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The Practical Journal for Emergency Physicians

Emergency Medicine Reports®

Volume 28, Number 20

September 17, 2007

On nearly every shift, the emergency physician confronts a patient with atrial fibrillation (AF), either new onset or chronic. AF is often seen in patients with congestive heart failure or prior myocardial infarction. As the Baby Boomers continue to age, geriatric conditions such as AF will continue to increase, requiring the emergency physician to be able to detect the arrhythmia, search for underlying conditions that may cause or exacerbate the rhythm, and appropriately treat it. In some cases, there may be interest in converting the patient to sinus rhythm. Attempts at chemical or electrical cardioversion often take place during the ED visit. Where cardioversion is not attempted or not successful, rate control and anticoagulation may be needed. This paper will discuss the approach to the patient who presents to the ED with AF.

—Sandra M. Schneider, MD, FACEP, Editor

Evaluating and Treating Atrial Fibrillation in the Emergency Department

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Introduction

Atrial fibrillation (AF) is the most common sustained arrhythmia, the most common arrhythmia encountered by emergency department providers, and results in an overall and cardiovascular mortality twice that seen in patients without AF.[1] Atrial fibrillation may result in ventricular response rates of greater than 150 beats per minute, leading to a hemodynamically compromised state requiring emergent emergency department (ED) cardioversion. Or, it may herald myocardial ischemia, pulmonary embolism, or hyperthyroidism.

Regardless of the etiology of the AF, ED providers must be familiar with the pathophysiology and clinical features of AF. The presence of a left bundle branch block, an accessory pathway, or Ashman phenomenon cannot be allowed to preclude an accurate electrocardiographic

Statement of Financial Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Farel (CME question reviewer) owns stock in Johnson & Johnson. Dr. Casaletto (author) received grant/research support from the state of Arizona Governor's Office for Children, Youth, and Families. Dr. Berman (author), Dr. Glauser (peer reviewer), Dr. Robinson (peer reviewer), Dr. Schneider (editor), and Dr. Stapczynski (editor) report no relationships with companies related to the field of study covered by this CME activity.

diagnosis of AF. Management based upon symptomatology and etiology is crucial in reducing the risk of cardiac and thromboembolic complications triggered by AF. Current clinical controversies including identification of hemodynamically stable patients in which ED cardioversion is indicated, examination of the role of anticoagulation in ED patients with AF, and exploration of which patient population may be safely discharged from the ED recreate diagnostic dilemmas ED physicians face daily.

While reading the following article, readers should consider the management of these three cases:

Case 1. A 42-year-old male with hypercholesterolemia presents to the ED after noting lightheadedness and dyspnea upon awakening. His vital signs are normal; however, his ECG reveals an irregularly, irregular narrow complex rhythm without clear P waves. Is cardioversion or anticoagulation required in the ED?

Case 2. A 65-year-old female with hypertension and a history of AF presents with dyspnea. She has rales and jugular venous distention. Her vital signs are as follows: BP 104/58, P 175, RR 20, Sa02 95%. Cardioversion or rate control in the ED? If rate control, what agent is the best choice for rate control in this patient?

Case 3. An otherwise healthy 29-year-old male presents following a syncopal episode sustained while playing volleyball. He complains of intermittent palpitations, but is currently asymptomatic. His blood pressure is 124/76. His ECG reveals an irregularly, irregular wide complex tachycardia with delta waves visible in V4 and V5. Cardioversion or rate control in the ED? If rate control, what agent is the best choice for rate control in this patient?

Definition

Atrial fibrillation is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. Atrial fibrillation is depicted on electrocardiogram (ECG) by the replacement of a consistent P wave preceding every QRS complex by rapid oscillations or fibrillatory waves that vary in size, shape, and timing. Ventricular response to this disorganized atrial activity, and hence QRS placement on ECG, is irregularly, irregular. (*See Figures 1-5.*)

Acute AF describes an episode of less than 48 hours duration; it can apply to new-onset or recurrent AF. *Paroxysmal AF* lasts less than seven days and may be recurrent, defined as two or more episodes. *Persistent AF* fails to self-terminate and lasts longer than seven days. *Permanent AF* lasts more than one year; cardioversion has not been attempted or has failed. *Secondary AF* occurs in the setting of acute myocardial infarction, cardiac surgery, pericarditis, myocarditis, hyperthyroidism, pulmonary embolism, pneumonia, or acute pulmonary disease; therefore, treatment of the underlying disorder with concurrent management of the AF may result in termination of the arrhythmia without recurrence. *Lone AF* applies to individuals younger than 60 years of age without clinical or echocardiographic evidence of cardiopulmonary disease. These patients have a favorable prognosis with respect to thromboembolism and mortality. Atrial fibrillation with rapid ventricular response occurs when ventricular rates exceed 100 beats per minute.

Epidemiology

Atrial fibrillation is a common arrhythmia. Its prevalence in the United States is estimated at 2.3 million people.[2] It is believed to be an independent risk factor for death, with a relative risk of 1.5 for men and 1.9 for women.[1] Atrial fibrillation can result in serious complications, including congestive heart failure and thromboembolism. It is the underlying cause of 30,000 to 40,000 embolic strokes per year in the United States.[3]

Treatment of AF has changed in recent years. The Rate Control versus Electrical Cardioversion for Persistent Atrial Fibrillation (RACE) trial found that rate and rhythm control are comparable in terms of mortality from cardiovascular causes, heart failure, thromboembolic complications, bleeding, pacemaker implantation, and serious adverse effects of drugs.[4] The Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) trial, designed to assess mortality, confirmed no significant difference in the above end points between the rate and rhythm control groups at five years.[5]

Pathophysiology

In most cases of atrial fibrillation, small reentrant circuits constantly arise in the atria.[6] The "multiple wavelet" model suggests that AF is sustained by multiple simultaneous wavelets wander-

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by AHC Media LLC, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Senior Vice President/Group Publisher: Brenda Mooney
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Marketing Manager: Shawn DeMario
GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $31. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $359 each; 10 to 20 additional copies, $319 each.

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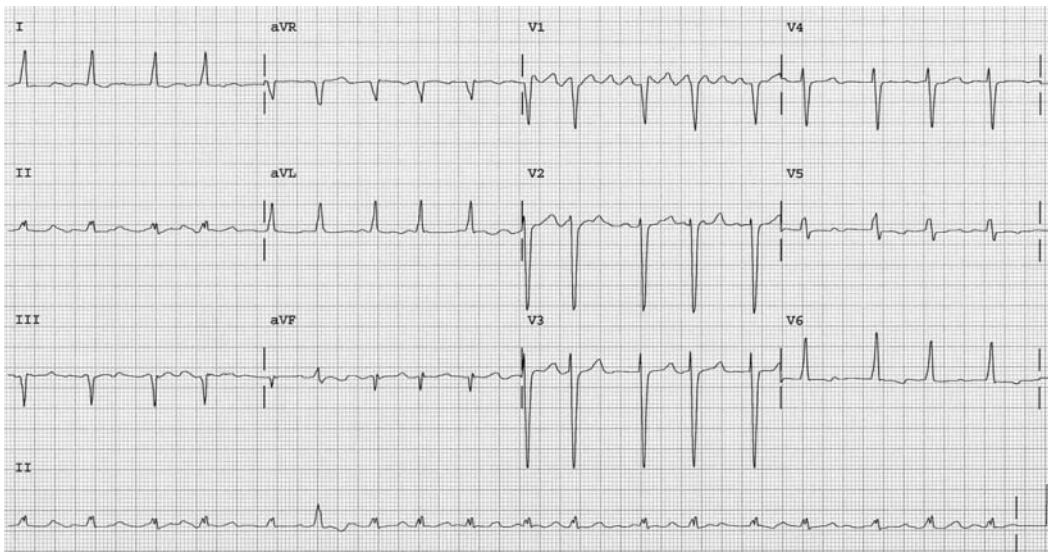
Figure 1. AF in a 12 Lead



ing throughout the atria.[7] Therefore, therapy is aimed at making these wavelets less likely to sustain and propagate. Such treatments include antiarrhythmic medications and surgical interruption of the atrial tissue. More recently, it has been recognized that the initiation of AF is due to premature atrial contractions caused by triggering beats arising from the pulmonary veins in close proximity to their junction with the left atrium.[8] These triggers may also fire repetitively and contribute to the maintenance of atrial fibrillation, essentially becoming drivers of AF.

