WHY WE DO IT

Rhythm vs. Rate Control for New Onset Atrial Fibrillation

Atrial Fibrillation is the most common arrhythmia in humans, with increasing incidence in the aging population[1]. This abnormal arrhythmia is due to small areas of myocardium continuously discharging and contracting. Instead of uniform contraction of the atria, there is a quivering motion which leads to less ventricular filling and overall, less cardiac output. Atrial fibrillation is usually associated with pulmonary causes, ischemia/infarction, rheumatic heart disease, alcohol/anemia, thyrotoxicosis, electrolyte abnormalities, sepsis (PIRATES). In addition, atrial fibrillation leads to an increase in mortality, myocardial infarction, stroke, and heart failure.

Classically, patients with atrial fibrillation typically present asymptomatic or complaining of shortness of breath or palpitations. Diagnosis is made on EKG which shows absence of discernible p waves and an irregularly irregular ventricular rhythm (Figure 1)[2].

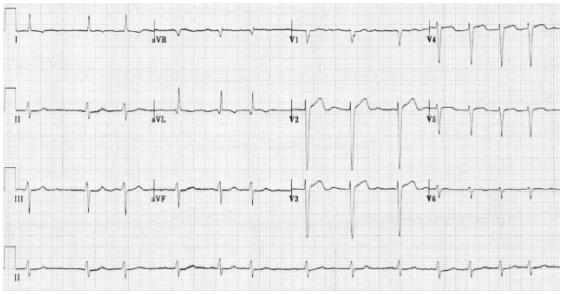

Figure 1. EKG showing Atrial Fibrillation

Upon arrival in the emergency department, multiple clinical conditions as well as patient status all play an integral role in the management of atrial fibrillation vs. atrial fibrillation rapid ventricular response (RVR). Patients' medical history, especially any role of alcohol, drug use, and history of congestive heart failure are all determinants of treatment. In addition, patients presenting in rapid ventricular response (RVR) or stable heart rate also affects treatment. The last condition to ascertain is if the patient has a history of atrial fibrillation, and if not, how long their symptoms have been present.

Now that we have reviewed a general overview of atrial fibrillation, we will now move on to the major discussion of rhythm vs rate control for new onset atrial fibrillation. We will primarily focus on hemodynamically stable presentations however we will briefly mention on unstable patients. For this subset of patients, treatment is performed with synchronized cardioversion. For our stable patients with RVR, management can begin with either Diltiazem 0.25 mg/kg bolus followed by a titratable drip or Metoprolol 5mg pushes every 5 minutes for a total of 3 times. However, there must be caution in patients with heart failure as Diltiazem is contraindicated. In a

comparison trial between these two classes of medications, Diltiazem was found to outperform Metoprolol in reduction of heart rate at 5, 10, and 15 minutes while both agents lowered blood pressure by 15-20 mm Hg at 15 minutes[3]. For patients that are not responding to either intervention above or have contraindications, IV digoxin 0.25-0.5 mg can be utilized. The final subset of patients include those in critical illness, cardiogenic shock, decompensated heart failure, IV amiodarone at 150 mg bolus followed by a drip is a more suitable option.

7.2.1. Acute Rate Control

Recommendations for Acute Rate Control
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	B-R	1. In patients with AF with rapid ventricular response who are hemodynamically stable, beta blockers or nondihydropyridine calcium channel blockers (verapamil, diltiazem; provided that EF >40%) are recommended for acute rate control (Figure 17).[1–4]
2a	B-R	2. In patients with AF with rapid ventricular response in whom beta blockers and nondihydropyridine calcium channel blockers are ineffective or contraindicated, digoxin can be considered for acute rate control, either alone or in combination with the aforementioned agents.[5–9]
2a	A	3. In patients with AF with rapid ventricular response, the addition of intravenous magnesium to standard rate-control measures is reasonable to achieve and maintain rate control.[10,11]
2b	B-NR	4. In patients with AF with rapid ventricular response who are critically ill and/or in decompensated HF in whom beta blockers and nondihydropyridine calcium channel blockers are ineffective or contraindicated, intravenous amiodarone may be considered for acute rate control.*[12,13]
3: Harm	B-NR	5. In patients with AF with rapid ventricular response and known moderate or severe LV systolic dysfunction with or without decompensated HF, intravenous nondihydropyridine calcium channel blockers should not be administered.[14,15]

*Consider the risk of cardioversion and stroke when using amiodarone as a rate-control agent.

