community.
- Improve early assessment of patients to ensure appropriate discharge or disposition based on clinical presentation, comorbidities, and response to treatment and to avoid unnecessary hospital admissions.
- Ensure that patients are risk stratified for potential thromboembolic events and are prescribed anticoagulation when appropriate.
- Ensure that guideline-directed DC and pharmacologic cardioversion protocols are in place.
- Improve transitions of care, discharge AF-specific education, and follow up processes to engage the patient and avoid unnecessary rehospitalization.
- Engage the care team to effectively and efficiently manage AF patients across all transitions of care.
- Network and collaborate with community primary care physicians to ensure that they are also able to deliver evidence-based guideline-driven care.
- Provide real-time monitoring of patient data and calculate performance and outcome measures to give you an up-to-date snapshot of how your facility is meeting its thresholds and goals.

- Align processes with evidence-based science to improve the quality of care which also impacts patient satisfaction and cost efficiencies.
- Demonstrate a commitment for the care of AF patients to the staff of your hospital and individuals in your community.

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Chapter 20
Quality Metrics in Atrial Fibrillation

Christopher William Baugh

Introduction

Observation unit leaders rely on metrics to inform a data-driven understanding of the care delivered in their unit. These data are typically reported at regular intervals (e.g., monthly) to a physician, nursing, and administrative leader. Most metrics are applicable across all conditions/protocols (i.e., daily patient throughput, rate of subsequent inpatient admission) but others can be more specific to individual protocols (e.g., rate of successful cardioversion for atrial fibrillation). The most important time to closely track and report metrics for a specific protocol is the period after it is newly launched or significantly updated. Leaders share data to stakeholders both within and outside their department, both clinical and nonclinical. The data are presented in various formats to optimize the message for the intended audience.

As discussed elsewhere in this book, a subgroup of patients arriving to the emergency department (ED) with atrial fibrillation and atrial flutter are good candidates for continued outpatient management in an observation unit. When initiating or maintaining an observation protocol for atrial fibrillation, creating, tracking, and reporting metrics specific to this protocol are essential tasks. Metrics should be accurate, up to date, important, and actionable.

A successful atrial fibrillation observation unit strategy relies heavily on continuous oversight and data collection. Key metrics inform volume, efficiency, safety, and compliance with evidence-based guidelines. Information systems should be optimally configured to feed into a report or dashboard with timely and accurate data. In addition, chart review may be needed to fully capture important details of the clinical encounter

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not easily abstracted automatically. When possible, department-wide resources should be leveraged; for example, a nurse may be assigned to complete a chart review on all procedural sedation cases. This nurse could inform metrics specific to the atrial fibrillation observation protocol as part of his or her routine chart review.

In this chapter we provide an overview of suggested metrics for all observation visits as well as those specific to an atrial fibrillation protocol. An administrator should provide these metrics in a dashboard format that is easy to interpret, including graphs, and other tools optimized to effectively communicate data. The use of statistical process control charts is another effective method to identify when trends in the data are significant.

Metrics Common to All Protocols

Benchmarks may vary by setting and patient population. Trends are likely to be more useful for tracking process improvement efforts.

Volume

- Monthly patient volume by protocol
  - Benchmark: Chest pain is the most common observation condition at about 20 % of visits; atrial fibrillation should be expected to comprise less than 5 % of all ED observation volume. Observation units typically have multiple protocols, sometimes over 20 [1, 2].
- Observation volume as a percentage of overall ED visit volume
  - Benchmark: Total ED observation volume typically runs between 5 and 10 % of ED visit volume, with increasing use over time after establishing a dedicated observation unit [3].
- Volume of ED visits resulting in observation stays managed by inpatient teams:
  - Benchmark: There is no consensus benchmark for the appropriate number of observation patients managed outside of dedicated observation units. Some hospital administrators would argue this number should be near zero, but recent payer policy changes (e.g., the Centers for Medicare and Medicaid Services' "2-Midnight Rule") have broadened the definition of observation, and as a result, some observation patients may be inappropriate for traditional ED observation units [4]. Alternatively, there may be times when the observation unit is at or over capacity and the most reasonable plan is to admit to an inpatient team.

Timing/Efficiency

- Emergency department length of stay by protocol (mean, median, outliers over 6 h)
  - Benchmark: There is no widely accepted benchmark for this time interval; one could compare the length of stay compared with those patients discharged directly home or admitted as a frame of reference. Prolonged ED length of stay prior to an observation visit may indicated an unnecessary delay in decision making.
- Observation length of stay by protocol (mean, median, outliers below 6 h and over 24 h)
  - Benchmark: The average observation length of stay is about 15 h across all conditions. Patients with atrial fibrillation may cluster into short- and long-stay groups, depending on whether early an cardioversion or rate control strategy is used [5].
- Inpatient conversion rate (overall, by protocol and by initial attending)
  - Benchmark: The average inpatient conversion rate clusters around 20 % and may be higher for more challenging conditions (e.g., congestive heart failure) [6]. Atrial fibrillation patients would be expected to convert at or just above the 20 % threshold. At the attending level, outliers can be identified who select patients inappropriate for observation (i.e., patients who should have been directly discharged if the conversion rate is too low or patients who should have been initially admitted if the conversion rate is too high)
- Rate and duration of boarding for patients converted to inpatient status
  - Benchmark: There is no consensus value for boarding in observation units, but measuring changes over time in your institution will help identify worrisome trends in boarding that may impact the operational performance of your unit.
- ED/hospital revisit rate (overall and by protocol at 3, 7, and 14 days)
  - Benchmark: The typical ED revisit window is 3 days, whereas the inpatient time frame is 30 days. For observation unit recidivism, an intermediate time frame such as 7 or 14 days has been recommended. Published rates are around 10 % at 14 days but data are limited and monitoring the trend may be more valuable than aiming for an absolute target [7].
- Observation use by attending (overall and by protocol)
  - Benchmark: Individual attendings should be using the observation unit as a disposition option in the 5–10 % range that mirrors overall use [3]. Outliers (i.e., those much below 5 % or above 10 %) should receive feedback that they may be under- or overutilizing observation, respectively.

Satisfaction

- Patient satisfaction rates (e.g., Press Ganey)
  - Benchmark: Directors should focus on reaching the threshold of the 90th percentile or above for the survey average. Low scores on specific questions may prompt a focus in that particular area (i.e., cleanliness of rooms, nursing interactions, etc.).
- Patient comments/complaints
  - Benchmark: Medical and nursing leadership should review, investigate, and follow up on all patient comments and complaints in a timely manner.

Safety

- Intensive care unit (ICU) upgrades directly from observation
  - Benchmark: There is no consensus value for ICU upgrades; these should be a rare event prompting a timely in-depth case review.
- Deaths in the observation unit
  - Benchmark: There is no consensus value for observation unit deaths; these are sentinel events that prompt a timely in-depth case review, with involvement from senior department leadership and risk management.
- Code Blue in observation
  - Benchmark: There is no consensus value for code activations; these should be a rare event prompting a timely in-depth case review.
- Safety reports/care concerns
  - Benchmark: There is no consensus value for safety reports/care concerns; all staff should be encouraged to file safety reports and forward cases for review to unit leadership.

Quality Metrics Specific to Atrial Fibrillation

Documentation

- Time of onset of symptoms. Important for determining if a rhythm control strategy is an option in the ED or observation unit

- Documentation of thromboembolic risk (e.g., $CHADS_2VASc$ score) [8]. Important to use an evidence-based tool to determine risk of thromboembolic complications

Cardioversion

- Cardioversion success rate

Quality metrics associated with the sedation if electrical cardioversion is used (each sedation should be reviewed in depth for the following):

If electrical cardioversion used:

- Pre-procedure checklist (i.e., medical history, meds/allergies, cardiac, respiratory, and airway exam documented)
- Operator credentialed for level of sedation and medications used
- Use of end-tidal CO_2
- Medications used, including doses
- Documentation of consent for procedure signed by patient
- Documentation of "time-out" prior to procedure start
- Number of attempts (with energy level used)
  - Pearl: Traditional teaching has been to start at low energy (e.g., 75 J) and repeat at escalating energy if unsuccessful. However, the likelihood of success with fewer attempts is higher when starting with maximum energy (e.g., 250 J).
- Whether hypoxia was encountered during sedation
  - Typically defined as desaturations below 90 %
- Use of rescue breathing
- Other complications

If chemical cardioversion used:

- Documentation of consent for procedure signed by patient
- Correct dosage and administration of drug
- Confirmation of proper monitoring and duration according to your institution's drug administration guidelines
- Documentation and review of complications, especially malignant arrhythmias and hypotension

Discharge Planning

- Documentation of adequate rate control or conversion to normal sinus rhythm prior to discharge

- Outpatient clinic follow-up rate. Best practice is to confirm appointment date/time while patient is still present; follow up with an appropriate provider (i.e., cardiologist, primary care provider) within a 3–7-day window following the observation visit.
- Rate of discharge with appropriate anticoagulation (if applicable). Once thromboembolic risk is calculated, appropriate anticoagulation should be initiated prior to discharge, including follow-up plans (i.e., anticoagulation clinic follow-up).

Conclusion

The ED observation unit is an important resource that enables safe and efficient evaluation and treatment of a wide range of patients presenting to the ED with atrial fibrillation and atrial flutter. Metrics play a central role in ensuring a high-performing practice. Observation unit leadership should maintain reports or dashboards that enable them to closely monitor metrics across all protocols and those specific to atrial fibrillation in order to track new interventions and monitor for deviations from practice recommendations.

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Chapter 21
Atrial Fibrillation Research in the Observation Unit and the Emergency Department

Jesse M. Pines, Ali Pourmand, Ted Fan, and Ryan Tansek

Background

Atrial fibrillation (Afib) is a common cardiac dysrhythmia seen in the emergency department (ED). Afib is a chronic disease that can cause acute problems that require ED care, particularly when the Afib is symptomatic, newly diagnosed, or the rate is out of control, or medications used to treat Afib such as warfarin cause bleeding. Afib can also cause secondary problems that require emergency care such as stroke and other embolic phenomena. Finally, because of the high prevalence of Afib in the community, Afib patients can often present to the ED with noncardiac complaints, and the presence of Afib as a chronic diagnosis must be considered [1–6]. When longer-term care is required than the ED can provide, patients are sometimes admitted to an observation unit (OU) which is a short-stay unit sometimes physically located in the ED. Alternatively, patients may require hospital admission for further care. The focus in this chapter is to describe the recent literature on Afib on ED and OU and describe the common issues that researchers face when studying the care of Afib patients.

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Troponin and Afib

The use of cardiac troponin levels as a way to risk stratify ED patients with Afib is a major area of study in Afib research [7]. Cardiac troponin is a laboratory blood test which, when elevated, may indicate damage to the heart muscle. Because increased troponin is interpreted in the ED as a sign of acute cardiac injury, patients are often admitted to rule out life-threatening complications such as myocardial ischemia and acute myocardial infarction (AMI). In a multicenter, retrospective cohort study of 452 patients presenting with Afib and rapid ventricular response (RVR), 197 had an elevated troponin level. After controlling for the presence of comorbid conditions, the incidence of AMI was higher at 1 year in patients with mild troponin elevations, suggesting that elevated troponin places patients at higher risk of poorer outcomes [8].

In a German study investigating the association of Afib and elevated troponin levels, patients who had Afib with rapid ventricular response (RVR) and chest pain were more likely to have a positive troponin level compared with patients with another rate or rhythm disturbance such as sinus tachycardia. However, an elevated troponin level was a poor predictor (positive predictive value = 26 %) for coronary stenosis. The study was limited because all patients did not receive definitive cardiac testing to detect coronary disease [7].

Another retrospective, single-center study described 948 patients with ECG-confirmed Afib lasting less than 48 h and an elevated troponin in Italy. Patients were enrolled from the ED, and then followed up via phone contact and chart reviews. The authors found that patients that showed a positive troponin had a higher risk of short-term adverse events specifically in patients with ischemic heart disease and advanced age [9].

A prospective study was conducted analyzing all consecutive patients admitted with Afib over a 2-year period in the Netherlands. Minor troponin elevation was defined as a troponin I level between 0.15 and 0.65 ng/m. A total of 407 patients were studied, with a median duration of follow-up of 688 days. Minor elevations in troponin I at time of admission to the hospital were associated with higher rates of mortality and later cardiac events [10].

In a retrospective chart review of patients with ECG-confirmed Afib presenting to the two academic EDs and one community ED, 86 % of the patients had troponins ordered, approximately 14 % of patients had elevated troponin, and 5 % of patients were diagnosed with an acute coronary syndrome [11]. In a retrospective study which enrolled all patients admitted to an acute stroke unit, an initial cTnI assay was used to help predict new-onset Afib early during an ischemic stroke in patients who initially had a sinus rhythm [12].

In a multicenter, retrospective study, 662 patients with Afib were divided into three groups based on troponin levels. These included group 1 with a mildly elevated level of troponin, group 2 with a normal troponin, and group 3 with an unmeasured troponin. Levels of troponin I were measured in 76 % of patients. They were elevated in 33 % of group 1, normal in 43 % group 2, and not measured in 24 %,

group 3. Further cardiac testing was done more often in group 1. This study showed that a mildly elevated troponin I was associated with higher rates of coronary artery disease and a greater 1-year incidence of AMI [13].

Bottom Line Cardiac troponin is a sensitive test that helps predict morbidity and mortality in Afib patients seen in the ED. With a considerable literature that supports the association between even mild leakage of troponin in Afib patients and mortality and serious cardiovascular events, ED patients with Afib and a positive troponin are often admitted to the OU or the hospital for further risk stratification and/or treatment.