Etiology

Specific cardiovascular conditions associated with AF include valvular heart disease, coronary artery disease, and hypertension, particularly when left ventricular hypertrophy is present. Patients with AF presenting to the ED have a higher incidence of organic heart disease than those seen in an ambulatory clinic setting, where the incidence of lone atrial fibrillation can be higher than 30%.[9]

Atrial fibrillation may be related to temporary causes, including alcohol intake (known as "holiday heart syndrome"), surgery, myocardial infarction, pericarditis, myocarditis, pulmonary embolism or other pulmonary diseases, and hyperthyroidism or other metabolic disorders. Holiday heart syndrome occurs after bouts of heavy alcohol ingestion and is thought to be due to catecholamine release. In such cases, successful treatment of the underlying condition may eliminate AF. Atrial fibrillation commonly occurs in the early postoperative period as complication of cardiac or thoracic surgery. Approximately 30% to 45% of paroxysmal and 20% to 25% of persistent cases of AF occur in younger patients without demonstrable underlying cardiac disease.[10] Furthermore, alcohol may be responsible for as many as two-thirds of patients under 65 years of age with new-onset AF.

In addition, AF may also be associated with structural causes such as hypertrophic or dilated cardiomyopathies or congenital heart disease, especially atrial septal defects that persist into adulthood. Less common structural etiologies include restrictive cardiomyopathies, cardiac tumors, and constrictive pericarditis.

Clinical Features

Some patients with atrial fibrillation have no symptoms, while others are severely affected. Presenting symptoms range from palpitations to acute pulmonary edema, but fatigue and other nonspecific symptoms such as weakness, dizziness, and dyspnea are more common. Interestingly, asymptomatic atrial fibrillation occurs frequently.[11] The Canadian Registry of Atrial Fibrillation found that 21% of newly diagnosed patients were asymptomatic.[12] In addition, patients initially presenting with symptomatic AF commonly have asymptomatic recurrences.[13] Decreased cardiac output resulting from tachycardia, loss of atrial contribution to left ventricular filling, increased valvular regurgitation, and irregular ventricular response lead to the constellation of symptoms with which patients present.

When taking a history, ask about common AF triggers including excessive alcohol, caffeine, sleep deprivation, or emotional stress. Determine whether the onset and termination of palpitations is abrupt, suggesting an atrial or another supraventricular tachyarrhythmia, or gradual, suggesting sinus tachycardia. Dyspnea may indicate underlying heart disease, whereas anginal symptoms point toward coronary artery disease. Syncope may be associated with AF, but is more commonly associated with ventricular arrhythmias.

Irregular pulse, irregular jugular venous pulsations, and variation in the volume of the first heart sound imply AF on physical examination. Listen for a heart murmur indicating the presence of valvular heart disease.

Diagnostic Studies

In addition to a conscientious history and physical examination, diagnostic studies aid in the evaluation of patients with AF. Confirming the diagnosis of AF requires ECG documentation by at least single-lead ECG, revealing the lack of P waves and an irregularly, irregular ventricular response. Electrocardiography will not only confirm the presence of AF, but may suggest an etiology by bringing to light abnormal intervals, prior or current myocardial infarction, or chamber hypertrophy. A rapid, irregular, sustained, wide-complex tachycardia strongly suggests AF with conduction over an accessory pathway, an aberrant conduction (Ashman phenomenon), or an underlying bundle-branch block. Extremely rapid rates (greater than 200 bpm) suggest the presence of an accessory pathway, such as AF with Wolff-Parkinson-White (WPW). (*See Figures 6-11.*)

Figure 2. AF: Note the Fibrillation Waves in V1

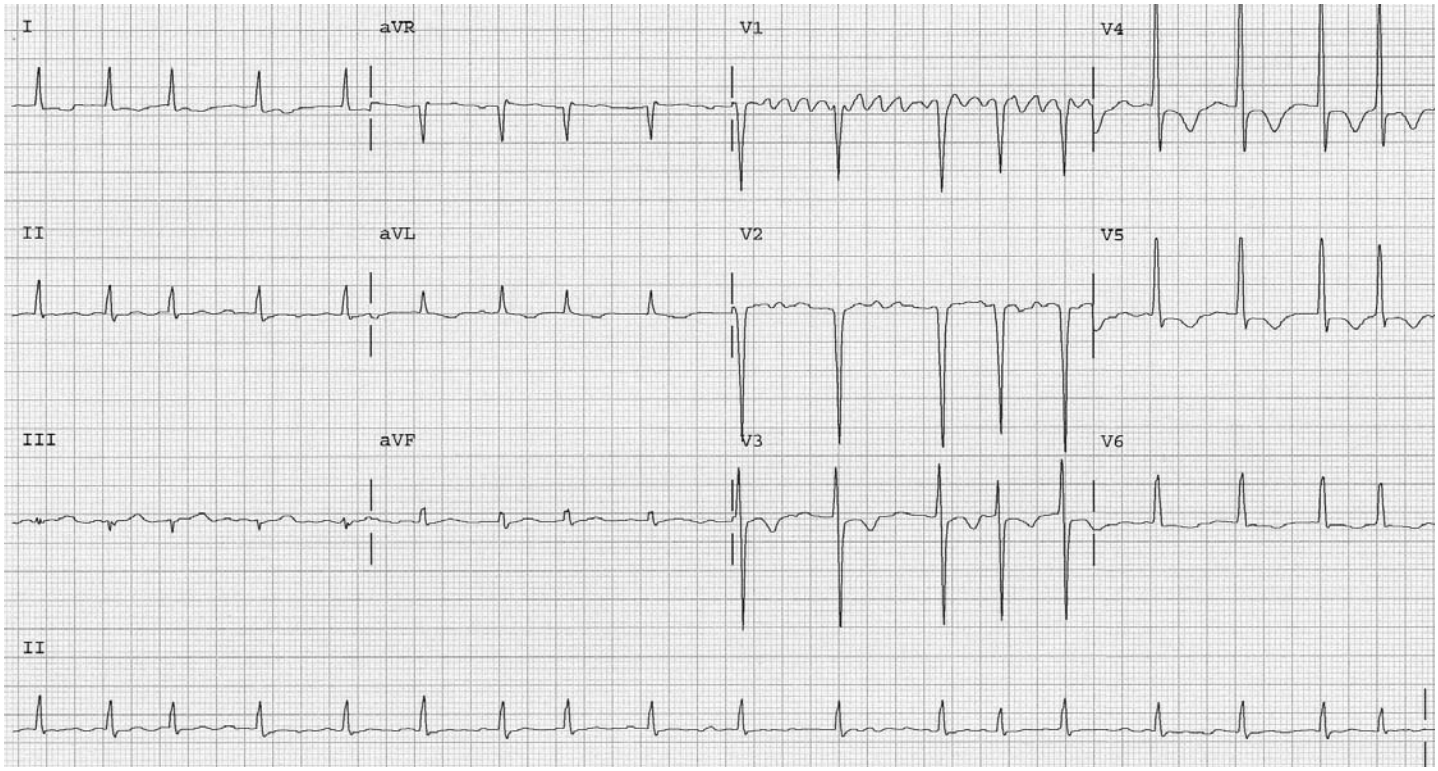
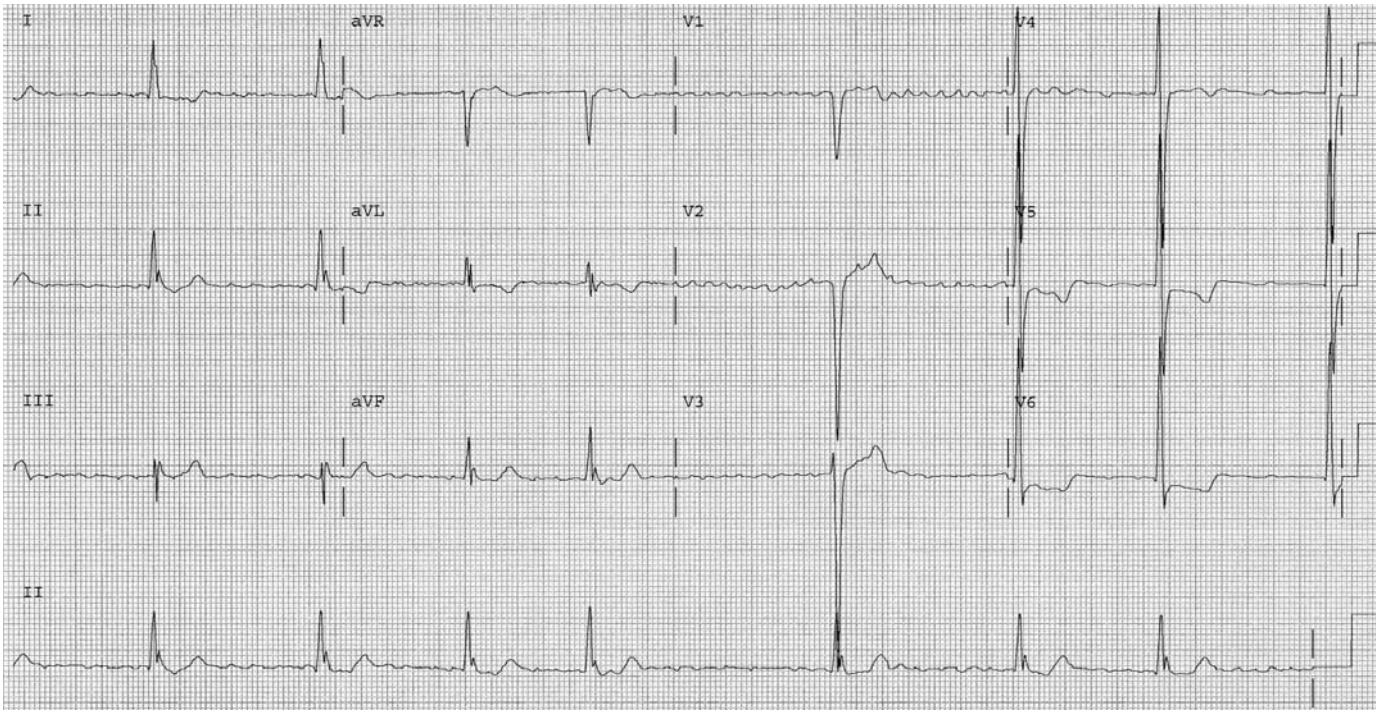


