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Continuous Core Content

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Atrial Fibrillation

Jessica Mason MD, Stuart Swadron MD, Mel Herbert MD

* *Drug doses are a guide only, always check second source and follow local practice guidelines*

Take Home Points:

- **Atrial fibrillation (AF) is the most common arrhythmia**
- **Patients with AF may present to the emergency department with palpitations or more vague symptoms like fatigue or shortness of breath**
- **New onset AF may be a sign of a more serious underlying condition such as PE, CHF, ACS, decompensated valvular disease, thyroid storm or a shock state**
- **Unstable patients may be electrically cardioverted**
- **Stable patients who are candidates for rhythm control may be electrically or chemically cardioverted**
- **Cardioversion should be avoided in patients with AF > 48 hours unless they have been anticoagulated for at least 3 weeks, or if they are unstable**
- **Rate control with AV nodal blockers is the default strategy for all of those who do not qualify for cardioversion**
- **Anticoagulation should be considered on a case-by-case basis considering both the patient's stroke risk and bleeding risk**

Introduction

Atrial fibrillation (AF) is the most common tachyarrhythmia. As such, AF is bread and butter for the emergency provider. We need to really understand and be comfortable with this rhythm.

In AF, instead of contracting regularly, the atrium fibrillates, meaning that it vibrates inefficiently - the classical description is that of a "bag of worms." Patients may suffer from AF transiently, intermittently or as a chronic persistent heart rhythm. Chronic AF occurs most commonly in the elderly and in patients with other chronic medical illnesses such as ischemic heart disease, hypertension, and diabetes.

The major consequences of AF to the patient are twofold. First, without the coordinated contraction of the atrium, there is an impairment in the filling of the ventricles and hence a reduction in the cardiac output. Second, the stagnant blood flow that results from AF tends to promote clot formation in the atria; clots which can embolize throughout the systemic circulation. In fact, the rate of embolic stroke alone is 5% per year across all patients with AF. This risk of embolization becomes especially pronounced when patients in chronic AF convert to sinus rhythm.

Atrial fibrillation comes into play in the emergency department (ED) in a variety of ways. In many cases, the onset of AF is the primary reason for a patient's presentation to the ED. In other cases, AF is a secondary feature of another process, like pulmonary embolism or myocardial ischemia. In still others, AF is an important co-morbidity that plays a role in diagnosis and decision making. In this episode of C3, we focus on patients that present to the ED specifically on account of their AF.

Common Misunderstandings

- Many clinicians assume that the risk of embolic disease associated with cardioversion also applies only to those converted by electricity. In fact, the same risk also applies for patients who are convert spontaneously or with medications.
- There is an important difference between rate control drugs and rhythm control drugs
  - Both will slow the ventricular rate but only rhythm control drugs will "convert" the patient to sinus rhythm
  - Rate control
    - Nodal blockers such as digoxin, beta-blockers, calcium channel blockers
  - Rhythm control
    - Antiarrhythmics such as procainamide (Class Ia) and amiodarone (Class III)
    - Using these to convert the rhythm to sinus carries a risk of embolization

Key Considerations On History, Examination And Workup

- Is the patient stable or unstable?
  - No specific vital sign numbers define stability
  - In addition to vital signs, the overall appearance of the patient is critical
  - Unstable patients require immediate rate control or cardioversion as part of their initial resuscitation
  - This may change at any time
    - Ongoing cardiac monitoring, IV access and resuscitation equipment should be by the bedside
    - Apply defibrillation pads if any concern for instability

- Symptoms
  - Patients vary in the way that they perceive AF
  - Younger patients are more likely to experience palpitations
  - Patients may report shortness of breath, chest pain, or vague symptoms such as fatigue or dizziness

- Time of Onset
  - Many will be able to identify the exact time of onset, many will not
  - Patients with intermittent AF may not realize it

- Heart Rate
  - The heart rate in AF is the rate of ventricular response to the chaotic electrical storm in the atria
  - The AV node offers critical protection - without its slowing effect, patients in AF would degenerate into ventricular fibrillation (VF)
  - A typical ventricular response rate in AF is in the 140-180 bpm range
    - A slower ventricular response rate may indicate:
      - Medications on board (e.g., AV nodal blockers)
      - Ischemia
      - Hyperkalemia
      - Hypothermia
      - Digoxin toxicity
    - A faster ventricular response rate may indicate:
      - Drugs on board (e.g. sympathomimetics like cocaine)
      - Another emergency such as PE or another shock state
      - An accessory pathway is present (e.g., WPW) that enables impulses to bypass the AV node
      - If the rate is >200 bpm in the absence of drugs, it is likely that an accessory pathway is present

- Could this be something worse than just AF?
  - Some important conditions that may present with AF
    - Acute coronary syndrome (ACS)
    - Congestive heart failure (CHF)
    - Pulmonary embolism (PE)
    - Myocarditis/pericarditis
    - Hyperthyroidism/Thyroid storm
    - Valvular heart disease (e.g., mitral stenosis, regurgitation)
    - Pulmonary hypertension/Sleep apnea
    - Alcohol withdrawal
    - Hyperadrenergic states (e.g., drugs, shock states)

- What is the patient's anticoagulation status and bleeding risk?
  - If on warfarin, what was last PT/INR?