Cardioversion in Afib

Rhythm control – specifically performing interventions to cardiovert patients from Afib to normal sinus rhythm – is an important area of research in the ED management of Afib. To achieve this, cardioversion can be performed in two ways: electrical cardioversion with procedural sedation or by pharmacologic cardioversion with medication such as ibutilide. However, concerns about complications such as stroke limit the routine performance of cardioversion in the ED in Afib patients. Therefore, patients must be selected carefully, with contraindications for cardioversion carefully considered. Some of these complications are Afib lasting more than 48 h, high risk for stroke, and risk for ventricular tachycardia/fibrillation [14]. While some literature suggests that termination of the Afib rhythm may not be expected to change the clinical outcome and also may not be safe, other studies suggest feasibility and safety of this approach [15–17].

One study conducted in an ED and OU in Italy tested an aggressive strategy (specifically as intensive pharmacological treatment followed by electrical treatment as required) for rhythm control of Afib with RVR aiming in to improve rhythm control and reduce hospital admission rates. They improved rhythm conversion overall up to 62 % from 55 % and decreased admissions up to 60 %. The admission rate among standard treatment group was 50 % versus 29 % in aggressive group [18]. By comparison, in Canada, Afib patients are more frequently treated with cardioversion in the ED and then discharged. A study from Canada was designed to track the growing proportion of patients treated using this approach. Patients were followed 90 days after discharge to monitor for factors associated with death. Lack of follow-up within 90 days had the strongest association with mortality, and a filled warfarin prescription was associated with a lower risk of death [19]. Like the other Canadian population health studies, the Canadian health system which has easier access to outpatient care may not generalize to other parts of the world. However, while this was a well-designed study focused on 90-day all-cause mortality after the index visit, as with any observational study, causality cannot be directly inferred.

Another Canadian study evaluated ventricular rate control prior to cardioversion to assess success rate of cardioversion. A total of 634 patients with Afib or atrial

flutter underwent cardioversion. This study demonstrated that successful electrical cardioversion of Afib or atrial flutter was less likely when patients were pretreated with either rate or rhythm control medications [20]. The data were collected by trained research nurses across eight different academic centers in Canada via chart review; however, each center did not have a set pretreatment protocol.

Bottom Line Cardioversion in ED or OU is a point of interest in Afib patients, and there are several studies that advocate this procedure where patients who are successfully cardioverted can be discharged home assuming they have close follow-up with a responsible physician. There are many different approaches and protocols that can be used including electrical or pharmacological cardioversion.

Rate Control in Afib

There is also a considerable amount of ED research on the use of pharmacologic agents to help control heart rate in ED Afib patients. The goal of rate control in the ED is to improve the patient's overall hemodynamic status, resulting in better ventricular filling, less myocardial oxygen demand, and improved cardiac output. Rate control can also be helpful in reducing symptoms such as chest pain or palpitations. There are several options to control rate in the acute setting of Afib. Research has compared medications including calcium channel blockers, beta-blockers, digoxin, and several other agents.

One study examined Canadian ED patients with Afib who were otherwise healthy, who were administered rate control with beta-blockers or calcium channel blockers. Both were found to be equally safe in terms of the rates of ED adverse events, strokes, and deaths at 30 days [21]. There are several aspects of this study that are important to conducting research studies in general in the ED or OU. The sites in this study shared electronic charts, and the region had a shared pharmacy database that tracked all prescriptions – both very helpful tools for the researcher, especially when detecting post-ED outcomes is needed. In addition, as described earlier not all health systems are similar with respect to primary care access such as in Canada. Therefore, it may be difficult to produce similar research quality or results elsewhere. The challenges facing this study and other retrospective chart reviews include adequate/accurate of charges and variable treatment decisions. Since charts were flagged by diagnosis codes, improper coding may exclude otherwise eligible patients or include them in wrong categories for analysis. The retrospective nature of the chart review also does not allow for standardization of treatments and allows treatment bias to influence results.

Another study was focused on comparing diltiazem versus metoprolol in the management of Afib with RVR. They found diltiazem was more effective in achieving rate control in ED than metoprolol [22]. However, the maximum dose of metoprolol (10 mg) used in this study was based on a separate study and advanced cardiac life support (ACLS) guidelines [23]. Some studies suggest that higher doses

could have been used but the institutional review board (IRB) and the study's participating physicians deemed those doses to be "excessively high." These doses could have been associated with different results.

Afib patients with acute underlying medical illness could have different outcomes. In a retrospective, descriptive cohort study, reviewing ECG databases from two urban EDs produced a heterogeneous cohort of complex patients that only had atrial fibrillation or flutter as a unifying feature. The primary outcome was the safety of rate or rhythm control attempts, measured as having a predefined adverse event in the ED. The rate control was attempted by IV metoprolol, while some patients received diltiazem, verapamil, and digoxin. Overall, the success rates of the calcium channel blockers appeared to be higher than beta-blockers, and adverse events were similar across all groups. Patients who received digoxin had neither successful rate control nor adverse events. It is difficult to conclusively demonstrate that the high adverse event and low success rate directly resulted from attempted rate versus rhythm control or from medication type, dosage, or timing. However, after adjusting for the attending physician and ED and patient-level factors, the results were found to be similar. When Afib patients were grouped as being managed with rate or rhythm control attempts, the stroke and mortality rates were similar for the two groups. This may imply that ED-based treatments and adverse events do not have a strong influence on 30-day outcomes. However, many unmeasured factors can affect physician decisions and patient outcomes [24].

Bottom Line Calcium channel blockers such as diltiazem tend to be more effective in controlling heart rate in ED Afib patients than other medication such as beta-blockers, as it has been associated with fewer side effects and higher success rates. However, studies that have directly studied this question are limited and there are no randomized trials.

The Risk of Stroke and Afib

The risk of stroke and Afib is an important area of research that has implications in the ED; however, ED physicians do not commonly prescribe medication to help control stroke risk. That is almost always reserved for physicians who follow patients longitudinally such as cardiologists and primary care physicians. However, this is an important area for ED providers to be aware of because effective medication and risk scores to guide use are available, and sometimes Afib patients who should be on a particular stroke risk regimen should receive a referral back to their primary care physician if stroke risk does not appear to be addressed.

Cerebral thromboembolism of cardiac origin accounts for approximately 20 % of ischemic strokes [25]. Structural vascular disease, abnormal blood stasis, and abnormal hemostasis (also known as Virchow's triad) are three mechanisms that explained for risk of thromboembolism in patients with Afib [26]. Based on the stroke guideline, Afib is a cause responsible for both first and recurrent stroke. Every year more

than 75,000 cases of stroke are related to Afib [27]. The CHA2DS2-VASc score is a validated tool to risk stratify patients with Afib and includes age ranges from 65 to 74 and age 75 or older, history of congestive heart failure, hypertension, smoking, diabetes, vascular disease, stroke, transient ischemic attack (TIA), thromboembolism, and female sex [28]. Patients with age 75 and above and also with stroke, TIA, or thromboembolism have a higher score. Some patients may require an aspirin to address stroke risk; however, for patients at higher risk, a Vitamin K antagonist such as warfarin is the mainstay in treatment of Afib patients to lower stroke risk and can decrease risk of stroke by up to 60 % [29]. There are several limitations to this group of medication including, but not limited to necessity of monitoring INR on a regular basis, medication interaction and risk of bleeding. New generation of anticoagulation therapies such as dabigatran, rivaroxaban, apixaban, and edoxaban have been shown to be similar with respect to reducing stroke risk [29].

Bottom Line While there are strong associations between Afib and stroke, there are also several new types of anticoagulation medications that a patient with Afib can take to reduce stroke risk.

Disposition and Afib

One of the important issues in the ED is disposition decision, specifically whether to discharge patients or admit them to the OU or the inpatient area. One study assessed outcomes of healthy patients with no comorbidities or past medical history up to a year after an ED visit for Afib. These healthy patients were more likely to be discharged than patients with a more complicated past medical history, and this was supported by a practice with less than 1 % stroke or death rate at 30 days for this population [30]. This was a retrospective chart review, where emergency physicians managed patients and used no standardized protocol. There was also no formal comparison of groups, so no statistical analysis was done. Patients who received rate control were older, had higher CHADS2 sores, and had been in Afib longer; it would be very hard to adjust for these baseline differences. One difficulty with the research of Afib is defining the time patients were in it. This study attempted to do so via "rigorous predefined chart review methodology with reliability assessment and specialist adjudication where necessary." Other issues included following up patients and missing patients who move and were addressed by some census data regarding low migration rates.

Clinical decision rules to aid disposition are another topic of interest in the world of Afib research. The atrial fibrillation and flutter outcomes and risk determination (AFFORD) is the newest decision rule to identify low risk of adverse event among ED patients with Afib. The study that was used to derive AFFORD was a prospective cohort study with convenience sampling of symptomatic Afib patient in ED. It is designed based on patient's age, triage vitals, medical history, and ED data (blood results, x-ray, and cardiovascular monitoring) [31]. AFFORD study has strong

internal (but not external) validation to identify patients who are at low risk for 30-day adverse events and candidates for safe discharge from ED. This study was done at a referral center and their population may not be generalizable to other settings.

Limitations to Afib Research in the ED and OU

There are several limitations limitation to Afib research in the ED and OU. First, many of the studies involve small numbers of patients. There were multiple differences between the various study protocols which do not allow pooling of data across studies. Variable use of antiarrhythmic medications and variable protocols can confound findings. The definition of a successful cardioversion varied as well and it is unknown if these patients remained in sinus rhythm or converted back into Afib after discharge. Many studies did not report their outcomes in a consistent fashion. In some studies, the dosage of electricity used for cardioversion is lower than current standard doses recommended in ACLS guideline. Authors were unable to assess for publication bias again due to small amount of original studies [32]. In addition, many Afib-related studies are done in Canada, where there is universal healthcare coverage, with a closed system of primary care coverage and good access to physicians after ED discharge. This broad access to outpatient care is not generalizable in the USA and the rest of the world. The small sample size, proportion of chart reviews, and individual preference instead of universal protocol are some other challenges that researchers should take into account for future studies.

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Chapter 22
Atrial Fibrillation Protocols

Anna Ek

Introduction

Atrial fibrillation incidence is growing exponentially, far beyond earlier estimations. In the past, physicians and nurses were under the impression that this was a fairly benign occurrence, much to the detriment of patient well-being and survival. We are generally slow to adopt evidence and guideline-directed medical therapy, but in light of the phenomenon, we must employ evidence-based practices sooner and save these patients from lifestyle deterioration, heart failure, the eventual stroke, and potential death.

Standardized order sets and flowcharts created following guideline-directed medical therapy will ensure patients receive the recommended care. Anticoagulation, rate and rhythm control, and cardioversion protocols should be addressed. Absolute confirmation of the onset of atrial fibrillation guides cardioversion decision, with or without TEE and anticoagulation. If unknown, assume it is longer than 48 h.

The Society has received permission to share these examples from certified and accredited atrial fibrillation centers (Figs. 22.1, 22.2, 22.3, 22.4, 22.5, 22.6, 22.7, 22.8, 22.9, 22.10, 22.11, 22.12, 22.13, 22.14, 22.15, and 22.16). It is the responsibility of all to share knowledge and proven practices with others to provide efficient, cost-effective, and, above all, safe care.

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Contemporary Cardiology, DOI 10.1007/978-3-319-31386-3_22

ARRHYTHMIAS

EMT-B

A. Open and manage the airway and provide 100% oxygen by NRB mask. Apply pulse oximeter

B. Make patient comfortable and provide reassurance

C. Evaluate patient's general appearance, relevant history of condition, and determine the following:

Onset	Allergies
Provokes	Medication
Quality	Past medical history (especially recent
Radiates	surgery, any abnormal ingestion,
Severity	previous trauma, related conditions)
Time	Last meal
Interventions	Events leading to present illness

D. If the patient is experiencing an unusual and/or irregular heart rate or pulse, the cardiac monitor may be applied, if available, and a strip run for evaluation by qualified personnel. This should only be done during transport and you must advise the patient that you do not have the ability to interpret the tracing

E. Establish communication with Medical Control and advise of patient condition. Transport **IMMEDIATELY**

EMT-I

A. Start IV normal saline TKO (provide 500 cc fluid bolus if signs of hypoperfusion are present and no signs of pulmonary edema are present)

PARAMEDIC

A. Assume charge of situation and confer with EMTs about condition of patient and situation.

B. Apply cardiac monitor and determine identity of arrhythmia

C. Start IV normal saline TKO (provide 500 cc fluid bolus if signs of hypoperfusion are present and no signs of pulmonary edema are present)

D. Treat arrhythmia as follows:
   1. Bradycardia, second and third degree heart blocks
      a. good perfusion – transport
      b. poor perfusion:

Fig. 22.1

1. atropine 0.5-1.0 mg IV push, repeat 1.0 mg IV push every 5 minutes up to 3 mg total dose or until heart rate is greater than 60 and systolic BP is greater than 90
2. external pacemaker set at 80 beats per minute with output at 20 milliamps, increasing by 20 milliamps until mechanical capture is obtained
3. contact Medical Control
4. if still poor perfusion and approved by Medical Control, start dopamine drip (400 mg dopamine in 500 cc D_5W or NS to yield a solution of 800 ug/cc) and titrate the infusion until heart rate is above 60 and systolic BP is above 90