Figure 3. AF with Slow Ventricular Response



Chest radiography may detect enlargement of the cardiac chambers and heart failure. However, it is more valuable for detection of intrinsic pulmonary or pulmonary vasculature pathology, which may lead to right atrial dilatation. Radiography is less important than echocardiography for routine evaluation of patients with AF. Acquire two-dimensional transthoracic echocardiography to determine left atrial and left ventricular size and function, and to assess for the presence of wall motion

Figure 4. Rhythm Strip of AF and a Bundle Branch Block

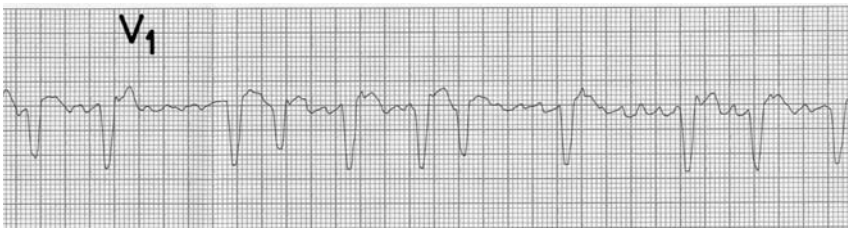


Figure 5. Rhythm Strip of AF: Note the Fibrillation Waves Seen Best in V1

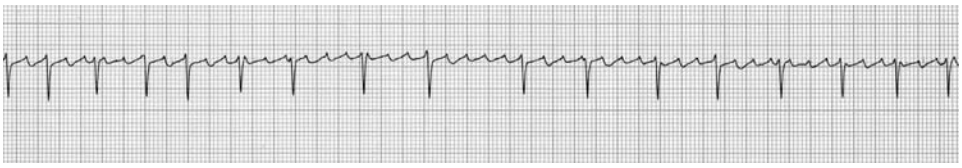
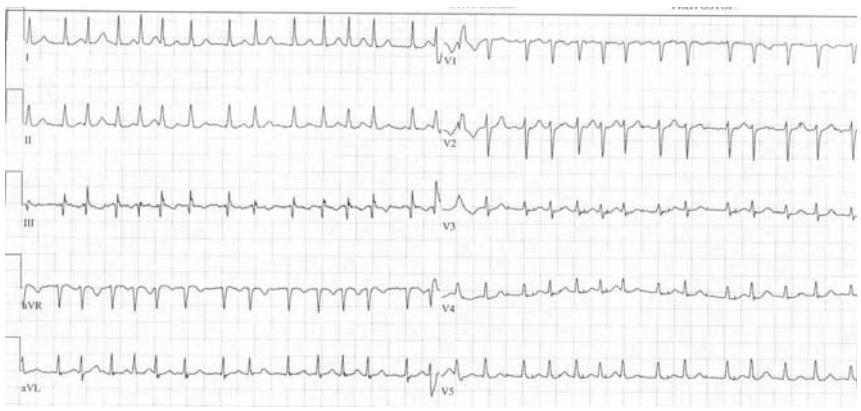


Figure 6. Atrial Fibrillation with Rapid Ventricular Response and Ashman Phenomenon



Aberrant conduction occurs due to a change in the QRS cycle length. When a supraventricular impulse reaches the His-Purkinje system while one of its branches is still in the refractory period, conduction through the bundle branch is slow or delayed; therefore, depolarization takes place through the ventricular myocytes, resulting in a bundle branch block (BBB) pattern. Right BBB is more common than LBBB due to the longer refractory period of the right bundle branch.

abnormalities suggestive of ischemia, valvular disease, or pericardial effusion. Echocardiography can evaluate for thrombus in the atria, but thrombi are seldom detected without transesophageal echocardiography (TEE).[14] Measure thyroid function, serum electrolytes, and cell blood count at least once; these tests are likely to be of limited value.[15] Measure cardiac markers if acute coronary syndrome is suspected.

Management

Emergency department management of AF is based upon differentiation from other dysrhythmias, identification of the underlying etiology, and symptomatology. Asymptomatic atrial fibrillation in hemodynamically stable patients with a rate of 120 beats/minute or less does not require emergent treatment. As mentioned previously, AF related to temporary causes, such as binge ethanol intake (holiday heart syndrome), surgery, pericarditis, myocarditis, pulmonary embolism, hyperthyroidism, or other metabolic disorders, may resolve following successful treatment of the underlying condition. In the case of holiday heart, the treatment is abstinence. This also indirectly decreases excessive cigarette smoking, diuresis-induced electrolyte disturbances, and sleep apnea—all of which are considered arrhythmogenic.

Patients who are unstable from AF associated with rapid ventricular response require sedation and synchronized electrical cardioversion. Once the patient's airway and breathing have been stabilized and an intravenous line established, electrical cardioversion is generally preferred over chemical cardioversion based on its higher success (85-90% vs 50%) and similar complication rate.[16,17] To achieve maximum effectiveness: pads should be placed in an anterior-posterior position; cardioversion should be performed at expiration, when the anterior-posterior distance is minimized; and cardioversion should be synchronized. If available, use biphasic cardioversion at 150 Joules (J). It has been shown to be more effective than monophasic cardioversion of equal strength (86% vs 51%).[18] If biphasic cardioversion is not available, begin monophasic cardioversion at 200 J to minimize repetitive shocks, as only 50% of patients will be successfully cardioverted at 100 J.[19] If the primary cardioversion attempt is unsuccessful, successive shocks should be delivered with 200 J biphasic or 360 J monophasic. Electrical cardioversion may result in malignant ventricular arrhythmias in patients who are digoxin toxic. Hypotension and a bradycardic ventricular response to AF herald digoxin toxicity and mandate electrolyte correction and administration of digoxin specific antibody fragments; electrical cardioversion is contraindicated. However, electrical cardioversion is safe in patients taking digoxin without toxicity, but must be initiated at 10-20 J, with succeeding shocks increasing in 10-20 J increments. Sedation remains the choice of the treating physician. Awareness of a 6% sedation complication rate in AF patients mandates consideration of common complications such as hypotension with propofol or benzodiazepines, tachycardia with ketamine, and myoclonus with etomidate.[17,19]

When cardioversion is indicated and procedural sedation cannot be accomplished, attempt chemical cardioversion. The 2006 AHA guidelines offer class I evidence for use of flecainide,

Figure 7. Rhythm Strip with Ashman's Phenomena. AF with Right Bundle Branch Block (rSR)