- Labs
  - Decision of what to order should come from history and physical
  - May not need any labs or just one or two (e.g., PT/INR)
  - Consider:
    - Troponin if there is suspicion for ACS
    - D-dimer if there is some suspicion of PE (low to moderate suspicion)
    - TSH if >55 yrs (and not recently tested) or younger with other symptoms
    - Electrolytes and magnesium if suspicious for abnormalities

- Imaging
  - As directed by history and physical
  - A chest x-ray may be helpful to identify heart size, signs of failure, and pulmonary pathology

- ECG
  - The hallmark of AF on ECG is that the rhythm is irregularly irregular
  - This is much easier to see when the rate is slow - a rapidly conducted AF can look very much like sinus tachycardia or paroxysmal SVT
  - Measuring out each R-R interval and revealing that they are all slightly different is helpful in confirming AF
  - The background atrial fibrillatory waves may appear differently from patient to patient
    - In some patients, coarse waves can be seen
    - In others, the baseline is relatively flat
    - It is possible to mistake fibrillation and flutter waves for P waves - if the rhythm is irregularly irregular, AF is most likely

Mangement

- Overall, the treatment options for AF with rapid ventricular response (RVR) include
  - Rhythm control
  - Rate control
  - Neither and treat the underlying cause
  - Anticoagulation to prevent embolic stroke
- The 2 main ways that we control the rate are:
  - **Rhythm conversion**
    - By converting the rhythm to normal sinus the heart rate will usually decrease
    - This is done either by electrical (shock!) or chemical (IV or PO meds) cardioversion
    - Do not attempt to cardiovert a stable patient unless:
      - They have been anticoagulated for at least 3 weeks OR
      - The onset of AF is clear and less than 48 hours ago
    - Cardioversion is unlikely to be successful in patients with longstanding AF and a large dilated atrium - these patients tend to regress back to AF after conversion
    - Electrical conversion
      - For unstable patients and selected stable patients
      - Use sedation and analgesia first
        - Options include fentanyl, midazolam, propofol, etomidate, methohexital, and ketamine
      - Deliver a **synchronized** shock at 200J
    - Chemical conversion (e.g., using medication)
      - For selected stable patients
      - All agents can have unintended side effects/protocols vary
      - **Magnesium**
        - Magnesium may convert AF to sinus and it is relatively safe
        - Given IV (2.5 g IV over 10 minutes)
      - **Amiodarone (Class III)**
        - Amiodarone initially slows the rate (it has beta blocker like properties) but after a few hours can convert the patient to sinus rhythm
        - Initial bolus is 300 mg IV given over 30 mins, followed by infusion at 50-100 mg/hour
      - **Procainamide (Class Ia)**
        - Procainamide is given as an IV infusion over 60 min until conversion to sinus rhythm (maximum dose 1g)
        - Infusion is also stopped for marked hypotension, QT prolongation/Torsades
      - **Ibutilide (Class Ic)**
        - Given as an IV infusion over 10 mins with repeat dose if no conversion to sinus
        - Watch for QT prolongation - stop if excessive or any Torsades
      - **Propafenone (Class Ic)**
        - Can be administered orally as part of an outpatient management strategy
  - **Rate control**
    - Patients who do not qualify for cardioversion are generally treated with rate control
    - Most patients will feel symptomatically better once their rate is controlled even when they remain in AF
    - AV blockade medications will reduce the ventricular response rate
    - The ventricular response rate is extremely important
    - Patients with an underlying pathology such as CHF or PE may need a rapid ventricular rate in order to maintain adequate cardiac output
      - Once the ventricular rate exceeds 140-150 bpm, however, tachycardia is counterproductive as the heart no longer has time to fill in diastole - cardiac output then begins to decrease
      - Treatment of the heart rate in these scenarios is often simply the treatment of the underlying cause (e.g., heparin/tPA for PE, nitroglycerin for pulmonary edema)
      - AF/Aflutter secondary to medical illness should not be rate or rhythm controlled
      - There is 6-fold higher adverse event rate (hypotension, intubation, stroke, CPR, death, BVM) with treatment of AF itself (Scheuermeyer)
    - For most patients without a serious underlying cause, there is no consensus on what target heart rate that we should aim for
    - AHA/ACC guidelines are:
      - <80 bpm for symptomatic patients
      - <110 if asymptomatic and LV systolic function is preserved (the so-called lenient strategy)
    - Choices include:
      - Calcium channel blockers
        - Most popular among emergency providers
        - **Diltiazem** 20 mg initial bolus over 2 minutes, 2nd dose of 25 mg after 15 minutes if initial bolus is ineffective at slowing rate
          - Weight based dosing is 0.25 mg/kg initial bolus and 0.35 mg/kg 2nd bolus
          - May start with lower doses in elderly or hypotensive patient
        - Adding a dose of calcium (e.g., calcium gluconate 1g IV slow push) may help mitigate the hypotension caused by diltiazem (peripheral vasodilation of peripheral calcium blockade)
        - Due to the short half-life of diltiazem IV, patients typically need to be placed on a continuous IV drip (2.5-15 mg/hour) as a bridge to oral therapy
      - Beta blockers
        - More popular with cardiologists
        - Treatment of choice for AF in hyperthyroidism
        - Contraindicated in CHF and asthma/COPD
        - **Metoprolol** 5 mg initial slow IV bolus, 2nd dose of 5 mg after 5 minutes if initial bolus ineffective at slowing

rate (up to 15 mg)
- Metoprolol can be continued orally
- In many cases PO therapy alone from the outset may be sufficient