2. Atrial flutter/fibrillation with rapid ventricular rate
   a. good perfusion – transport
   b. poor perfusion with systolic BP less than 90 and ventricular rate greater than 150:
      1. attempt to determine the time of onset of the symptoms
      2. if less than 72 hours:
         a. consider sedation (*Versed* 2-4 mg IV)
         b. cardiovert 100 joules (100 joules biphasic)
         c. cardiovert 200 joules (120 joules biphasic)
         d. cardiovert 300 joules (150 joules biphasic)
         e. cardiovert 360 joules (170 joules biphasic)
         f. transport **IMMEDIATELY**
         g. contact Medical Control for assistance
         h. consider *Verapamil* 10 mg IV
      3. if greater than 72 hours or unknown:
         a. transport **IMMEDIATELY**
         b. contact Medical Control for assistance
3. Supraventricular tachycardia
   a. patients who are alert and oriented with normal blood pressure and ventricular rate less than 150
      1. transport
   b. patients with ventricular heart rate greater than 150 beats per minute along with blood pressure greater than 70, chest pain, and/or shortness of breath
      1. administer adenosine 6 mg rapid IV bolus followed IMMEDIATELY with a 20 cc saline bolus
      2. if no conversion, repeat adenosine with 12 mg rapid IV bolus followed IMMEDIATELY with a 20 cc saline bolus
      3. if no conversion, repeat adenosine with 12 mg rapid IV bolus followed IMMEDIATELY with a 20 cc saline bolus

Fig. 22.1 (continued)

4. if no response to adenosine, attempt synchronized cardioversion:
    a. consider sedation (*Versed* 2-4 mg IV)
    b. cardiovert 50 joules (use same setting for biphasic)
    c. cardiovert 100 joules (100 joules biphasic)
    d. cardiovert 200 joules (120 joules biphasic)
    e. cardiovert 300 joules (150 joules biphasic)
    f. cardiovert 360 joules (170 joules biphasic)
5. transport **IMMEDIATELY**
6. consider *Verapamil* 10 mg IV (contact Medical Control)

c. patients with blood pressure less than 70 or decreased level of consciousness
    1. synchronized cardioversion
        a. consider sedation (*Versed* 2-4 mg IV)
        b. cardiovert 50 joules (use same setting for biphasic)
        c. cardiovert 100 joules (70 joules biphasic)
        d. cardiovert 200 joules (120 joules biphasic)
        e. cardiovert 300 joules (150 joules biphasic)
        f. cardiovert 360 joules (170 joules biphasic)
    2. transport **IMMEDIATELY**

4. Frequent PVCs with symptoms (chest pain, shortness of breath, palpitations, hypotension, dizziness)
    a. treat underlying causes (i.e. hypoxia, hypoperfusion, cardiac chest pain)
    b. contact Medical Control
    c. lidocaine 75-100 mg IV (if approved by Medical Control)
    d. may repeat lidocaine dose once in 8-10 minutes (if approved by Medical Control)
    e. if lidocaine is effective, initiate lidocaine drip 2-4 mg/min (if approved by Medical Control)
5. Ventricular tachycardia
    a. patients with minimal symptoms and normal blood pressure
        1. amiodarone 150 mg IV over 10 minutes
        2. if no effect after 10 minutes, lidocaine 75-100 mg IV bolus (may repeat every 5 minutes to a maximum total dose of 300 mg)
        3. if lidocaine bolus is effective, start lidocaine drip at 2-4 mg/min
        4. if still no effect, attempt synchronized cardioversion:
            a. consider sedation (*Versed* 2-4 mg IV)
            b. cardiovert 100 joules (70 joules biphasic)
            c. cardiovert 200 joules (120 joules biphasic)
            d. cardiovert 300 joules (150 joules biphasic)
            e. cardiovert 360 joules (170 joules biphasic)

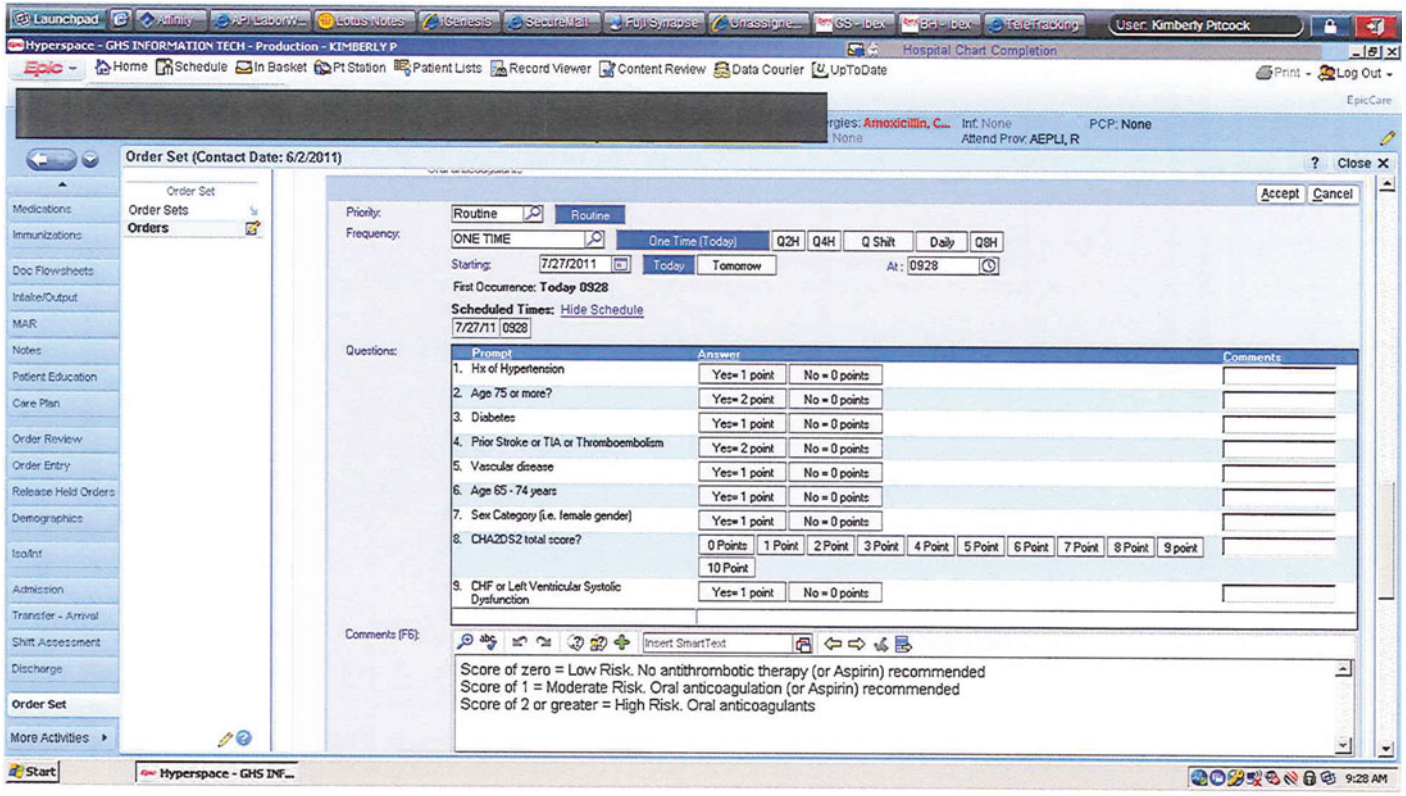
Fig. 22.1 (continued)

5. transport **IMMEDIATELY**

b. patients with significant symptoms, hypotension, and/or decreased level of consciousness
   1. synchronized cardioversion:
      a. consider sedation (*Versed* 2-4 mg IV)
      b. cardiovert 100 joules (70 joules biphasic)
      c. cardiovert 200 joules (120 joules biphasic)
      d. cardiovert 300 joules (150 joules biphasic)
      e. cardiovert 360 joules (170 joules biphasic)
   2. amiodarone 150 mg IV over 10 minutes
   3. if no effect after 10 minutes, lidocaine 75-100 mg IV bolus (may repeat every 5 minutes to a maximum total dose of 300 mg)
   4. if lidocaine bolus is effective, start lidocaine drip at 2-4 mg/min
   5. repeat synchronized cardioversion, if necessary
      a. cardiovert 100 joules (70 joules biphasic)
      b. cardiovert 200 joules (120 joules biphasic)
      c. cardiovert 300 joules (150 joules biphasic)
      d. cardiovert 360 joules (170 joules biphasic)
   6. transport **IMMEDIATELY**

c. patients without a pulse
   1. treat as ventricular fibrillation (see Cardiac Arrest protocol)

Fig. 22.1 (continued)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Lotus Notes
iGenesis
SecureMail
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GS - Ibex
BH - Ibex
TeleTracking
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Hyperspace - GHS INFORMATION TECH - Production - KIMBERLY P
Hospital Chart Completion
Epic
Home
Schedule
In Basket
Pt Station
Patient Lists
Record Viewer
Content Review
Data Courier
UpToDate
Print
Log Out
EpicCare
Amoxicillin, C...
Inf: None
PCP: None
Attend Prov: AEPLI, R
Order Set (Contact Date: 6/2/2011)
Close
Order Set
Order Sets
Orders
Medications
Immunizations
Doc Flowsheets
Intake/Output
MAR
Notes
Patient Education
Care Plan
Order Review
Order Entry
Release Held Orders
Demographics
Isolinf
Admission
Transfer - Arrival
Shift Assessment
Discharge
Order Set
More Activities
Accept
Cancel
Priority:
Routine
Frequency:
ONE TIME
One Time (Today)
Q2H
Q4H
Q Shift
Daily
Q8H
Starting:
7/27/2011
Today
Tomorrow
At:
0928
First Occurrence: Today 0928
Scheduled Times: Hide Schedule
7/27/11 0928
Questions:
Prompt
Answer
Comments
1. Hx of Hypertension
Yes= 1 point
No = 0 points
2. Age 75 or more?
Yes= 2 point
No = 0 points
3. Diabetes
Yes= 1 point
No = 0 points
4. Prior Stroke or TIA or Thromboembolism
Yes= 2 point
No = 0 points
5. Vascular disease
Yes= 1 point
No = 0 points
6. Age 65 - 74 years
Yes= 1 point
No = 0 points
7. Sex Category (i.e. female gender)
Yes= 1 point
No = 0 points
8. CHA2DS2 total score?
0 Points
1 Point
2 Point
3 Point
4 Point
5 Point
6 Point
7 Point
8 Point
9 point
10 Point
9. CHF or Left Ventricular Systolic Dysfunction
Yes= 1 point
No = 0 points
Comments (F6):
Insert SmartText
Score of zero = Low Risk. No antithrombotic therapy (or Aspirin) recommended
Score of 1 = Moderate Risk. Oral anticoagulation (or Aspirin) recommended
Score of 2 or greater = High Risk. Oral anticoagulants
Start
Hyperspace - GHS INF...
9:28 AM

Fig. 22.2

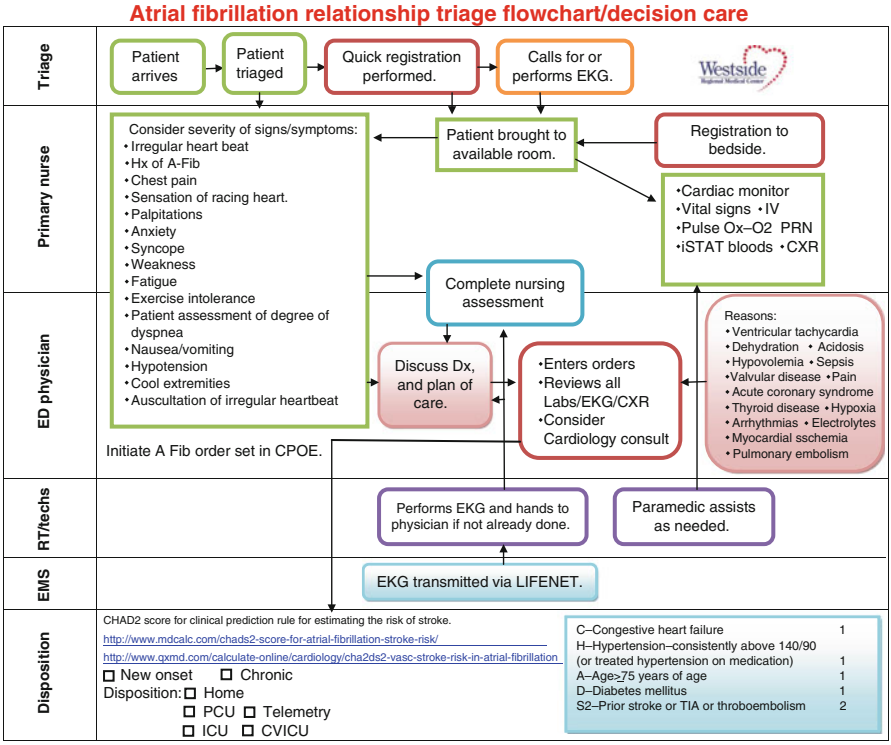
Atrial fibrillation relationship triage flowchart/decision care
Triage
Primary nurse
ED physician
RT/techs
EMS
Disposition
Patient arrives
Patient triaged
Quick registration performed.
Calls for or performs EKG.
Westside
Consider severity of signs/symptoms:
• Irregular heart beat
• Hx of A-Fib
• Chest pain
• Sensation of racing heart.
• Palpitations
• Anxiety
• Syncope
• Weakness
• Fatigue
• Exercise intolerance
• Patient assessment of degree of dyspnea
• Nausea/vomiting
• Hypotension
• Cool extremities
• Auscultation of irregular heartbeat
Patient brought to available room.
Registration to bedside.
•Cardiac monitor
•Vital signs •IV
•Pulse Ox–O2 PRN
•iSTAT bloods •CXR
Complete nursing assessment
Discuss Dx, and plan of care.
•Enters orders
•Reviews all Labs/EKG/CXR
•Consider Cardiology consult
Reasons:
• Ventricular tachycardia
• Dehydration • Acidosis
• Hypovolemia • Sepsis
• Valvular disease • Pain
• Acute coronary syndrome
• Thyroid disease • Hypoxia
• Arrhythmias • Electrolytes
• Myocardial sschemia
• Pulmonary embolism
Initiate A Fib order set in CPOE.
Performs EKG and hands to physician if not already done.
Paramedic assists as needed.
EKG transmitted via LIFENET.
CHAD2 score for clinical prediction rule for estimating the risk of stroke.
http://www.mdcalc.com/chads2-score-for-atrial-fibrillation-stroke-risk/
http://www.qxmd.com/calculate-online/cardiology/cha2ds2-vasc-stroke-risk-in-atrial-fibrillation
☐ New onset ☐ Chronic
Disposition: ☐ Home
☐ PCU ☐ Telemetry
☐ ICU ☐ CVICU
C–Congestive heart failure 1
H–Hypertension–consistently above 140/90 (or treated hypertension on medication) 1
A–Age≥75 years of age 1
D–Diabetes mellitus 1
S2–Prior stroke or TIA or throboembolism 2