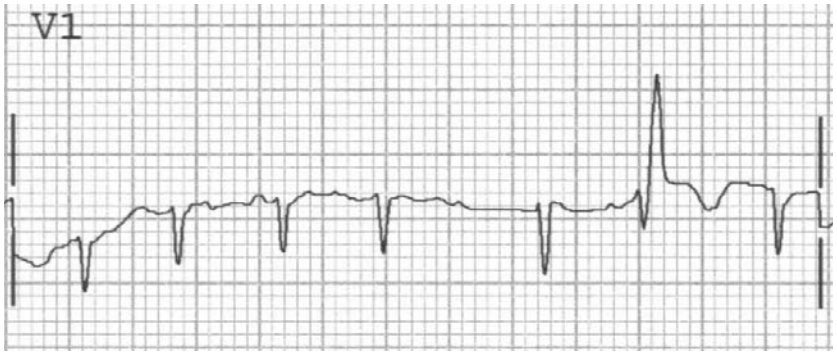
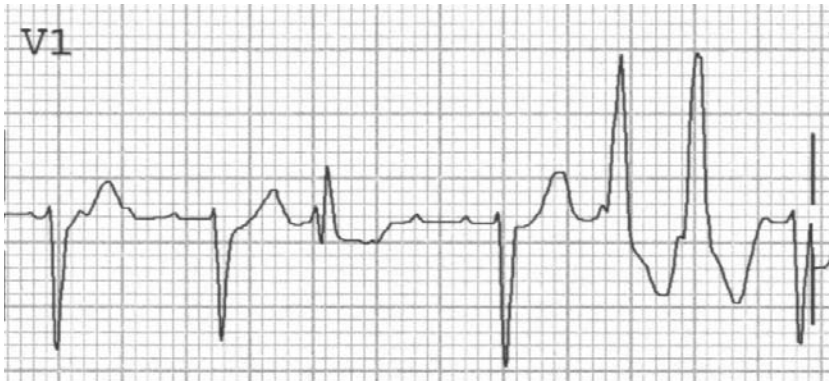


Figure 8. Rhythm Strip with Ashman's Phenomena with Right Bundle Branch Block (rRS changing to rR)



dofetilide, propafenone, and ibutilide and class IIa evidence for use of amiodarone in acute pharmacologic cardioversion of atrial fibrillation.[20] Randomized trials have shown the above medications to be more effective than placebo, converting 30-60% of patients.[21] Use a beta-adrenergic or calcium channel blocker to reduce the ventricular rate to below 120 beats/minute before administering a type IA or IC anti-arrhythmic to minimize the possibility of an increase in ventricular rate caused by a recurrence of atrial flutter with 1:1 conduction through the atrioventricular node. Monitor closely during the pericardioversion period regardless of the mode pharmacologic cardioversion chosen. If an anti-arrhythmic fails to achieve cardioversion, do not switch to an agent from different class in order to avoid complications. Doses and potential adverse effects are contained in Table 1. Type IC agents (flecainide and propafenone) are not available for intravenous use in the United States (although are used routinely in Europe and Australia) and therefore are not indicated in emergent pharmacologic cardioversion of AF.

Emergency department management decisions rely on above-mentioned hemodynamic stability, in addition to the classification and duration of AF. (*See Definition.*) Chronic AF and paroxysmal AF of greater than 48-72 hours duration are associated with atrial thrombus formation and thromboembolic events. Therefore, these patients are best initially managed by rate control, due to an increased risk of thromboembolic complications in the hours to days following conversion to sinus rhythm. Atrial fibrillation duration, underlying valvular disease, and chamber hypertrophy positively correlate with the risk of thromboembolism while negatively correlating with successful cardioversion. Furthermore, a long-term rhythm control strategy does not offer a clear morbidity or mortality benefit when compared with a rate control strategy.[4,5] In patients with atrial fibrillation of less than 48-72 hour duration, either ventricular rate control or cardioversion can be attempted. For further discussion regarding emergency department cardioversion of the hemodynamically stable patient with less than 48-72 hours of atrial fibrillation, see the Additional Aspects section below.

In many cases, intravenous calcium channel blockers (verapamil and diltiazem) are given as first-line agents to control rapid ventricular response. While both beta-adrenergic and calcium channel blockers decrease AV nodal conduction, rate control begins earlier, and the percentage decrease in ventricular rate is higher using diltiazem when compared with metoprolol.[22] Diltiazem is a less negative inotrope than verapamil, resulting in less hypotension and safe, effective use in patients with mild to moderate congestive heart failure. (*See Table 2 for dosing and contraindications.*) A review of case reports and a few small case series suggested that pre-treatment with calcium gluconate may reduce vasodilator mediated hypotension.[23] However, this evidence was refuted by a placebo-controlled trial investigating the efficacy of calcium chloride prior to intravenous diltiazem, which failed to show a significant reduction in hypotension with calcium pre-treatment.[24]

Beta-adrenergic blockers (esmolol and metoprolol) are preferred over calcium channel blockers as first-line agents for rate control in atrial fibrillation due to suspected myocardial ischemia, hyperthyroidism, or other catecholamine excess. Caution should be applied, as beta-adrenergic blockers result in more hypotension and congestive heart failure than calcium channel blockers. While esmolol has a shorter effective half-life allowing for easier titration, it is both more expensive and more complicated to administer than metoprolol. (*See Table 2 for dosing and contraindications.*) Both beta-adrenergic and calcium channel blockers are first-line agents for long-term rate control. Second-line agents for rate control include digoxin and amiodarone. Digoxin cannot be considered a first-line agent due to a three-hour mean time to significant rate reduction, compared to five minutes using diltiazem.[25] While beta-adrenergic blockers, calcium channel blockers, and digoxin are contraindicated in the presence of an accessory pathway (such as WPW) due to the risk of ensuing ventricular fibrillation, amiodarone can be safely utilized. Amiodarone's expense, negative inotropic effect, and reduced effectiveness for rate control when compared to diltiazem or magnesium result in its designation as a second-line rate control agent.[26,27] Amiodarone may have a primary role for use in the critically ill population with AF. Two retrospective reviews of intensive care unit patients with hemodynamically compromising

Figure 9. WPW with Rapid Ventricular Response

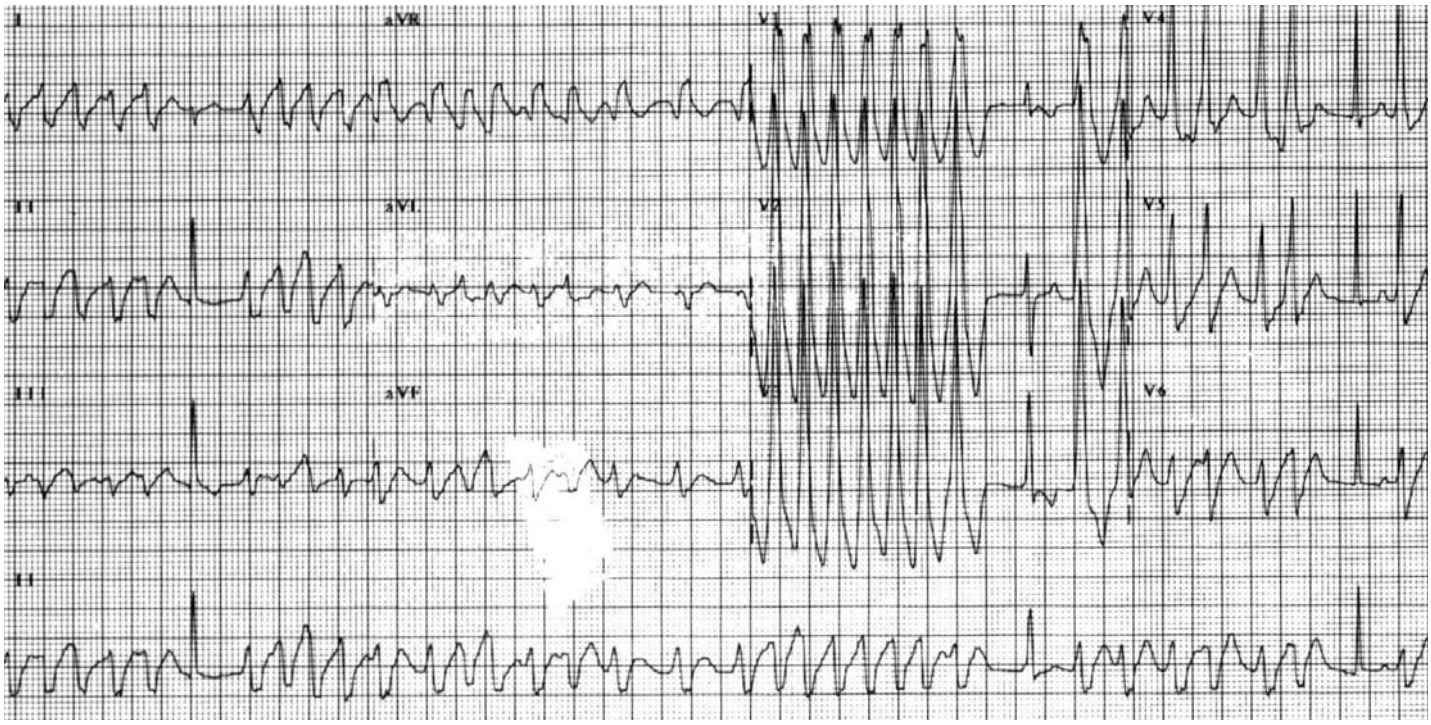
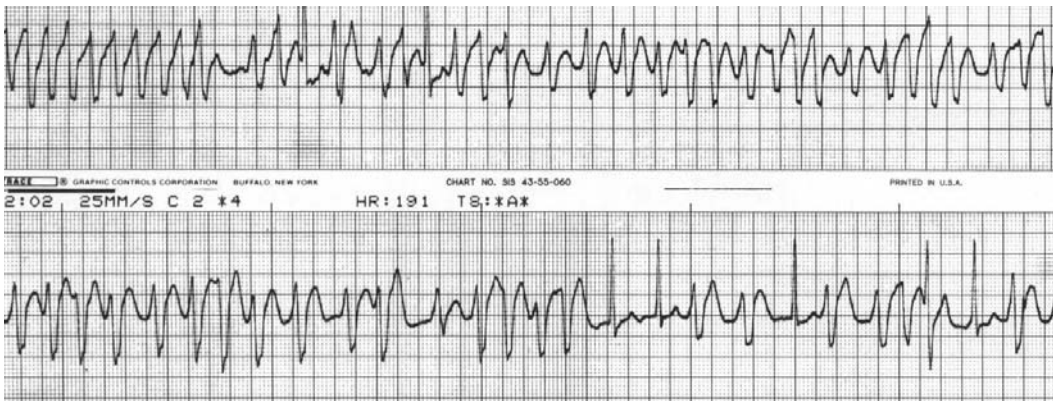