- Digitalis
  - "Old school"
  - "Digitalization" typically consisted of giving an IV dose of **digoxin** (0.25 mg) every 2 hours until the heart rate was controlled
    - It took several hours to get the rate under control
    - One advantage of digoxin is that it improves cardiac output unlike beta and calcium blockers

- **Anticoagulation**
  - Who needs to be started on anticoagulation?
    - Patients with transient AF (<48 hours) that convert to sinus rhythm do not need anticoagulation
    - The use of anticoagulation for patients with ongoing AF is dependent upon 2 factors:
      - The patient's risk of stroke
      - The patient's risk of bleeding
    - The **risk of stroke** can be estimated by using the CHADS2-VASC score (see Appendix 1)
      - Score increases with chronic illnesses
      - A score of 0 does not receive anticoagulation
      - A score of 2 or more should unless a major bleeding risk exists
      - A score of 1 remains controversial
    - The **risk of bleeding** can be estimated by using the HAS-BLED score (see Appendix 2)
      - A score >3 indicates a high risk of bleeding - a possible contraindication
    - Some of the same elements in the both CHADS2-VASC and HASBLED (age >65, prior stroke, HTN) so if you are high risk for one you are likely high risk for the other
    - Shared decision making with the patient is appropriate here
  - Many options are available for anticoagulation
    - **Heparin** IV bolus and infusion (for admitted patients only)
    - Unfractionated (UFH) or **low-molecular weight heparin** (LMWH) subcutaneously
    - Patients who receive heparin subcutaneously can be simultaneously started on **warfarin PO** (the heparin serves as a bridge for a few days until the warfarin becomes therapeutic)
    - **Novel Oral Anticoagulants** (NOACs)

Disposition

- We set the stage in the ED for the management that follows
  - What we start therapeutically is often continued on the ward and as outpatient
    - Continue on beta blocker, calcium channel blocker
    - Anticoagulation for at least 3 weeks if favorable stroke prevention/bleeding risk ratio
- A transesophageal echocardiogram (TEE) is sometimes performed to ensure that cardioversion is safe (e.g. to make sure that there is no atrial thrombus first)
- Who should be admitted?
  - Patients who present with AF and another active problem are generally admitted to a monitored bed
  - Some patients with AF may be appropriate for the ward if they are otherwise well and their heart rate is controlled
- Who can you discharge?
  - Patients who are:
    - Converted to sinus rhythm or if AF persists, with good rate control
    - No active comorbidities/underlying processes (e.g. CHF, thyroid storm, PE, ACS, pneumonia)
    - Have good follow up plan in place
    - If starting meds, they can get the meds you are prescribing

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APPENDIX 1
CHAD2VASC SCORE FOR RISK OF STROKE IN AF

	Condition	Points
C	Congestive heart failure (or Left ventricular systolic dysfunction)	1
H	*Hypertension*: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	1
A_2	Age ≥75 years	2
D	Diabetes Mellitus	1
S_2	Prior *Stroke* or *TIA* or *thromboembolism*	2
V	Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)	1
A	Age 65–74 years	1
Sc	Sex category (i.e. female sex)	1

CHA2DS2-VASc Score	Stroke Risk %	95% *CI*
0	0	-
1	1.3	-
2	2.2	-
3	3.2	-
4	4.0	-
5	6.7	-
6	9.8	-
7	9.6	-
8	12.5	-
9	15.2	-

Source: Wikipedia
https://en.wikipedia.org/wiki/CHA2DS2–VASc_score

APPENDIX 2
HASBLED SCORE TO ESTIMATE RISK OF BLEEDING IN AF

	Condition	Points
H	*Hypertension*: (uncontrolled, >160 mmHg systolic)	1
A	*Abnormal renal function*: Dialysis, transplant, Cr >2.26 mg/dL or >200 µmol/L	1
	Abnormal liver function: Cirrhosis or Bilirubin >2x Normal or AST/ALT/AP >3x Normal	1
S	*Stroke*: Prior history of stroke	1
B	*Bleeding*: Prior Major Bleeding or Predisposition to Bleeding	1
L	Labile *INR*: (Unstable/high INRs), Time in Therapeutic Range < 60%	1
E	Elderly: Age > 65 years	1
D	Prior Alcohol or Drug Usage History (≥ 8 drinks/week)	1
	Medication Usage Predisposing to Bleeding: (Antiplatelet agents, NSAIDs)	1

Source: Wikipedia
https://en.wikipedia.org/wiki/HAS-BLED