Fig. 22.3

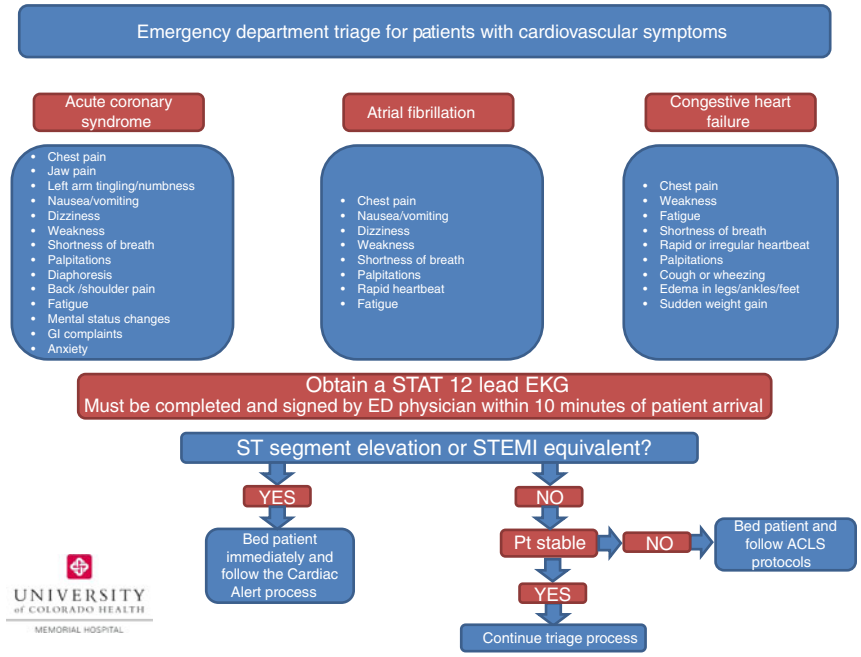
Emergency department triage for patients with cardiovascular symptoms
Acute coronary syndrome
Chest pain
Jaw pain
Left arm tingling/numbness
Nausea/vomiting
Dizziness
Weakness
Shortness of breath
Palpitations
Diaphoresis
Back /shoulder pain
Fatigue
Mental status changes
GI complaints
Anxiety
Atrial fibrillation
Chest pain
Nausea/vomiting
Dizziness
Weakness
Shortness of breath
Palpitations
Rapid heartbeat
Fatigue
Congestive heart failure
Chest pain
Weakness
Fatigue
Shortness of breath
Rapid or irregular heartbeat
Palpitations
Cough or wheezing
Edema in legs/ankles/feet
Sudden weight gain
Obtain a STAT 12 lead EKG
Must be completed and signed by ED physician within 10 minutes of patient arrival
ST segment elevation or STEMI equivalent?
YES
Bed patient immediately and follow the Cardiac Alert process
NO
Pt stable
NO
Bed patient and follow ACLS protocols
YES
Continue triage process
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Fig. 22.4

Atrial Fibrillation, New Onset, Quickpick
Page 1 of 4

The tools below can be used to support decisions on anticoagulant and antiplatelet therapy. Use the CHA_2DS_2 – VASc to estimate risk for stroke and HAS-BLED to estimate risk for bleeding.

CHA_2DS_2 – VASc

Risk Factor		Points
C	CHF/LV dysfunction	1
H	HTN	1
A_2	Age 75 years or more	2
D	Diabetes	1
S_2	Stroke/TIA/thromboembolism	2
V	Vascular disease: CAD, MI, PAD, aortic plaque	1
A	Age 65-74 years	1
Sc	Sex category - female	1

Score	Recommendations
0	Aspirin 81 - 325 mg po daily
1	Aspirin 81 - 325 mg po daily, warfarinwith target INR 2.5 (2-3), or new oral anticoagulant agent
2 or more	Warfarin, with target INR 2.5 (2-3) or new oral anticoagulant agent

HAS-BLED

Risk Factor		Points
H	Hypertension (systemic BP over 160 mmHG)	1
A	Abnormal renal function	1
	Abnormal liver function	1
S	Previous Stroke	1
B	Prior major bleeding or predisposition (reduced platelet count or function, anemia, genetic factors, excessive fall risk)	1
L	Labile INR (less than 60% of time in therapeutic range)	1
E	Age over 65 (elderly)	1
D	Drugs predisposing to bleeding (antiplatelet agents, NSAIDs)	1
	Alcohol Use (over 8 drinks per week)	1

The HAS-BLED score was derived from a real-world cohort of 3978 with AF and assesses the 1-year risk for major bleeding (intracranial, hospitalization, hemoglobin decrease > 2 g/L, and/or transfusion) associated with oral anticoagulation. **Patients with a high risk of bleeding (HAS-BLED score >/= 3) should undergo regular clinical review following the initiation of oral anticoagulation.**

Pisters R, Lane DA, Nieuwlaat R, de Vos CB, Crijns HJ, Lip GY. A novel user-friendly score (HAS-BLED) to assess one-year risk of major bleeding in atrial fibrillation patients: The Euro Heart Survey. Chest. 2010 Mar 18

Date:	Time:	Physician Signature	
VO/TO taken, read back & verified by: Time:		from: (physician)	Date:
Authorization is given for dispensing generic equivalent unless checked here ☐			

Fig. 22.5

Atrial Fibrillation, New Onset, Quickpick
Page 2 of 4

ORDERS

- ☐ Cardiac telemetry
- ☐ Electrophysiology consult

MEDICATIONS

RATE CONTROL

- ☐ Metoprolol 25 mg PO BID
- ☐ Diltiazem 30 mg PO QID
- ☐ Diltiazem 60 mg PO QID
- ☐ Diltiazem 20 mg IV once over 2 minutes
- ☐ Diltiazem 25 mg IV once over 2 minutes, additional bolus after 15 minutes if needed if HR remains over 105
- ☐ Diltiazem start 125 mg/125 mL. Start: 5 mg/hour. Increase by: 5 mg/hour every 15 minutes to achieve: HR less than 105 and SBP greater than 90. Maximum 15 mg/hr
- ☐ Esmolol. Initial loading dose of 500 mcg/kg/min for 1 minute, followed by 50 mcg/kg/min for 4 minutes. If adequate response, continue with 50 mcg/kg/minute. If adequate response not achieved within 10 minutes, repeat the 500 mcg/kg/min for 1 minute loading dose followed by a 100 mcg/kg/min for 4 minutes. If necessary, repeat the 500 mcg/kg/min x 1 minutes load and increase the maintenance infusion by 50 mcg/kg/min for 4 minutes until HR less than 105 or SBP less than 90 mmHg.
- ☐ Digoxin 0.25 mg IV once, give slowly over 5 min
- ☐ Digoxin 0.5 mg IV once, give slowly over 5 min
- ☐ Digoxin 0.125 mg po daily
- ☐ Digoxin 0.125 mg IV daily

Date:	Time:	Physician Signature	
VO/TO taken, read back & verified by: Time:		from: (physician)	Date:
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Fig. 22.5 (continued)

Atrial Fibrillation, New Onset, Quickpick
Page 3 of 4

Anticoagulation/Antiplatelet

- ☐ Aspirin 81 mg po daily
- ☐ Aspirin 325 mg po daily
- ☐ Warfarin 2.5 mg po now. Target 2 to 3. Indication is A fib.
- ☐ Warfarin 5 mg po now. Target 2 to 3. Indication is A fib.
- ☐ Enoxaparin 1mg/kg SQ every 12 hours
- ☐ Link to heparin powerplan
- ☐ Apixaban (Eliquis) 2.5 mg po BID Comment: for patients with nonvalvular atrial fibrillation. This dose is for patients with any two of the following: 80 years or older, body weight 60 kg or less, or serum creatinine 1.5 mg/dL or higher
- ☐ Apixaban (Eliquis) 5 mg po BID Comment: for patients with nonvalvular atrial fibrillation.
- ☐ Dabigatran (Pradaxa) 75 mg po BID Comment: for patients with nonvalvular atrial fibrillation. 75 mg bid should be considered for patients with estimated creatinine clearance 30-50 mL/min or treated with interacting medications including dronedarone.
- ☐ Dabigatran (Pradaxa) 150 mg BID Comment: for patients with nonvalvular atrial fibrillation and estimated creatinine clearance over 30 mL/min
- ☐ Rivaroxaban (Xarelto) 20 mg po QPM Comment: for patients with nonvalvular atrial fibrillation and estimated creatinine clearance over 50 mL/min
- ☐ Rivaroxaban (Xarelto) 15 mg po QPM Comment: for patients with nonvalvular atrial fibrillation and estimated creatinine clearance15-50 mL/min

Date:	Time:	Physician Signature	
VO/TO taken, read back & verified by: Time:		from: (physician)	Date;
Authorization is given for dispensing generic equivalent unless checked here ☐			

Fig. 22.5 (continued)

Atrial Fibrillation, New Onset, Quickpick
Page 4 of 4

RHYTHM CONTROL

Note to prescriber: consider the 'pill in the pocket' approach for appropriate patients with paroxysmal atrial fibrillation. Patients must have no structural heart disease. Agents used are flecainide 300 mg po X 1 or propofenone 600 mg PO X 1.

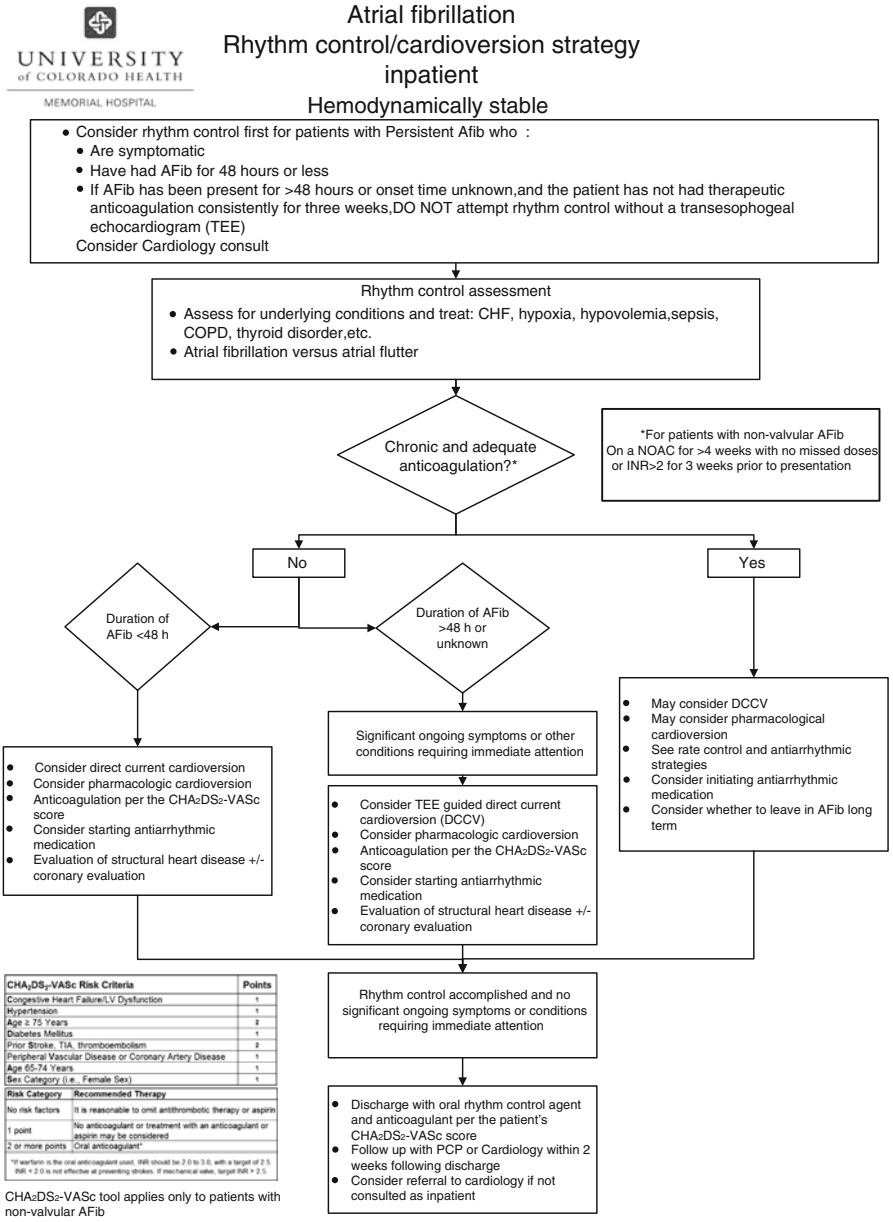
- ☐ Link to "amiodarone for Afib" powerplan
- ☐ Link to "dofetilide" (Tikosyn) powerplan
- ☐ Ibutilide (Corvert) 0.01 mg kg once over 10 min MR X 1, for patients less than 60 kg
- ☐ Ibutilide (Corvert) 1 mg IV once, MR X 1, for patient 60 kg or more
- ☐ Implement Ibutilide (Corvert) protocol
- ☐ Dronedarone (Multaq) 400 mg po BID
- ☐ Flecainide (Tambocor) 50 mg po every 12 hours
- ☐ Flecainide (Tambocor) 100 mg po every 12 hours
- ☐ Flecainide (Tambocor) 150 mg po every 12 hours
- ☐ Propafenone (Rythmol) 50 mg po every 8 hours
- ☐ Propafenone (Rythmol) 225 mg po every 8 hours
- ☐ Propafenone (Rythmol) 300 mg po every 8 hours
- ☐ Sotolol (Betapace) 40 mg PO bid. Comment: Manufacturer recommends initiation/reinitiation and dose increases be done in a hospital setting with continuous monitoring, staff familiar with the recognition and treatment of life-threatening arrhythmias.
- ☐ Sotolol (Betapace)60 mg PO bid. Comment: Manufacturer recommends initiation/reinitiation and dose increases be done in a hospital setting with continuous monitoring, staff familiar with the recognition and treatment of life-threatening arrhythmias.
- ☐ Sotolol (Betapace)80 mg PO bid. Comment: Manufacturer recommends initiation/reinitiation and dose increases be done in a hospital setting with continuous monitoring, staff familiar with the recognition and treatment of life-threatening arrhythmias.