Figure 10. Rhythm Strip of a Patient with WPW and a Chaotic Rapid Ventricular Response



atrial tachyarrhythmias show amiodarone results in a significant decrease in heart rate while either maintaining or improving systolic blood pressure (SBP); whereas diltiazem, esmolol, and digoxin were found to have no effect on heart rate and result in significant decrease in SBP.[28,29]

Intravenous magnesium sulfate provides a second-line or adjunctive therapy for controlling ventricular response rate with atrial fibrillation. It slows conduction through the atrioventricular node and has been shown to have rate control success rates equivalent to those of amiodarone and digoxin in varying doses.[27] Magnesium is a successful adjunct to standard rate reduction therapy, two times more likely to achieve a heart rate of less than 100 beats/minute when compared with placebo.[30]

All patients presenting to the ED with AF without a contraindication to anticoagulation should be therapeutically anticoagulated to reduce the risk of thromboembolic events. The AHA recommends chronic anticoagulation with warfarin and a target INR of 2-3 in patients with chronic or paroxysmal AF, citing an annual risk of cerebrovascular accident of 4.5%.[20] Recommendations regarding anticoagulation therapy for patients with acute AF and those in the pericardioversion period are included below in the Additional Aspects section.

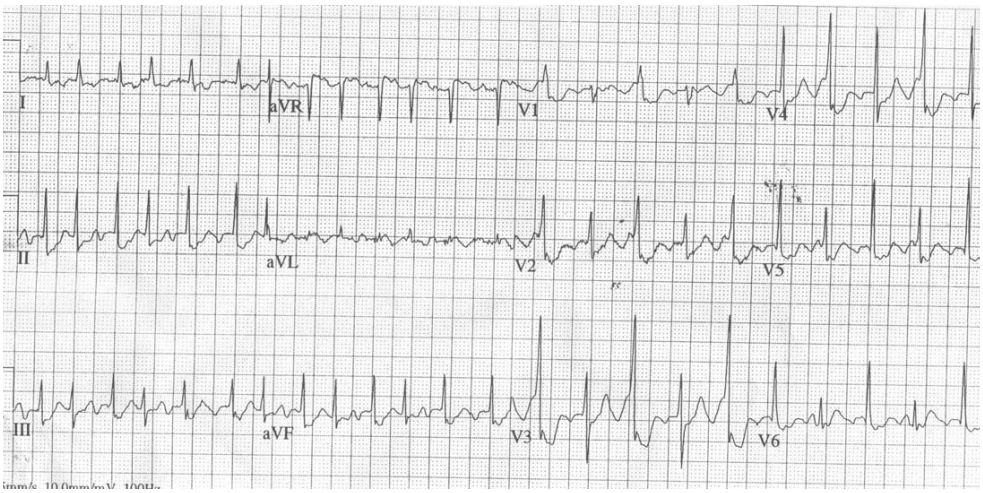
In the acute period, hemodynamically stable patients with a suspected or confirmed accessory pathway should be rate controlled with amiodarone. These patients should be urgently referred to a cardiologist for consideration of accessory pathway ablation.

Additional Aspects

Two main controversies currently exist surrounding the acute management of patients presenting with atrial fibrillation in the emergency department. Primarily, should any population of hemodynamically stable patients in atrial fibrillation undergo cardioversion in the ED? Secondly, what role does anticoagulation play in AF patients in the ED?

While there exists no controversy regarding cardioversion of patients presenting to the ED with rapid AF associated with hemodynamic or cardiopulmonary instability, there are multiple

Figure 11. Atrial Fibrillation with Rapid Ventricular Response in the Presence of Wolff-Parkinson-White



Note the intermittent wide QRS complex with delta waves, most easily recognized in V3 and V4.

Table 1. Acute Pharmacologic Conversion of Atrial Fibrillation

Flecainide 1.5-3.0 mg/kg IV over 10-20 minutes
Cautions: hypotension, rapidly conducting atrial flutter

Ibutilide 1 mg IV over 10 minutes, may repeat when necessary (conversion usually occurs within 20 minutes if successful)
Cautions: QT prolongation, torsade de pointes

Amiodarone 5-7 mg/kg IV over 30-60 minutes, then 1.2-1.8 g/day continuous IV or divided in oral doses until 10 g total
Cautions: hypotension, bradycardia, QT prolongation, torsade de pointes, nausea/vomiting, constipation, phlebitis

If needed for ventricular rates exceeding 120 beats/min:
Verapamil 5-10 mg IV or diltiazem 20-25 mg IV can be given before flecainide to reduce the ventricular rate to less than 120 beats/minute.

other patient populations for which controversy exists: AF with a duration of less than 48 hours, asymptomatic AF, AF with an associated exacerbation of congestive heart failure (CHF), and AF in the presence of anticoagulation contraindications. The American Heart Association (AHA) guidelines recommend cardioversion of AF with a duration of less than 48 hours, citing a low risk of thromboembolism.[20] This recommendation raises several questions: Is patient perception of the duration of atrial fibrillation accurate? What is the safety profile and success rate of ED cardioversion? What is the actual rate of thromboembolism after ED cardioversion?

Thus far, studies indicate that patient perception of AF is inaccurate. Interrogating implantable loop recorders in patients with paroxysmal AF revealed that only 68% of patient activated events were appropriate.[31] Perhaps even more concerning, as many as 40% of patients diagnosed with AF are asymptomatic.[32] Furthermore, greater than 50% of those with implanted rhythm control devices have revealed episodes of AF; 58% of these were asymptomatic.[32] These numbers make it difficult to rely on patient history as a sign of AF duration.

Advocates for ED cardioversion in acute or new onset AF of less than 48 hours duration cite safety, success rate of cardioversion, and patient satisfaction as their arguments. Maisel and colleagues studied 417 consecutive patients hospitalized with AF who underwent 597 attempted chemical cardioversion trials resulting in a 30% first-attempt success rate and a 60% overall success rate.[16] Complications occurred 13% of the time, most often in the 24 hours immediately following cardioversion.[16] Bradyarrhythmias constituted greater than 60% of the complications, followed by QT prolongation requiring drug discontinuation, and ventricular arrhythmias necessitating inpatient observation for 24-48 hours following successful chemical cardioversion.[16] While the 247 electrical cardioversions in Maisel's study achieved an 84% success rate, there were plagued complications in 11% of the cases; however, the authors did not remark with regard to the type or severity.[16] Patients with prior myocardial infarction were at a slightly increased risk for complications, but multivariate analysis was unable to identify any further risk factors.[16] Similarly, Stiell and colleagues reviewed a cohort of 289 acute symptomatic AF patients presenting to the ED.[17] Those who were unstable or had other conditions mandating admission were eliminated from the study cohort. Stiell's complication rate was 6%, of which 95% were regarded as minor; his success rate was similar to Maisel's.[17] One patient had a complication caused by a rate control medication, which necessitated hospital admission. Further clarification regarding complications was provided in a review of 388 patients in acute AF who underwent ED cardioversion, 30% of which had undergone at least one previously unsuccessful chemical cardioversion attempt.[19] Six percent suffered sedation complications, while 2% suffered complications unrelated to sedation including three burns, two patients who sustained ventricular tachycardia, and one patient with unstable bradycardia—suggesting a small, but not insignificant complication rate.[19] Two small prospective studies including only patients treated with electrical cardioversion confirm the success and complication rates established by larger retrospective reviews and offer follow-up data indicating patient satisfaction ratings of 97-100%.[33,34]