Figure 2. Acute Rate Control Strategy for Atrial Fibrillation

The next question to ask is: What about rhythm control? Well, this depends on the patient that is presenting to your ER. Typically, these patients have some finite criteria required to consider rhythm control, which includes the following: New onset atrial fibrillation, being a good historian, less than 12-48 hours onset, young age, low CHAD2VASC score, already on therapeutic anticoagulation, hemodynamically stable. If your patient falls into this category, great you can consider rhythm control with cardioversion. Now we review the efficacy of this approach.

The EAST-AFNET 4 trial conducted in 2020 asked the question if there was a difference in rate vs. rhythm control. The study showed that early rhythm-control therapy was associated with a lower risk of adverse cardiovascular outcomes than usual care among patients with early atrial fibrillation and cardiovascular conditions[4]. Prior to the AFNET trial was a prospective cohort study in Canada performed by Stiell et al. who compared outcomes in patients with atrial fibrillation treated with either IV procainamide or cardioversion. 1091 patients were enrolled and the authors found that 10.5% of recent-onset atrial fibrillation and flutter patients had adverse events within 30 days, there were no related deaths and 1 stroke (0.1%)[5]. They concluded that an ED strategy of sinus rhythm restoration and discharge in most patients is effective and safe.

Another two major studies for the support of rhythm control were the RAFF and RAFF-2 trials. The RAFF trial included 1736 patients all receiving electro cardioversion, with a success

rate of 90.2% and serious events rate of 0.4%[6]. The important adverse event rate was 13.9 %, with the vast majority related to sedation, mainly transient hypotension and respiratory events. These events were associated age 85 years, history of coronary disease, use of midazolam and use of fentanyl with the procedural sedation. The RAFF-2 trial focused on the comparison of electrical cardioversion vs. pharmacologic conversion. A total of 396 patients were enrolled in this randomized, blinded, placebo-controlled comparison of attempted pharmacological cardioversion with IV procainamide (15mg/kg in 500mL of 0.9% saline over 30min with max dose of 1500mg) followed by electrical cardioversion if necessary (up to 3 shocks, each at ≥200J) vs placebo infusion followed by electrical cardioversion[7]. The results of the comparison are below.

Drug-Shock: 96% (52% converted prior to shock) conversion to normal sinus rhythm
Shock Only: 92% (9% converted with placebo prior to shock) conversion to normal sinus rhythm
No adverse events in either group
No difference between anterior posterior vs anterior lateral pad placement

Figure 3. RAFF-2 Trial Results[7]

The ultimate decision remains with the clinician to adopt the role of electrical cardioversion into everyday practice for the patient that falls within acceptable criteria. The role of cardioversion is safe and efficacious for rhythm control in new onset atrial fibrillation. The final and important fact to consider is to utilize follow-up anticoagulation, especially in elderly patients age > 65 as well as with patients with CHAD2VASC score > 1. For this clinician, I will certainly be using rhythm control for new onset atrial fibrillation in the appropriate setting.

8.2.2. Electrical Cardioversion

Recommendations for Electrical Cardioversion
Referenced studies that support the recommendations are summarized in the Online Data Supplement.

COR	LOE	Recommendations
1	C-LD	1. In patients with hemodynamic instability attributable to AF, immediate electrical cardioversion should be performed to restore sinus rhythm.[1]
1	B-R	2. In patients with AF who are hemodynamically stable, electrical cardioversion can be performed as initial rhythm-control strategy or after unsuccessful pharmacological cardioversion.[2]
1	C-LD	3. In patients with AF undergoing electrical cardioversion, energy delivery should be confirmed to be synchronized to the QRS to reduce the risk of inducing VF.[3]
2a	B-R	4. For patients with AF undergoing elective electrical cardioversion, the use of biphasic energy of at least 200 J as initial energy can be beneficial to improve success of initial electrical shock.[4,5]
2a	B-NR	5. In patients with AF undergoing elective cardioversion, with longer duration of AF or unsuccessful initial shock, optimization of electrode vector, use of higher energy, and pretreatment with antiarrhythmic drugs can facilitate success of electrical cardioversion.[5–9]
2b	C-LD	6. In patients with obesity and AF, use of manual pressure augmentation and/or further escalation of electrical energy may be beneficial to improve success of electrical cardioversion.[10]

Figure 4. Recommendation For Cardioversion

Bibliography

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