LABS

- ☐ LFTs (recommended for patients being started on amiodarone, dronedarone or warfarin)
- ☐ INR STAT and daily
- ☐ CBC with diff
- ☐ Electrolytes

Date:	Time:	Physician Signature	
VO/TO taken, read back & verified by: Time:		from: (physician)	Date:
Authorization is given for dispensing generic equivalent unless checked here ☐			

Fig. 22.5 (continued)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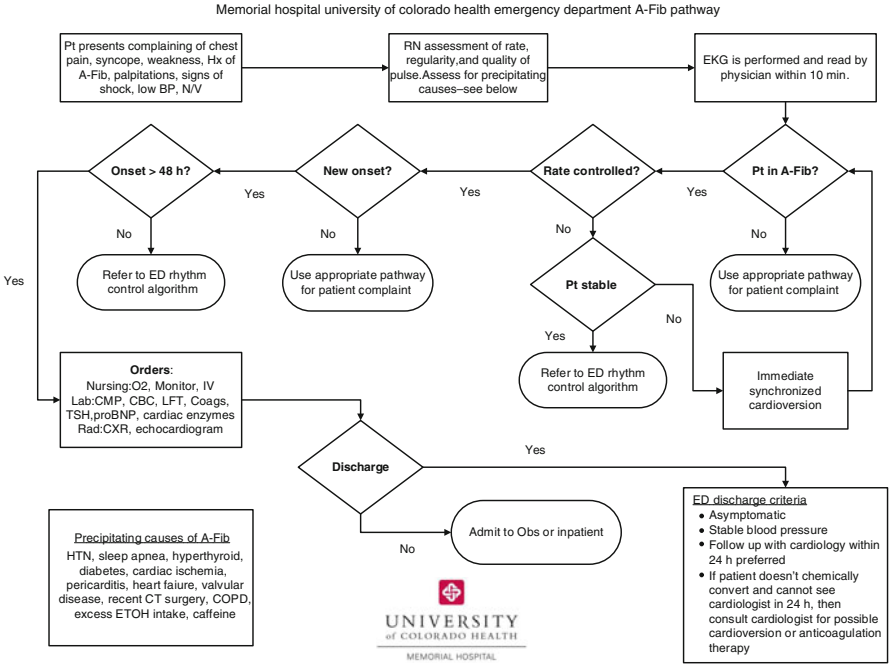
Atrial fibrillation
Rhythm control/cardioversion strategy
inpatient
Hemodynamically stable
• Consider rhythm control first for patients with Persistent Afib who :
• Are symptomatic
• Have had AFib for 48 hours or less
• If AFib has been present for >48 hours or onset time unknown,and the patient has not had therapeutic anticoagulation consistently for three weeks,DO NOT attempt rhythm control without a transesophogeal echocardiogram (TEE)
Consider Cardiology consult
Rhythm control assessment
• Assess for underlying conditions and treat: CHF, hypoxia, hypovolemia,sepsis, COPD, thyroid disorder,etc.
• Atrial fibrillation versus atrial flutter
Chronic and adequate anticoagulation?*
*For patients with non-valvular AFib
On a NOAC for >4 weeks with no missed doses or INR>2 for 3 weeks prior to presentation
No
Yes
Duration of AFib <48 h
Duration of AFib >48 h or unknown
• Consider direct current cardioversion
• Consider pharmacologic cardioversion
• Anticoagulation per the CHA2DS2-VASc score
• Consider starting antiarrhythmic medication
• Evaluation of structural heart disease +/- coronary evaluation
Significant ongoing symptoms or other conditions requiring immediate attention
• Consider TEE guided direct current cardioversion (DCCV)
• Consider pharmacologic cardioversion
• Anticoagulation per the CHA2DS2-VASc score
• Consider starting antiarrhythmic medication
• Evaluation of structural heart disease +/- coronary evaluation
• May consider DCCV
• May consider pharmacological cardioversion
• See rate control and antiarrhythmic strategies
• Consider initiating antiarrhythmic medication
• Consider whether to leave in AFib long term
CHA2DS2-VASc Risk Criteria | Points
Congestive Heart Failure/LV Dysfunction | 1
Hypertension | 1
Age ≥ 75 Years | 2
Diabetes Mellitus | 1
Prior Stroke, TIA, thromboembolism | 2
Peripheral Vascular Disease or Coronary Artery Disease | 1
Age 65-74 Years | 1
Sex Category (i.e., Female Sex) | 1
Risk Category | Recommended Therapy
No risk factors | It is reasonable to omit antithrombotic therapy or aspirin
1 point | No anticoagulant or treatment with an anticoagulant or aspirin may be considered
2 or more points | Oral anticoagulant*
*If warfarin is the oral anticoagulant used, INR should be 2.0 to 3.0, with a target of 2.5. INR < 2.0 is not effective at preventing strokes. If mechanical valve, target INR > 2.5.
CHA2DS2-VASc tool applies only to patients with non-valvular AFib
Rhythm control accomplished and no significant ongoing symptoms or conditions requiring immediate attention
• Discharge with oral rhythm control agent and anticoagulant per the patient's CHA2DS2-VASc score
• Follow up with PCP or Cardiology within 2 weeks following discharge
• Consider referral to cardiology if not consulted as inpatient

Fig. 22.6


Memorial hospital university of colorado health emergency department A-Fib pathway
Pt presents complaining of chest pain, syncope, weakness, Hx of A-Fib, palpitations, signs of shock, low BP, N/V
RN assessment of rate, regularity,and quality of pulse.Assess for precipitating causes–see below
EKG is performed and read by physician within 10 min.
Pt in A-Fib?
Yes
No
Use appropriate pathway for patient complaint
Rate controlled?
Yes
No
Pt stable
Yes
No
Refer to ED rhythm control algorithm
Immediate synchronized cardioversion
New onset?
Yes
No
Use appropriate pathway for patient complaint
Onset > 48 h?
Yes
No
Refer to ED rhythm control algorithm
Yes
Orders:
Nursing:O2, Monitor, IV
Lab:CMP, CBC, LFT, Coags, TSH,proBNP, cardiac enzymes
Rad:CXR, echocardiogram
Discharge
Yes
No
Admit to Obs or inpatient
ED discharge criteria
Asymptomatic
Stable blood pressure
Follow up with cardiology within 24 h preferred
If patient doesn't chemically convert and cannot see cardiologist in 24 h, then consult cardiologist for possible cardioversion or anticoagulation therapy
Precipitating causes of A-Fib
HTN, sleep apnea, hyperthyroid, diabetes, cardiac ischemia, pericarditis, heart faiure, valvular disease, recent CT surgery, COPD, excess ETOH intake, caffeine
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MEMORIAL HOSPITAL

Fig. 22.7

2900A [redacted]

Atrial Fibrillation Admission Orders

.PLACE LABEL HERE, .PLACE LABEL HERE
06/17/2014

Progress Notes	Physician's Orders Any pre-marked order that is changed by the MD requires MD initials beside the order
	Admit to:
	☒ Print & Complete Nursing Infection Control Admission Surveillance Protocol (ICE)
	☒ **Vital Signs**: Routine Call provider if SBP < 90, HR <50 >120, O_2 Sats < 90%
	Diet: ☐ Regular ☐ Cardiac ☐ Diabetic
	Rate Medications:
	☒ Saline Lock IV
	☐ Diltiazem Drip
	- Diltiazem 20mg slow IV push over 2 minutes
	- Diltiazem drip at 10mg/hr
	- After 15 minutes if heart rate is still > 120BPM, give an additional 25mg slow IV push over 2 minutes & increase drip to 15mg/hr
	- Discontinue diltiazem drip for HR< 60BPM or SBP< 90
	☐ Digoxin 0.5mg IV x 1 dose then 0.25mg IV Q 6 H x 2 doses
	- Then digoxin 0.25 mg po daily. Hold for HR < 60 BPM
	- Pharmacy to dose if CrCl < 30 mL/min
	☐ Metoprolol 25mg PO Q 12 H. Hold for SBP≤100 Or HR≤60
	☐ Amiodarone 150 mg IV bolus over 10 min **then** 1 mg/min IV X 6 hours **then** decrease to 0.5 mg/min IV x 18 hours **then** d/c drip.
	Antiarrhythmics: Hold for HR ≤ _____
	☐ Amiodarone ___ mg PO ___ (start after drip d/c'd)
	☐ Sotalol ___ mg PO Q 12 H
	☐ Dronedarone 400mg PO BID Meals
	☐ Propafenone SR ___ mg PO Q 12 H
	☐ Flecainide ___ mg PO Q 12 H
	Labs:
	☐ TSH ☐ T4 (Thyroxine) Total ☐ T4 (Thyroxine) FREE
	☐ CBC ☐ BMP ☐ CMP ☐ BNP ☐ Magnesium
	☐ CKMB ☐ Troponin Ultra I
	☐ PT/INR now ☐ PT/INR Q AM
	☒ Hemoglobin A1C if not drawn in past 90 days
	Radiology:
	☒ EKG now, Dr. _____ to read
	☐ EKG Q AM, Dr. _____ to read
	☐ 2D ECHO RE: _____, Dr. _____ to read
	☐ CXR PA and Lateral RE: _____
Physician's Signature: _____	Physician's Signature: _____
Date: _____ Time: _____	Date: _____ Time: _____

Fig. 22.8

2900A

[redacted]

Atrial Fibrillation Admission Orders

.PLACE LABEL HERE, .PLACE LABEL HERE
06/17/2014

Progress Notes	Physician's Orders Any pre-marked order that is changed by the MD requires MD initials beside the order
	Consults:
	☒Cardiology- Dr._____ RE: A Fib
	☒Consult the A Fib Coordinator at 260-AFIB (2342)
	☒Consult Baptist Sleep Center if pt high risk for sleep apnea or has witness apneic periods, 260-4300
	☒Consult Diabetes Educator if HgbA1C ≥ 7%
Reference for CHA_2DS_2VASC: ◄	**Anticoagulation:**
Congestive Heart Failure ☐ (1 pt)	☒Document pt's CHA_2DS_2VASC SCORE:_____
Hypertension History ☐ (1 pts)	☒If CHA_2DS_2VASC score ≥ 2, and anticoagulation is NOT ordered, document reason:_____
Age 75 years or older ☐ (2 pt)	☐EC ASA 81mg PO Daily
Age 65 – 74yrs ☐ (1 pt)	☐EC ASA 325mg PO Daily
Diabetes – type I or II ☐ (1 pts)	☐Heparin Nomogram-cardiac or other (not VTE)
Stroke, TIA or Thromboembolism history ☐ (2 pt)	☐Enoxaparin 1mg/kg SQ Q 12 H
Vascular Disease ☐ (1 pt)	☐Enoxaparin 1mg/kg SQ Q 24 H
Sex Female ☐ (1 pt)	☐Enoxaparin 0.75mg/kg SQ Q 12 H
TOTAL (0 – 9) _____	☐Rivaroxaban 20mg PO Q PM
	Rivaroxaban 15mg PO Q PM if ☐CrCl 30 - 50 mL/min
	☐Apixaban 5mg PO Q 12 H
	Apixaban 2.5mg PO Q 12H if pt has **at least 2** of following: ☐age >80yrs ☐body wt ≤60kg ☐serum creat ≥ 1.5mg/dl or CrCl 15-30 mL/min
	☐Dabigatran 150mg PO Q 12 H
	Dabigatran 75mg PO Q 12H if ☐CrCl 15-30 mL/min or ☐age ≥80
	☐Warfarin_____mg PO Daily
	☒Consult Pharmacy for Anticoagulant dose management
Physician's Signature:_____	Physician's Signature:_____
Date:_____ Time:_____	Date:_____ Time:_____

Fig. 22.8 (continued)