Despite success rates of electrical cardioversion approaching

Table 2. Rate Control Agents for Atrial Fibrillation with Rapid Ventricular Response

AGENT	DOSE	CONTRAINDICATIONS/CAUTIONS
Amiodarone	150-300 mg IV bolus, then 1 mg/min IV infusion	Hypotension, CHF
Digoxin	0.5 mg IV bolus, then 0.25 mg PO at 4 and 8 hours	Accessory pathway
Diltiazem	5-10 mg increment IV bolus to a maximum of 50 mg, then 5-20 mg/hr IV infusion	Hypotension, use of other AV blockers, accessory pathway
Esmolol	500 mcg/kg IV load, then 50-200 mcg/kg/minute IV infusion	Hypotension, CHF, asthma, COPD, accessory pathway
Magnesium	2.5 g IV over 20 minutes, then 2.5 g IV over 2 hours	Renal failure; rapid infusion may lead to respiratory failure and hypotension
Metoprolol	5 mg IV every 5 minutes to a maximum of 15 mg, then 50 mg PO	Hypotension, CHF, use of other AV blockers, asthma, COPD, accessory pathway
Verapamil	5 mg increments IV over 2 minutes to a maximum of 20 mg, then 5-24 mg/hr IV infusion	Hypotension, CHF, use of other AV blockers, accessory pathway

90% and early reports of patient satisfaction with ED cardioversion of AF, neither complications related or unrelated to sedation nor the risk of thromboembolism can be overlooked. Studies reveal that the risk of thromboembolism following cardioversion of AF without simultaneous anticoagulation ranges from 0-7%, with a mean of 1.5%.[35] Further downsides to ED cardioversion of AF include the argument that 66% will spontaneously convert to normal sinus rhythm within the first 24 hours; this number increases to 80% by 48 hours.[36] In addition, the atrial "kick" accounting for a 10-15% boost in cardiac output is shown not to return for days to weeks following cardioversion due to atrial myocardial stunning.[37] Finally, many critics cite increased cost associated with the risk of ED cardioversion; this may, however, be recouped if admission is avoided.

While further prospective study is needed to determine the role of cardioversion in stable AF with a presumed duration of less than 48 hours, other controversial patient populations are more easily addressed. In those with asymptomatic AF, ED cardioversion is unlikely to play a role as the 6% risk of sedation complications and a 1% risk each of arrhythmia, burn, and thromboembolism likely outweigh the benefits of an asymptomatic state. In those suffering from AF accompanied by a CHF exacerbation, rate control with diltiazem has been studied and found to be effective.[38] Furthermore, patients with left ventricular dysfunction are at much higher risk for thromboembolism and are less likely to be successfully cardioverted. Citing the stable hemodynamic profile of diltiazem and elevated risk profile of cardioversion with left ventricular dysfunction, this is another controversial population in which cardioversion is likely to play only a rare role. Finally, patients with AF in whom anticoagulation is contraindicated are considered by some to be candidates for ED cardioversion. A 4.5% annual risk of cerebrovascular accident in AF patients without anticoagulation seems to argue for rhythm control as a long-term management strategy in this patient population.[39] However, the RACE trial revealed equal thromboembolic risks in the rate and rhythm control groups. If this equal risk is not enough to argue against the need for ED cardioversion in this population, then the reason anticoagulation is contraindicated should be considered. If acute trauma is the contraindication, the patient is more likely than not going to be admitted, allowing for an inpatient cardioversion. If an elevated fall risk is the contraindication, this risk can be minimized by admission to a controlled setting, allowing for short-term anticoagulation and inpatient cardioversion.

While the need for long-term anticoagulation in patients with paroxysmal or chronic AF is well-accepted due to an annual risk of cerebrovascular accident approaching 5% in patients without anticoagulation, what concerns emergency physicians is the role of anticoagulation in patients with new onset AF or those patients for whom non-emergent ED cardioversion may be an option. To discern the role of anticoagulation in this population, two myths must first be debunked. First, there is a real thromboembolic risk associated with cardioversion of acute AF. It was believed that there is no risk of left atrial clot formation within a 48-hour time period. Disproving this belief, a TEE performed on 143 patients with AF of less than 48 hours duration found left atrial clot in 20 patients (14%).[40] While significantly lower than the 27% incidence of left atrial clot found in patients with chronic AF, the incidence of clot suggests a need for anticoagulation or TEE prior to cardioversion in patients with AF of less than 48 hour duration.[40] Secondly, there is thromboembolic risk associated with cardioversion preceded by a negative TEE: a 6% risk of thromboembolism following cardioversion preceded by a negative TEE was demonstrated.[41] This finding indicates either that TEE is an imperfect screen for left atrial clot or that clot may develop post-cardioversion. Furthermore, 40% of patients

showed increased spontaneous echo contrast in the left atrium within 10 seconds of cardioversion, making the possibility of atrial stunning resulting in the development left atrial clot post-cardioversion more likely and suggesting the need for anticoagulation with cardioversion.[41] The realization that anticoagulation is required in the pericardioversion period to prevent thromboembolism in patients with acute AF and those with negative TEE begs the question as to the optimal duration of anticoagulation required in those who remain in normal sinus rhythm. A lack of data leaves this question somewhat unanswerable. However, a study of 17 patients with thromboembolism occurring two hours to seven days following TEE-guided cardioversion without accompanying anticoagulation revealed 80% with increased left atrial echo contrast or clot, suggesting need for at least seven days of anticoagulation following TEE-guided cardioversion.[42] The AHA/ACC recommendations include pericardioversion heparin followed by post-cardioversion warfarin for four weeks with a target INR of 2-3.[20]

Disposition

By convention, patients with new onset AF are admitted to the hospital for treatment and monitoring for serious cardiovascular complications. This practice stems from studies performed in the 1930s demonstrating an association of new-onset AF with rheumatic heart disease, coronary artery disease, and thyroid disease. Admissions for AF have increased 60% in the last two decades, including a 34% increase between 1996 and 2001.[43,44]

Patients presenting with congestive heart failure, myocardial infarction, typical ischemic chest pain, hypotension, or acute diagnoses in addition to AF (e.g., renal failure, pulmonary embolism, hyperthyroidism) have a higher morbidity and must be admitted.[45] Laboratory findings requiring admission include: hematocrit less than 30%, hyponatremia, and renal insufficiency.[46] Admission should be considered for the following groups: the elderly, those with limited access to follow-up care, and patients with underlying cardiac disease. Finally, acute pharmacological anti-arrhythmic therapy mandates admission for extended cardiac monitoring and observation for pro-dysrhythmic complications.

Often, there is underlying concern that new-onset AF may be an atypical presentation of acute coronary syndrome (ACS). The incidence of ACS in patients with new-onset AF ranges from 1% to 11%; most present in a typical fashion, with the exception of elderly and diabetics. Atrial fibrillation is rarely the only manifestation of ACS.[47] Furthermore, AF with rapid ventricular response may trigger angina. Hence, non-elderly patients without diabetes who present without typical ACS symptoms are considered to be low risk and may be considered for outpatient management. Several studies have concluded that cardioversion and ED discharge of patients presenting with acute AF is safe and effective, citing ED discharge rates of 86-100% accompanied by 10% recidivism rates at seven days.[17,19,33,34]

Recently, the Ottawa Aggressive Protocol was proposed as safe and effective for ED treatment of acute AF.[48] The protocol consists of a procainamide intravenous infusion of 1 gram over an hour, followed by electrical cardioversion in the case of failed pharmacologic conversion, and discharge from the ED. Stiell and colleagues studied 660 patient visits over a five-year period during which 97% of patients were discharged from the ED with 90% in sinus rhythm. Prospective study of the Ottawa protocol has the potential to significantly decrease hospital admissions for acute AF.

Note that all patient discharges from the ED should be done in concert with a cardiologist to insure adequate follow-up.

Summary

For the emergency physician, the most critical aspect in the management of AF is to determine hemodynamic stability. If the patient is deemed to be unstable, one should proceed with sedation and synchronized electrical cardioversion. Consideration should be given as to whether the AF is secondary to another medical problem and whether it is a new or chronic issue.

Taking into consideration the AFFIRM and RACE trial results, choosing conservatively and opting for ED rate control seems to maximize patient safety. Diltiazem is the ideal rate control agent in nearly all patients, excepting those whose AF is the result of suspected ACS or hyperthyroidism, in which beta-adrenergic blockers are preferred. Use amiodarone if AF is complicated by an accessory bypass tract.

