

## Risk Management Pitfalls For Atrial Fibrillation

- 1. “The patient denied any chest pain, so I sent her home after she spontaneously cardioverted.”**  
Unfortunately, the patient was a 62-year-old female with diabetes who had a prior history of myocardial infarction. Had you compared her prior ECG, you would have noted new ischemic changes. Although she spontaneously converted, ECG changes and significant cardiac risk factors should have prompted an admission to further evaluate for ischemia.
- 2. “The ibutilide worked great. The patient felt much better and wanted to go home immediately.”** While ibutilide works to convert AF/AFL approximately 40% to 50% of the time, it has significant risks—most notably an 8% risk of torsades de pointes and other ventricular tachyarrhythmias, which may be mitigated by pretreatment with IV magnesium sulfate. Use of this drug requires a 4-hour period of monitoring after administration.
- 3. “The QRS complexes looked a little bizarre, but I figured she had an underlying bundle branch block. I didn’t think a 20-mg diltiazem bolus would cause her to go into cardiac arrest.”** Wide, bizarre QRS complexes with very rapid ventricular rates up to 300 beats per minute should lead you to suspect preexcitation such as Wolff-Parkinson-White syndrome, as should prior ECGs with delta waves, history of an accessory pathway, or very young patients with new-onset AF. Urgent electrical cardioversion should be performed for patients who are hemodynamically unstable with AF/AFL involving conduction over an accessory pathway, while IV procainamide, ibutilide, or amiodarone may be considered for hemodynamically stable patients.
- 4. “She was hypotensive, so I gave calcium gluconate before giving the commonly quoted starting dose of diltiazem: a 20-mg bolus. It is unfortunate that she became profoundly hypotensive and went into cardiac arrest, but I did nothing wrong.”** Pretreatment with calcium may potentially help blunt the hypotensive effects of diltiazem; however, had you started with a lower dose and titrated it slowly, you may have been able to prevent the hypotension and cardiac arrest. You can also consider using vasopressors, cardioversion, or an amiodarone drip to minimize hypotension and prevent decompensation.
- 5. “When the diltiazem didn’t give a good response, I decided to try metoprolol. I believe her complete heart block was from the acute coronary syndrome she was having, not what I did.”** Combining IV beta blockers and calcium channel blockers can result in hypotension and can precipitate dysrhythmia and complete atrioventricular nodal blockade. It is safe to give 1 of these 2 classes of drugs intravenously—cautiously—if the patient is on an oral version of the other class, but giving both intravenously in a short time period could potentially lead to decompensation.
- 6. “I thought the patient might be having acute coronary syndrome, so I used a beta blocker for its beneficial effects. I was not expecting the patient to decompensate around that same time.”** While there are advantages to using a beta blocker in new-onset AF in the setting of acute coronary syndromes or thyrotoxicosis, it is important to remember any contraindications to specific drug classes. Had you asked the patient about a history of asthma and her recent increased use of home nebulizers you might have considered a short-acting beta blocker such as esmolol or a calcium channel blocker instead.
- 7. “Sure, her AF was 1 week old, but I obtained a transesophageal echocardiogram that showed no left atrial clot, so I cardioverted the patient and sent her home. She was just one of those unfortunate people who had a thromboembolic event.”** Although there is some suggestion in the literature that the method you used might be reasonable, the data suggest that anticoagulation would be required even with a negative transesophageal echocardiogram in this situation. If someone has been in AF for more than 48 hours, transesophageal echocardiogram may not show a clot, but there may still be as high as a 2% incidence of thromboembolism after conversion due to atrial stunning and dysfunction after cardioversion.

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8. **“She didn’t look bug-eyed to me.”** In the elderly, thyrotoxicosis can present very atypically, without the common findings that usually occur in younger patients. A thyroidstimulating hormone screening is a reasonable test in patients > 55 years of age with new-onset AF.
9. **“The patient was hypotensive, so I tried cardioversion. I couldn’t get him to cardiovert after multiple attempts with 200 J, so I gave IV metoprolol to slow the heart rate down in hopes that the decreased rate would improve ventricular filling and increase his blood pressure. I couldn’t believe that it worsened his blood pressure and he ended up going into cardiac arrest.”** Failed cardioversion may occur in patients with long-standing AF/AFL, and pretreatment with an antiarrhythmic such as amiodarone may decrease the defibrillation threshold and improve success of cardioversion. Atrioventricular nodal blocking agents may slow the rate down, but this does not increase the “atrial kick” contribution to ventricular filling; thus, atrioventricular nodal blocking agents will likely only exacerbate the hypotension.
10. **“She was altered and couldn’t tell me how long she had been in AF. I didn’t want to cardiovert her and cause a stroke, so I gave diltiazem.”** The patient was showing evidence of poor perfusion with altered mental status, cool, clammy skin, and hypotension, and she needed immediate electrical cardioversion. Heparin should be started as soon as possible after cardioversion unless there is a significant contraindication.

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