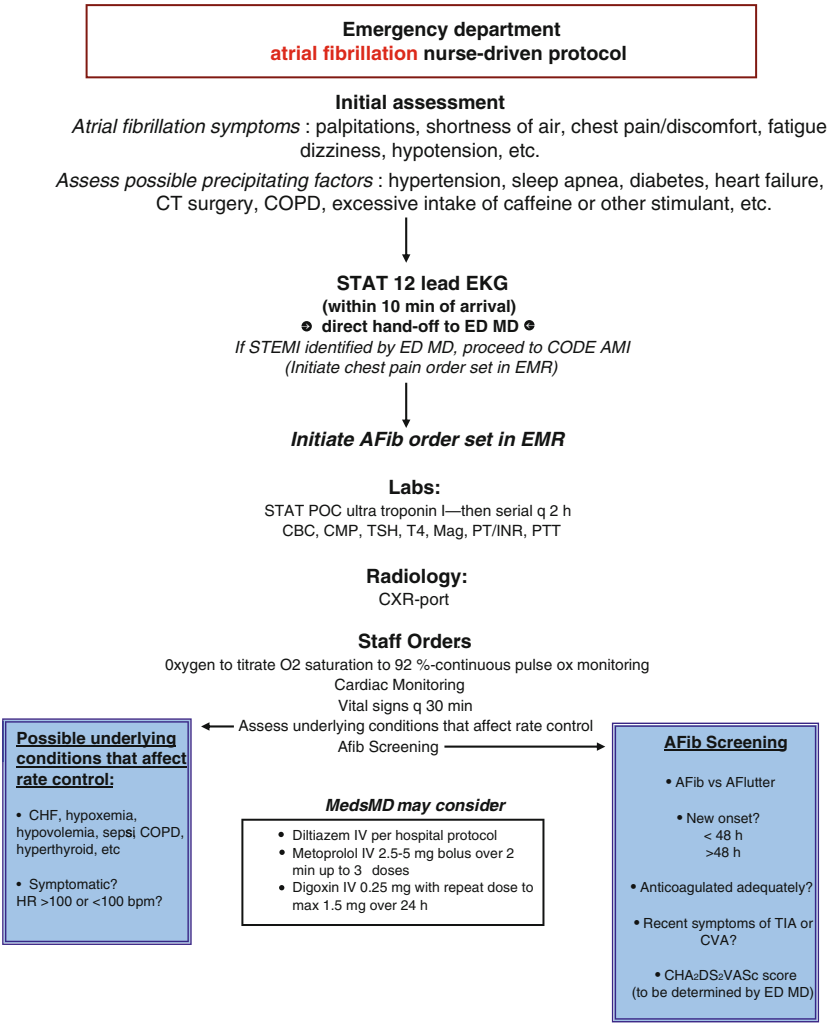
Emergency department
atrial fibrillation nurse-driven protocol
Initial assessment
Atrial fibrillation symptoms : palpitations, shortness of air, chest pain/discomfort, fatigue, dizziness, hypotension, etc.
Assess possible precipitating factors : hypertension, sleep apnea, diabetes, heart failure, recent CT surgery, COPD, excessive intake of caffeine or other stimulant, etc.
STAT 12 lead EKG
(within 10 min of arrival)
direct hand-off to ED MD
If STEMI identified by ED MD, proceed to CODE AMI
(Initiate chest pain order set in EMR)
Initiate AFib order set in EMR
Labs:
STAT POC ultra troponin I—then serial q 2 h
CBC, CMP, TSH, T4, Mag, PT/INR, PTT
Radiology:
CXR-port
Staff Orders
0xygen to titrate O2 saturation to 92 %-continuous pulse ox monitoring
Cardiac Monitoring
Vital signs q 30 min
Assess underlying conditions that affect rate control
Afib Screening
Possible underlying conditions that affect rate control:
• CHF, hypoxemia, hypovolemia, sepsi COPD, hyperthyroid, etc
• Symptomatic? HR >100 or <100 bpm?
MedsMD may consider
• Diltiazem IV per hospital protocol
• Metoprolol IV 2.5-5 mg bolus over 2 min up to 3 doses
• Digoxin IV 0.25 mg with repeat dose to max 1.5 mg over 24 h
AFib Screening
• AFib vs AFlutter
• New onset? < 48 h >48 h
• Anticoagulated adequately?
• Recent symptoms of TIA or CVA?
• CHA2DS2VASc score (to be determined by ED MD)

Fig. 22.9

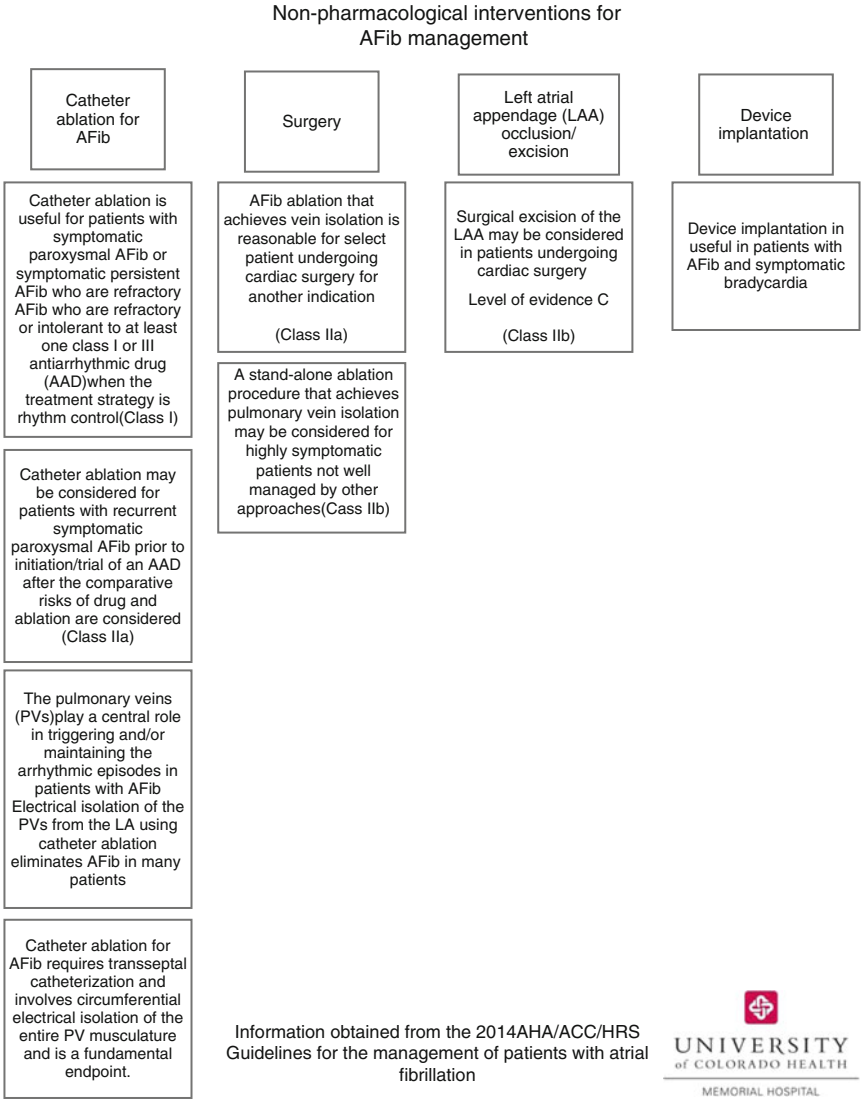
Non-pharmacological interventions for AFib management
Catheter ablation for AFib
Surgery
Left atrial appendage (LAA) occlusion/ excision
Device implantation
Catheter ablation is useful for patients with symptomatic paroxysmal AFib or symptomatic persistent AFib who are refractory AFib who are refractory or intolerant to at least one class I or III antiarrhythmic drug (AAD)when the treatment strategy is rhythm control(Class I)
AFib ablation that achieves vein isolation is reasonable for select patient undergoing cardiac surgery for another indication
(Class IIa)
Surgical excision of the LAA may be considered in patients undergoing cardiac surgery
Level of evidence C
(Class IIb)
Device implantation in useful in patients with AFib and symptomatic bradycardia
A stand-alone ablation procedure that achieves pulmonary vein isolation may be considered for highly symptomatic patients not well managed by other approaches(Cass IIb)
Catheter ablation may be considered for patients with recurrent symptomatic paroxysmal AFib prior to initiation/trial of an AAD after the comparative risks of drug and ablation are considered (Class IIa)
The pulmonary veins (PVs)play a central role in triggering and/or maintaining the arrhythmic episodes in patients with AFib Electrical isolation of the PVs from the LA using catheter ablation eliminates AFib in many patients
Catheter ablation for AFib requires transseptal catheterization and involves circumferential electrical isolation of the entire PV musculature and is a fundamental endpoint.
Information obtained from the 2014AHA/ACC/HRS Guidelines for the management of patients with atrial fibrillation
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Fig. 22.10

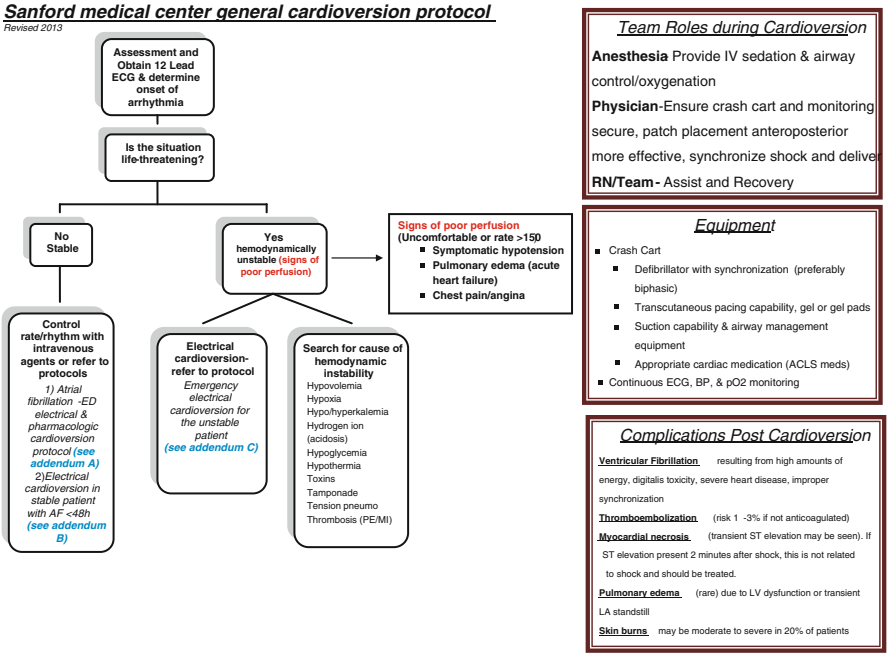

Sanford medical center general cardioversion protocol
Revised 2013
Assessment and Obtain 12 Lead ECG & determine onset of arrhythmia
Is the situation life-threatening?
No Stable
Yes hemodynamically unstable (signs of poor perfusion)
Signs of poor perfusion (Uncomfortable or rate >150
Symptomatic hypotension
Pulmonary edema (acute heart failure)
Chest pain/angina
Control rate/rhythm with intravenous agents or refer to protocols
1) Atrial fibrillation -ED electrical & pharmacologic cardioversion protocol (see addendum A)
2)Electrical cardioversion in stable patient with AF <48h (see addendum B)
Electrical cardioversion- refer to protocol
Emergency electrical cardioversion for the unstable patient
(see addendum C)
Search for cause of hemodynamic instability
Hypovolemia
Hypoxia
Hypo/hyperkalemia
Hydrogen ion (acidosis)
Hypoglycemia
Hypothermia
Toxins
Tamponade
Tension pneumo
Thrombosis (PE/MI)
Team Roles during Cardioversion
Anesthesia Provide IV sedation & airway control/oxygenation
Physician-Ensure crash cart and monitoring secure, patch placement anteroposterior more effective, synchronize shock and deliver
RN/Team- Assist and Recovery
Equipment
Crash Cart
Defibrillator with synchronization (preferably biphasic)
Transcutaneous pacing capability, gel or gel pads
Suction capability & airway management equipment
Appropriate cardiac medication (ACLS meds)
Continuous ECG, BP, & pO2 monitoring
Complications Post Cardioversion
Ventricular Fibrillation resulting from high amounts of energy, digitalis toxicity, severe heart disease, improper synchronization
Thromboembolization (risk 1 -3% if not anticoagulated)
Myocardial necrosis (transient ST elevation may be seen). If ST elevation present 2 minutes after shock, this is not related to shock and should be treated.
Pulmonary edema (rare) due to LV dysfunction or transient LA standstill
Skin burns may be moderate to severe in 20% of patients

Fig. 22.11

Addendum A

Atrial fibrillation-ED electrical and pharmacologic cardioversion protocol*

General measures:
- **Ensure atrial fibrillation of recent onset (< 48 h) and not recurrent**
- NPO, bedrest, IV, monitor

Investigations:
- CK, troponin, electrolytes, CR, TSH, PT (INR)/PTT
- Echocardiogram to be arranged if not previously obtained (not necessary acutely)
- TEE in selected cases for rapid cardioversion if unknown duration or Afib > 48 h

Medications:

Anti-coagulation:Consider prior to either electric **or** pharmacologic cardioversion
- 5000 U IV bolus followed by 1000 U/hr infusion (Target PTT 50-70)
- LMWH Enoxaparin 1 mg/kg q12h (pharmacy to adjust for decreased GFR)

Rate Control (IV or PO): Do not prescribe digoxin, β-blocker or CCB if pre-excitation.
- β-blocker: Metoprolol 5mg IV q5min X 3 followed by 5-10 mg IV q6h
  - PO 25-100 mg BID
- Diltiazem 20 mg IV over 2 minutes followed by 5-15 mg/hr IV
  - PO 30-90 mg QID or use sustained release preparation.