Published data on the safety of ED cardioversion in low risk patients with new onset AF is mounting. If choosing cardioversion, electrical cardioversion is more successful and accompanied by fewer complications than pharmacologic cardioversion. Risk of thromboembolism as well as non-thrombotic complications and a 24-hour spontaneous conversion rate of 66% must be considered when choosing rhythm control.

In the introductory cases, case 1 involves a 42-year-old patient in AF without need for rate control. AF appears to be recent onset and the patient is symptomatic. The AHA guidelines recommend cardioversion in this patient; the ideal choice is synchronized electrical cardioversion with 150 J using biphasic or 200 J using monophasic defibrillation. Heparin should be started prior to cardioversion and followed by one month of warfarin with a target INR of 2-3. Case 2 involves a 65-year-old female with AF and rapid ventricular response leading to a congestive heart failure. While she may be managed either with electrical cardioversion or rate control, rate control with diltiazem is the ideal choice. Her age, relative hypotension, and comorbid conditions make the use of sedation a major risk. In addition, electrical cardioversion has not demonstrated benefit in patients with AF accompanied by CHF. However, diltiazem has been shown to be safe and effective in mild to moderate congestive heart failure, as it has less negative inotropic effects than other calcium channel blockers and beta-adrenergic blockers. Finally, case 3 involves a 29-year-old male with AF, rapid ventricular response, and an accessory bypass tract. As he is asymptomatic with stable vital signs, rate control with amiodarone is the prudent choice. To achieve definitive care, he requires an urgent electrophysiology evaluation, and likely a bypass tract ablation.

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Physician CME Questions

61. Which of the following is true with regard to acute atrial fibrillation?
    A. It is only used to describe episodes of new-onset atrial fibrillation.
    B. It persists less than 72 hours.
    C. It can describe new-onset or recurrent episodes of atrial fibrillation.
    D. It is not accompanied by atrial clot formation.

62. The RACE trial concludes which of the following?
    A. Rate and rhythm control groups have equal mortality.
    B. Rate control results in fewer episodes of heart failure.
    C. Rhythm control requires less anticoagulation due to lower thromboembolic risk.
    D. Rhythm control results in more serious adverse drug effects.

63. In which case is treatment of the underlying disorder unlikely to eliminate atrial fibrillation?
    A. Binge ethanol intake
    B. Acute coronary syndrome
    C. Hyperthyroidism
    D. Hypertrophic cardiomyopathy

64. Which of the following is correct with regard to symptom manifestations of atrial fibrillation?
    A. Palpitations are more common than weakness and dizziness.
    B. Gradual symptom onset suggests an atrial arrhythmia.
    C. Syncope is more likely to accompany atrial than ventricular arrhythmias.
    D. Approximately 21% of patients are asymptomatic.

65. Wide-complex atrial fibrillation suggest the presence of which of the following conditions?
    A. An accessory bypass tract
    B. Hyperthyroidism
    C. Left ventricular hypertrophy
    D. Pulmonary embolism

66. When electrically cardioverting atrial fibrillation:
    A. place pads at the apical and sternal positions.
    B. synchronized cardioversion is not advised.
    C. start at 200 J if a monophasic defibrillator is used.
    D. avoid cardioversion in patients taking digoxin.

67. In most cases of atrial fibrillation with rapid ventricular response, the first-line rate control agent is:
    A. metoprolol.
    B. amiodarone.
    C. diltiazem.
    D. digoxin.

68. In cases of atrial fibrillation with rapid ventricular response and a wide-complex QRS, the safest rate control agent is:
    A. metoprolol.
    B. amiodarone.
    C. diltiazem.
    D. digoxin.

69. Which of the following strategies minimizes risk of thromboembolism in emergency department cardioversion of atrial fibrillation?
    A. Acute atrial fibrillation, TEE-guided, and pericardioversion heparin
    B. Persistent atrial fibrillation and therapeutic warfarin with an INR of 2-3
    C. Acute atrial fibrillation and TEE-guided
    D. Acute atrial fibrillation, TEE-guided, and pericardioversion heparin followed by warfarin with a target INR of 2-3

70. Which of the following is true with regard to emergency department cardioversion?
    A. Pharmacologic cardioversion is successful 75% of the time.
    B. Electrical cardioversion success rates approach 90%.
    C. Patients with acute atrial fibrillation are not at risk for thromboembolism.
    D. More complications result from electrical than from pharmacologic cardioversion.

CME Answer Key

61. C; 62. A; 63. D; 64. D; 65. A; 66. C; 67. C; 68. B; 69. D; 70. B

Emergency Medicine Reports®
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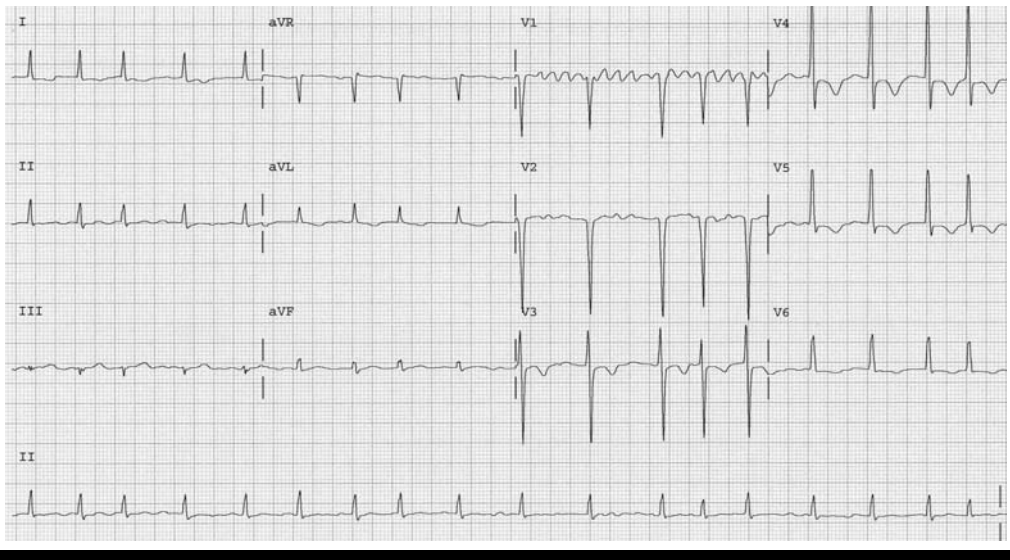
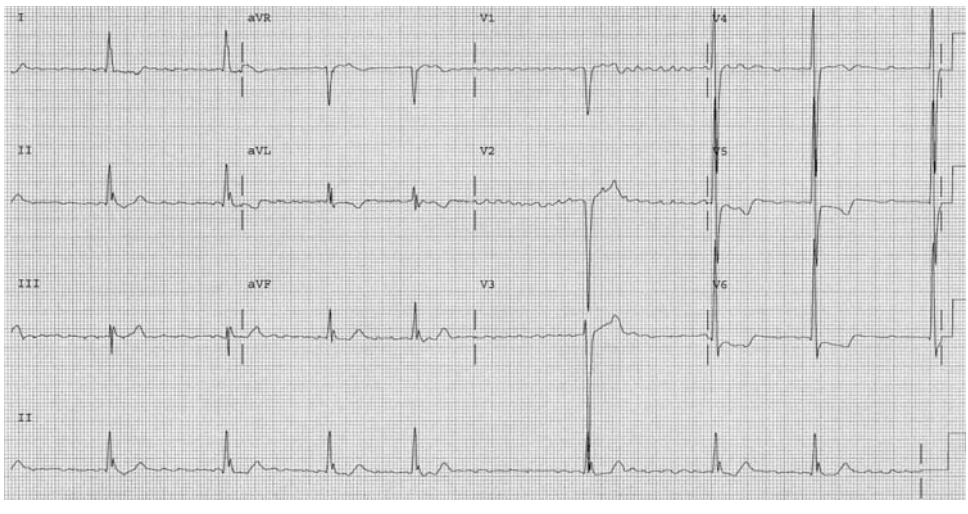
Evaluating and Treating Atrial Fibrillation