Pharmacologic cardioversion:
- * Ibutilide: IV 1 mg over 10 min (for pts < 60 kg give 0.01 mg/kg over 10 min)
  - May repeat x 1 dose if arrhythmia not terminated
- ** Flecainide: PO 300mg_50 mg q12h
- Propafenone: 600 mg PO single dose

Electrical cardioversion:
- Anesthesia present/IV sedation/Airway control/Oxygenation
- Synchronized shock: Refer to AHA (2010) guidelines below

Doses/Details
Synchronized Cardioversion Initial recommended doses: • Narrow regular: 50-100 J • Narrow irregular: 120-200 J biphasic or 200 J monophasic • Wide regular: 100 J • Wide irregular: defibrillation dose (NOT synchronized)

Post cardioversion:
- Home: ___ hours
- ASA: _____
- Warfarin, Dabigatran, Rivaroxaban, or Apixaban for 1 month regardless of $CHADS_2$ score:_____
- Rate control:_____

Followup:
- Cardiologist within 72hours:_____
- Family MD:_____

* **Ibutilide:**Caution if Class I,III antiarrhythmic drug within 4 hours, history of polymorphic VT, long QTc, hypokalemia, hypomagnesemia, bradycardia, CHF

** **Flecainide:**hypotension, rapidly conducting Aflutter

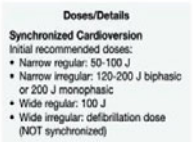
Atrial Fibrillation Committee 2013

Fig. 22.11 (continued)

Addendum B

Electrical cardioversion in stable patient with AF < 48 h
(see bottom for >48 hours or unknown onset)

1. Electrical Cardioversion [Stable patient with a tachydysrhythmia (rapid atrial fibrillation, supraventricular tachycardia/SVT, ventricular tachycardia/VT **with a pulse**)].
   a. Assessment
      i. Assess severity of symptoms. No ischemia, hypotension, or acute HF present.
      ii. Onset clear and **< 48 h.**
      iii. No evidence of digoxin toxicity or hypokalemia.
      iv. Determine amount and time of last PO intake.
      v. Assure no evidence of embolic stroke symptoms.
   b. Observation
      i. Observe rhythm 3-6 hours- keep patient NPO
   c. Cardioversion
      i. Properly place on monitor/defibrillation that is capable of providing synchronization. Apply quick combo patches anteroposterior (more effective).
      ii. Assess cardiac rhythm for appropriateness of cardioversion.
      iii. Have equipment available/prepared for possible worsening of patient's condition.
      iv. Anesthesia to provide procedural sedation and analgesia
      v. Ensure that all personnel are clear of patient before initiating shock.
      vi. Apply Synchronization—assure sensing of R wave (and not T wave)
      vii. See AHA (2010) guidelines for joule recommendations, pediatric=1 joule/kg for afib & VT, 0.5 joules/kg for SVT & aflutter



      viii. Reassess
         1. If no response to cardioversion, can repeat steps above up to 3 shocks, provider can recommend an increase in joules.**REMEMBER TO RE-SYNCRONIZE EACH TIME**. If pulseless, begin BLS/ACLS protocols for cardiac arrest. If rhythm stabilized, recover.
   d. Provide anticoagulation
      i. Recommend 4 weeks of anticoagulation after cardioversion
         1. Options: warfarin, dabigatran, rivaroxaban, or apixaban
         2. After the 4 weeks-Utilize $CHADS_2$ scoring for need of long-term anticoagulation
   e. Disposition
      i. Home after 1-2 hours of observation post procedure
      ii. Discharge home on rate control medication (BB or CCB)
      iii. Arrange for follow-up with cardiology or primary care in 1 week with suggestion for outpatient echocardiogram

If onset of a-fib (stable patient) is longer than 48 hrs or unknown, risk for stroke from embolized thrombus is increased. If stable, it is recommended to properly anticoagulate patient for 3-4 weeks before performing the cardioversion or perform a TEE immediately prior.

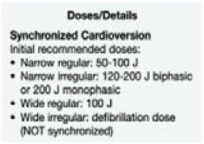
Atrial Fibrillation Committee 2013

Fig. 22.11 (continued)

Addendum C

Emergency electrical cardioversion for the unstable patient

1. Electrical Cardioversion [Unstable patient with a tachydysrhythm ia (rapid atrial fibrillation, supraventricular tachycardia/SVT, ventricular tachycardia/VT**with a pulse**)]
   a. Properly place on monitor/defibrillation that is capable of providing synchronization. Apply quick combo patches anteroposterior (more effective).
   b. Assess cardiac rhythm for appropriateness of cardioversion.
   c. Have equipment available/prepared for possible worsening of patient's condition.
   d. Try to provide procedural sedation and analgesia depending on stability.
   e. Ensure that all personnel are clear of patient before initiating shock.
   f. Apply Synchronization—assure sensing of R wave (and not T wave)
   g. See AHA (2010) guidelines below for adult joule recommendation, pediatric=1 joule/kg for afib & VT, 0.5 joules/kg for SVT & aflutter



Doses/Details
Synchronized Cardioversion Initial recommended doses: • Narrow regular: 50-100 J • Narrow irregular: 120-200 J biphasic or 200 J monophasic • Wide regular: 100 J • Wide irregular: defibrillation dose (NOT synchronized)

   h. Reassess
      i. If no response to cardioversion, can repeat steps above up to 3 shocks, provider can recommend an increase in joules.**REMEMBER TO RE-SYNCRONIZE EACH TIME**. If pulseless, begin BLS/ACLS protocols for cardiac arrest. If rhythm stabilized, recover.
   i. Follow protocol for anticoagulation post cardioversion and management of atrial fibrillation or sinus rhythm.

Atrial Fibrillation Committee 2013

Fig. 22.11 (continued)

Centura Health Call Center

PAH ATRIAL FIBRILLATION FOLLOW UP *CALLBACK*

Q - Is this a good time to talk with me?

1. Continue Script
2. Declined Callback
3. No Contact
4. Readmission
5. Expired

Info – Hello, this is _____. I'm a Registered Nurse with the Nurse Help Line at Porter Adventist Hospital. I'm calling to follow-up on how you're doing since we last talked and to try to answer any questions you may have.

Info – If you have selected Declined Callback, No Contact, Readmission or Expired, click on Next. Mark the survey as Incomplete and select a reason why from the drop down list. Document in the Comments box the circumstances of the incomplete survey. Begin the documentation with the patient's name/date of birth.

Q - Have you been in the hospital since we last talked?

- Yes
- No
- Admitted
- Seen in Emergency Department
    - **Info** – If answered Admitted or Seen in Emergency Department, indicate in the Comments at the end of the survey the patient's name and date of birth along with details of when, where and why they were admitted/seen in the hospital.

Q - Have your medications changed since we last talked?

- Yes
- No
    - **Info** – If YES, review medications that have been added or discontinued and document in survey comments. Ask the patient if they would like to review the side effects of any new medications.

Q - You need to call your physician if you start having heart symptoms. Can you name 5 symptoms that would indicate that your atrial fibrillation is worsening?

- Yes
- No
    - **Info** – Remind the patient/caregiver to call the physician for the following sustained palpitations or fast heart rate, dizziness, shortness of breath, chest pain, excessive fatigue.

Q – Are you having any of those symptoms now?

- Yes
- No
    - **Info** – If currently having symptoms, determine if the symptoms are new or worsened and do an assessment as needed.

Fig. 22.12

Anticoagulation Clinic Protocol[1,2]

I. Purpose□

The goal of the anticoagulation clinic is to achieve therapeutic anticoagulation levels in patients while minimizing bleeding risks. The secondary goal is the increase patient education and monitoring which will lead to better compliance and better outcomes.

II. INR Monitoring in patients not at goal:

Monitored weekly until stable. Patients with dosage adjustments, addition or deletion of drugs that interact with the effects of warfarin will be monitored weekly post-change until INR is stable. □**Stable patients:** Monitored every 4-6 weeks.

III. Goal of Anticoagulation Ranges & Duration of Therapy

:□This is determined by the patient's clinical indication for anticoagulation and the patient's response to anticoagulation. General guidelines are listed below. This will be modified according the patient's specific clinical condition

Warfarin Indication	Target INR	Duration of Therapy

[1] Ebell, MH. Family Practice Management. May 2005:77-83.

[2] Adapted from South Denver Cardiology Anticoagulation Clinic Protocal

Heart and Vascular Institute
Porter Adventist Hospital
Centura Health

Fig. 22.13

	Range	
Atrial Fibrillation with CHADS-VASC score of 2 or higher	2.0-3.0	Lifetime
Prophylaxis & treatment of venous thrombosis	2.0-3.0	3-6 months
Treatment of pulmonary embolism	2.0-3.0	6-12 months
First systemic embolism (any source)	2.0-3.0	1 year, longer depending on results of clinical testing
Tissue heart valves	2.0-3.0	3 months post surgery, can vary by specific type of heart valve
Acute myocardial infarction with decreased anterior wall motion and potential thrombus formation	2.0-3.0	12 months, longer depending on recovery of LV function.
Mechanical heart valves	2.5-3.5	Lifetime
Hypercoaguable states	2.0-3.0	Lifetime

IV. Dosage Adjustment

A. Dosage Adjustment Algorithms

For target INR of 2.0 to 3.0, with no bleeding

INR	<1.5	1.5 – 1.9	2.0-3.0	3.1-3.9	4.0-4.9	> 5.0
Adjustment	Increase dose by 20% weekly	Increase dose by 10-15% weekly	none	Decrease weekly dose by 5-10%	Hold for 1 day then decrease weekly	See below

Fig. 22.13 (continued)

					dose by 10%	
Next INR	4-7 days	4-7 days	2-4 weeks	7 -10 days	7 days	

B. Dosage Adjustment Algorithms
For target INR of 2.5 to 3.5, with no bleeding

INR	<1.5	1.5 – 2.4	2.5-3.5	3.6-4.5	4.5-6.0	>6.0
Adjustment	Increase dose by 20% weekly	Increase dose by 10-15% weekly	none	Decrease weekly dose by 5-10%	Hold for 1 day then decrease weekly dose by 10%	See below
Next INR	4-7 days	4-7 days	2-4 weeks	7-10 days	4-7 days	

C. Dosage Adjustment for significantly elevated INR

1. INR 5.0 to 8.9 – with no significant bleeding. Omit 2 doses, reduce weekly dose by 20%. Next INR in 7 days or less. Vitamin K 2.5 to 5.0 mg if bleeing.

2. INR > 9.0 – Omit until INR values less than 5.0. Administer Vitamin K 2.5 to 5.0 mg.

3. Bleeding – If the patient is actively bleeding, Vit K 5-10mg IV, consider for FFP and transfer to ED.

VI. Patient Monitoring

If the INR values are out of range, the patient will be questioned regarding the following:

a. Compliance with recommended dose

b. Other medications, including over the counter, started or discontinued since last visit. This may contribute to the INR values out of range

c. Herbal products started or discontinued since last visit

d. Changes in lifestyle such as diet or exercise since last visit

Fig. 22.13 (continued)

e. Use of alcohol

f. Bruising or bleeding problems

h. The patient will also be counseled regarding the importance of maintaining anticoagulation, signs of hypercoagulation, and the importance of compliance and follow-up monitoring. VIII.

VII. Novel Oral Anticoagulants (NOACs)

Dabigitran, Rivarobaxan and Apixiban are three new oral anticoagulants that have been introduced in the last few years. These new medications provide therapeutic levels of anticoagulation for patients with non-valvular atrial fibrillation. Additional indications are currently being evaluated. These medications do not require follow up with frequent INR monitoring. These medications do have an associated risk of bleeding similar to warfarin and have unique contraindications and side effects. Patients will be counseled regarding the use of these medications.

Fig. 22.13 (continued)

Porter Adventist Hospital
Centura Health.
2525 South Downing Street
Denver, Colorado 80210-5876
Phone: (303) 765-6500
Fax: (303) 765-6535

ANTICOAGULATION CENTER REFERRAL & AGREEMENT

Patient Name_____
Address_____
Telephone #_____
M___F___DOB_____
Insurance_____
Anticoagulation indication (ICD-9 Code)_____
Target INR_____
Duration of Therapy_____
Current Coumadin dose_____
Primary Physician_____
Physician to follow therapy _____
Last INR_____ **Date**_____

I authorize the Porter Adventist Anticoagulation Center to manage this patient's anticoagulation therapy according to the Center's protocols that have been approved by the Medical Director, and as outlined by the State of Colorado Board of Pharmacy Rules and Regulations. I understand that INRs will be ordered and performed according to the Center's guidelines, and that they will be billed to the patient's insurance with my name as the ordering physician. This standing order is effective for the three months beginning _____and ending_____.
****Protocols are available upon request.*

_____ _____/_____
Referring Physician's signature License # Date

_____ _____
Physician's name (printed) Susan Warburton, PharmD

_____ _____
Physician's address Mistie Nguyen, BSPharm, PharmD

_____ _____
Heather Seashore, PharmD

_____
Physician's phone number

PLEASE FAX TO (303) 765-6535 WITH A COPY OF FRONT AND BACK OF INSURANCE CARD AND ANY PERTINENT CLINICAL NOTE
CONTACT PATIENT TO SCHEDULE APPOINTMEN

Heart and Vascular Institute
Porter Adventist Hospital
Centura Health.

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Fig. 22.13 (continued)

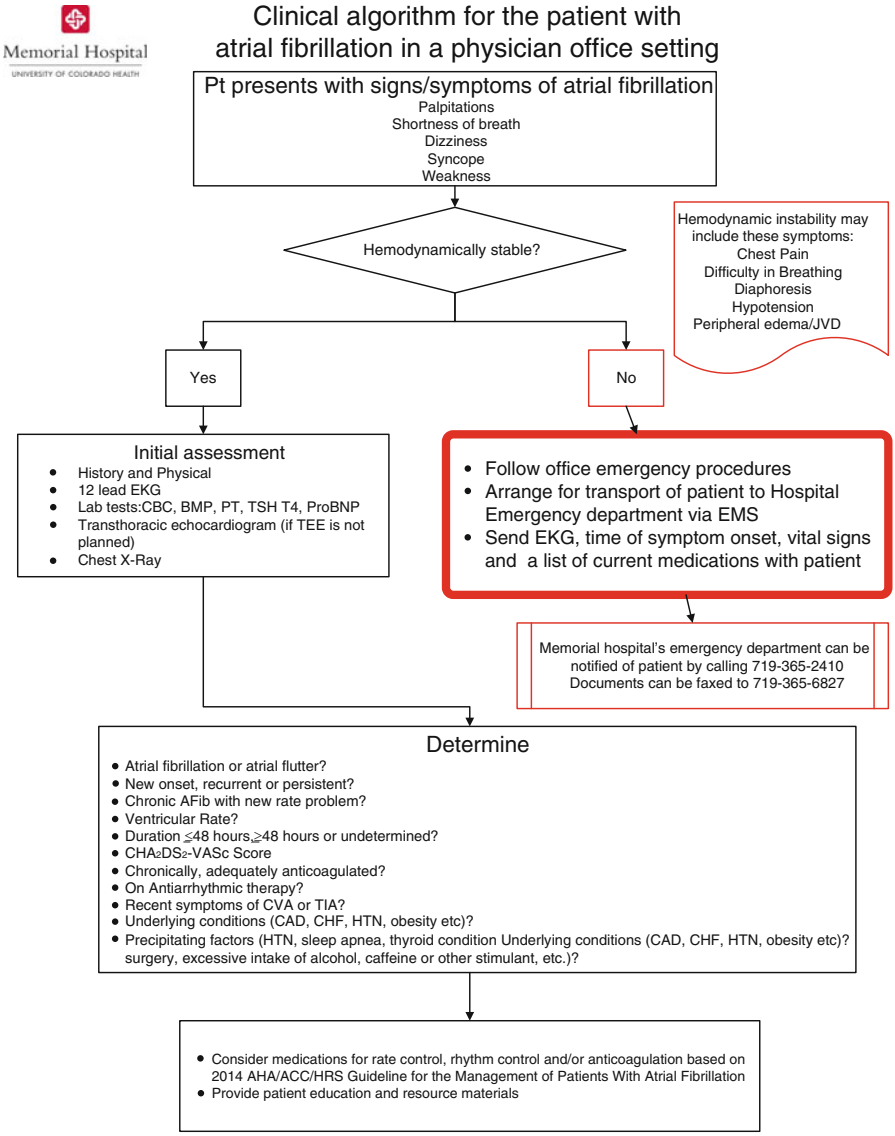
Memorial Hospital
UNIVERSITY OF COLORADO HEALTH
Clinical algorithm for the patient with atrial fibrillation in a physician office setting
Pt presents with signs/symptoms of atrial fibrillation
Palpitations
Shortness of breath
Dizziness
Syncope
Weakness
Hemodynamically stable?
Hemodynamic instability may include these symptoms:
Chest Pain
Difficulty in Breathing
Diaphoresis
Hypotension
Peripheral edema/JVD
Yes
No
Initial assessment
History and Physical
12 lead EKG
Lab tests:CBC, BMP, PT, TSH T4, ProBNP
Transthoracic echocardiogram (if TEE is not planned)
Chest X-Ray
Follow office emergency procedures
Arrange for transport of patient to Hospital Emergency department via EMS
Send EKG, time of symptom onset, vital signs and a list of current medications with patient
Memorial hospital's emergency department can be notified of patient by calling 719-365-2410
Documents can be faxed to 719-365-6827
Determine
Atrial fibrillation or atrial flutter?
New onset, recurrent or persistent?
Chronic AFib with new rate problem?
Ventricular Rate?
Duration ≤48 hours,≥48 hours or undetermined?
CHA2DS2-VASc Score
Chronically, adequately anticoagulated?
On Antiarrhythmic therapy?
Recent symptoms of CVA or TIA?
Underlying conditions (CAD, CHF, HTN, obesity etc)?
Precipitating factors (HTN, sleep apnea, thyroid condition Underlying conditions (CAD, CHF, HTN, obesity etc)? surgery, excessive intake of alcohol, caffeine or other stimulant, etc.)?
Consider medications for rate control, rhythm control and/or anticoagulation based on 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation
Provide patient education and resource materials

Fig. 22.14

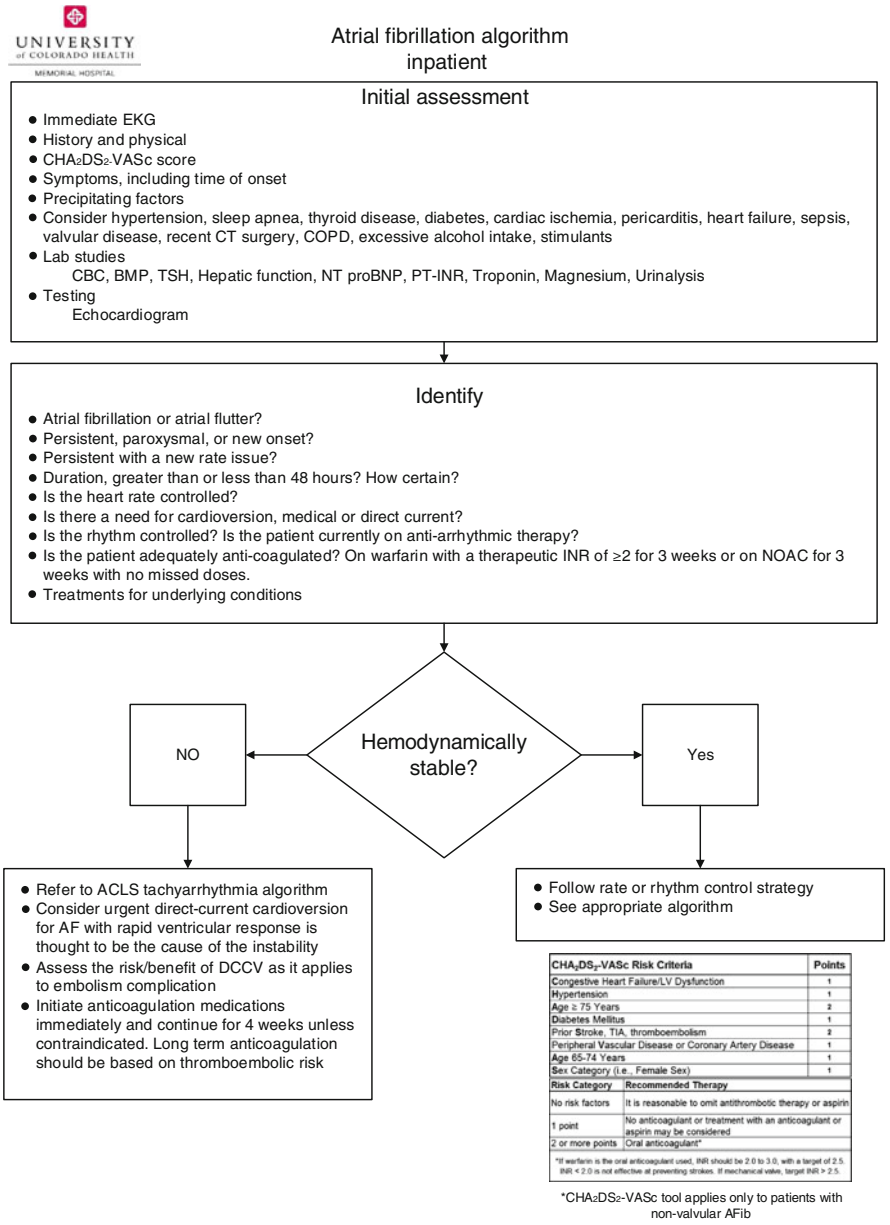
UNIVERSITY of COLORADO HEALTH
MEMORIAL HOSPITAL
Atrial fibrillation algorithm
inpatient
Initial assessment
Immediate EKG
History and physical
CHA2DS2-VASc score
Symptoms, including time of onset
Precipitating factors
Consider hypertension, sleep apnea, thyroid disease, diabetes, cardiac ischemia, pericarditis, heart failure, sepsis, valvular disease, recent CT surgery, COPD, excessive alcohol intake, stimulants
Lab studies
CBC, BMP, TSH, Hepatic function, NT proBNP, PT-INR, Troponin, Magnesium, Urinalysis
Testing
Echocardiogram
Identify
Atrial fibrillation or atrial flutter?
Persistent, paroxysmal, or new onset?
Persistent with a new rate issue?
Duration, greater than or less than 48 hours? How certain?
Is the heart rate controlled?
Is there a need for cardioversion, medical or direct current?
Is the rhythm controlled? Is the patient currently on anti-arrhythmic therapy?
Is the patient adequately anti-coagulated? On warfarin with a therapeutic INR of ≥2 for 3 weeks or on NOAC for 3 weeks with no missed doses.
Treatments for underlying conditions
Hemodynamically stable?
NO
Yes
Refer to ACLS tachyarrhythmia algorithm
Consider urgent direct-current cardioversion for AF with rapid ventricular response is thought to be the cause of the instability
Assess the risk/benefit of DCCV as it applies to embolism complication
Initiate anticoagulation medications immediately and continue for 4 weeks unless contraindicated. Long term anticoagulation should be based on thromboembolic risk
Follow rate or rhythm control strategy
See appropriate algorithm
CHA2DS2-VASc Risk Criteria
Points
Congestive Heart Failure/LV Dysfunction
1
Hypertension
1
Age ≥ 75 Years
2
Diabetes Mellitus
1
Prior Stroke, TIA, thromboembolism
2
Peripheral Vascular Disease or Coronary Artery Disease
1
Age 65-74 Years
1
Sex Category (i.e., Female Sex)
1
Risk Category
Recommended Therapy
No risk factors
It is reasonable to omit antithrombotic therapy or aspirin
1 point
No anticoagulant or treatment with an anticoagulant or aspirin may be considered
2 or more points
Oral anticoagulant*
*If warfarin is the oral anticoagulant used, INR should be 2.0 to 3.0, with a target of 2.5. INR < 2.0 is not effective at preventing strokes. If mechanical valve, target INR > 2.5.
*CHA2DS2-VASc tool applies only to patients with non-valvular AFib

Fig. 22.15

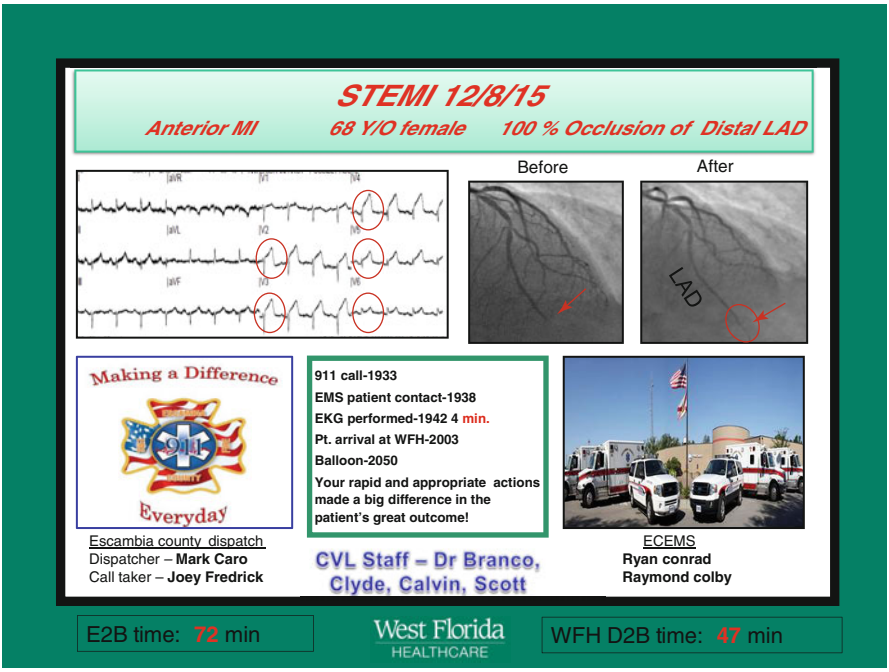
STEMI 12/8/15
Anterior MI
68 Y/O female
100 % Occlusion of Distal LAD
Before
After
LAD
Making a Difference
911
Everyday
911 call-1933
EMS patient contact-1938
EKG performed-1942 4 min.
Pt. arrival at WFH-2003
Balloon-2050
Your rapid and appropriate actions made a big difference in the patient's great outcome!
Escambia county dispatch
Dispatcher – Mark Caro
Call taker – Joey Fredrick
CVL Staff – Dr Branco, Clyde, Calvin, Scott
ECEMS
Ryan conrad
Raymond colby
E2B time: 72 min
West Florida HEALTHCARE
WFH D2B time: 47 min

Fig. 22.16

Index

W.F. Peacock, C.L. Clark (eds.), *Short Stay Management of Atrial Fibrillation*, Contemporary Cardiology, DOI 10.1007/978-3-319-31386-3