AF: Note the Fibrillation Waves in V1



AF with Slow Ventricular Response



Rate Control Agents for Atrial Fibrillation with Rapid Ventricular Response

AGENT	DOSE	CONTRAINDICATIONS/CAUTIONS
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Digoxin	0.5 mg IV bolus, then 0.25 mg PO at 4 and 8 hours	Accessory pathway
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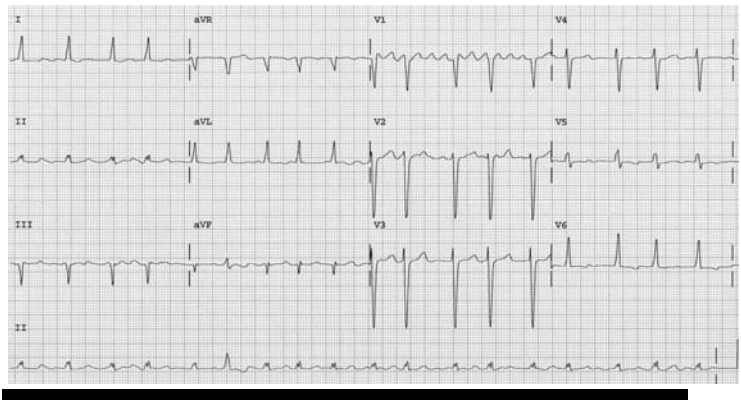
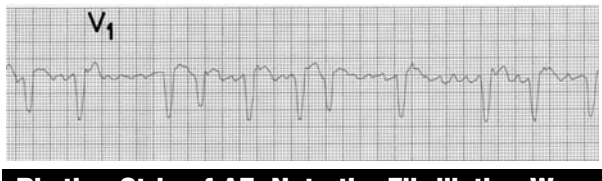
Magnesium	2.5 g IV over 20 minutes, then 2.5 g IV over 2 hours	Renal failure; rapid infusion may lead to respiratory failure and hypotension
Metoprolol	5 mg IV every 5 minutes to a maximum of 15 mg, then 50 mg PO	Hypotension, CHF, use of other AV blockers, asthma, COPD, accessory pathway
Verapamil	5 mg increments IV over 2 minutes to a maximum of 20 mg, then 5-24 mg/hr IV infusion	Hypotension, CHF, use of other AV blockers, accessory pathway

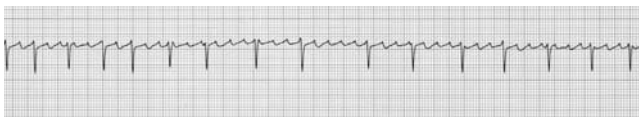
AF in a 12 Lead



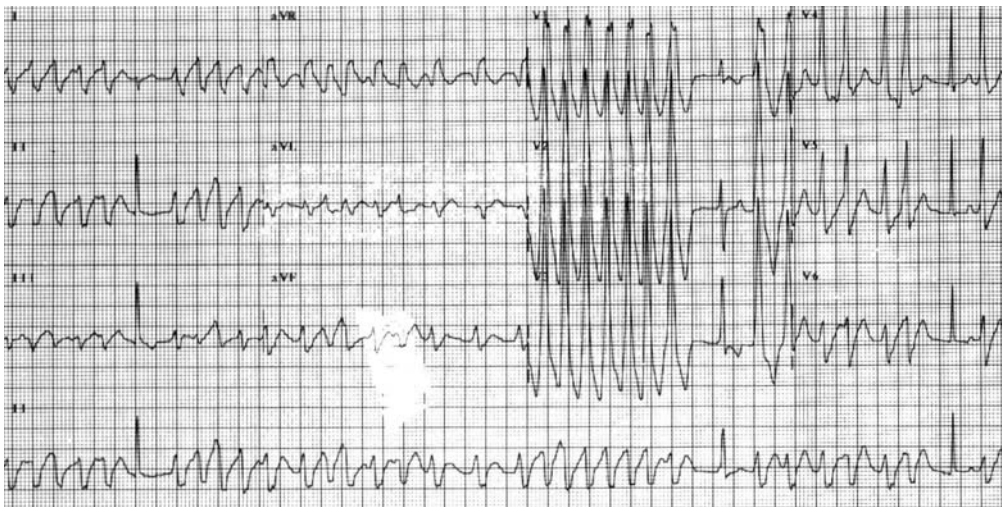
Rhythm Strip of AF and a Bundle Branch Block



Rhythm Strip of AF: Note the Fibrillation Waves Seen Best in V1



WPW with Rapid Ventricular Response



Acute Pharmacologic Conversion of Atrial Fibrillation

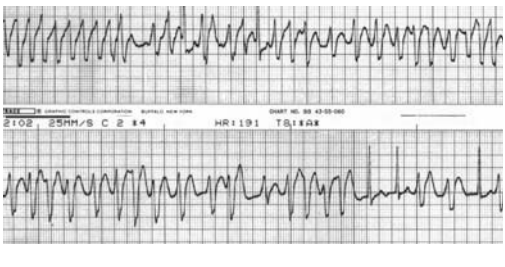
Flecainide 1.5-3.0 mg/kg IV over 10-20 minutes
Cautions: hypotension, rapidly conducting atrial flutter

Ibutilide 1 mg IV over 10 minutes, may repeat when necessary
(conversion usually occurs within 20 minutes if successful)
Cautions: QT prolongation, torsade de pointes

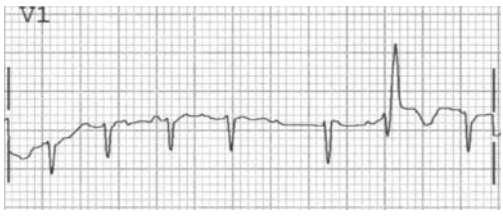
Amiodarone 5-7 mg/kg IV over 30-60 minutes,
then 1.2-1.8 g/day continuous IV or divided in oral doses until 10 g total
Cautions: hypotension, bradycardia, QT prolongation, torsade de pointes, nausea/vomiting, constipation, phlebitis

If needed for ventricular rates exceeding 120 beats/min:
Verapamil 5-10 mg IV or diltiazem 20-25 mg IV can be given before flecainide to reduce the ventricular rate to less than 120 beats/minute.

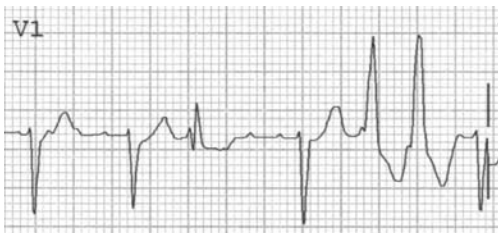
Rhythm Strip of a Patient with WPW and a Chaotic Rapid Ventricular Response



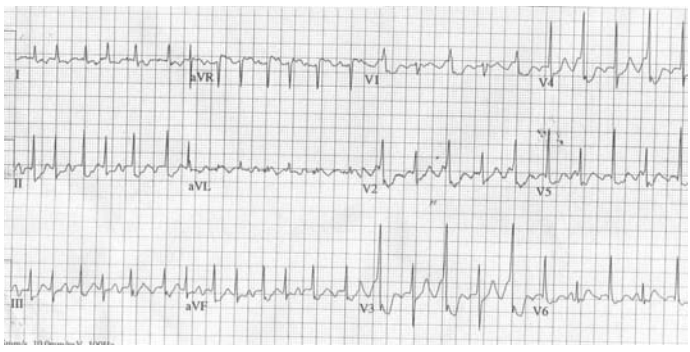
Rhythm Strip with Ashman Phenomenon. AF with Right Bundle Branch Block (rSR)



Rhythm Strip with Ashman Phenomenon with Right Bundle Branch Block (rRS changing to rR)

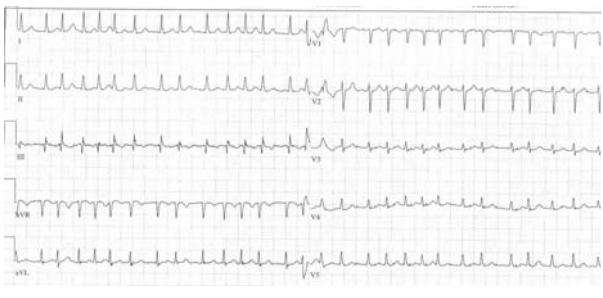


Atrial Fibrillation with Rapid Ventricular Response in the Presence of Wolff-Parkinson-White



Note the intermittent wide QRS complex with delta waves, most easily recognized in V3 and V4.

Atrial Fibrillation with Rapid Ventricular Response and Ashman Phenomenon



Aberrant conduction occurs due to a change in the QRS cycle length. When a supraventricular impulse reaches the His-Purkinje system while one of its branches is still in the refractory period, conduction through the bundle branch is slow or delayed; therefore, depolarization takes place through the ventricular myocytes, resulting in a bundle branch block (BBB) pattern. Right BBB is more common than LBBB due to the longer refractory period of the right bundle branch.

Supplement to *Emergency Medicine Reports*, September 17, 2007: "Evaluating and Treating Atrial Fibrillation in the Emergency Department." *Authors:* **Jennifer Casaletto, MD, FACEP**, Assistant Professor, Associate Residency Director, Department of Emergency Medicine, Maricopa Medical Center, Phoenix, AZ; and **Paul Berman, MD**, Emergency Medicine Resident, Department of Emergency Medicine, Maricopa Medical Center, Phoenix, AZ.