Atrial Fibrillation

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THE UNIVERSITY OF ARIZONA

How to get ahold of us:

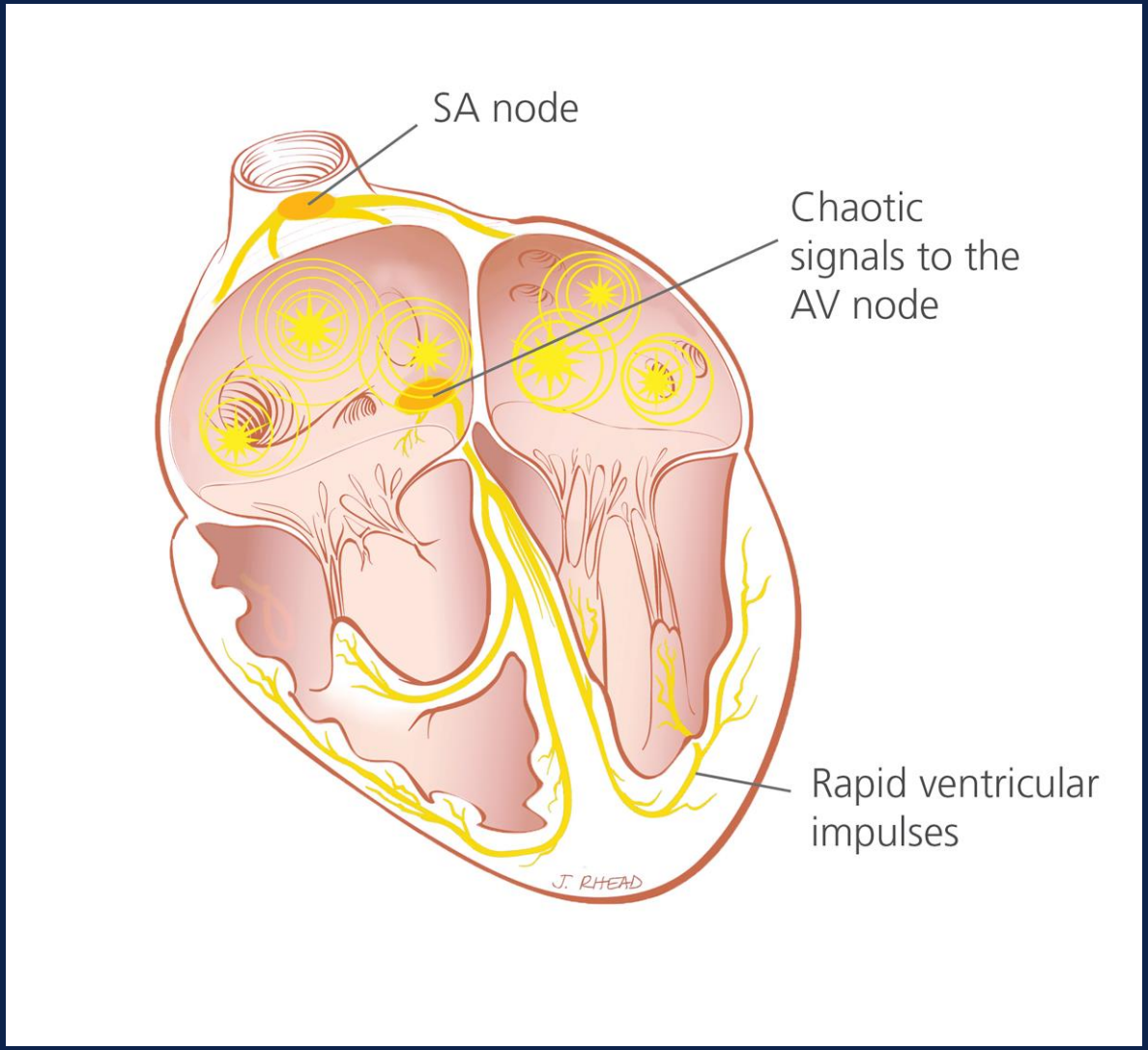
- Academic cards rounds daily
- Fellow in-house during weekdays and on-call weekends/holidays
- Answering service: 602-521-3090

Objectives:

- Make atrial fibrillation great again (using guidelines)
- Understand AFib basics
- Treatment:
  - Rhythm vs. Rate:
    - Describe the patient who benefits from rhythm control
  - Describe the appropriate anticoagulation recommendations
  - Rapid Ventricular Response:
    - Hemodynamically stable patient
    - Hemodynamically unstable patient

AFib = Sinus Tach*

Anatomy


SA node
Chaotic signals to the AV node
Rapid ventricular impulses
J. RHEAD

Basics: Definition

EKG is irregularly irregular with no clear discernable P waves

- There is atrial activity - chaotic and disorganized
- Fibrillatory waves can be mistaken as flutter waves (coarse Afib)
- Flutter waves look identical/organized (also ~300 beats/min)
- Atrial rate ranging from 400 to 700 beats/min

Definitions

- Paroxysmal AF
  - AF that terminates spontaneously or with intervention within 7 d of onset.
  - Episodes may recur with variable frequency.
- Persistent AF: Continuous AF that is sustained >7 d.
- Long-standing persistent AF Continuous AF >12 mo in duration.
- Permanent AF - Used when the patient and clinician make a joint **decision** to stop further attempts to restore and/or maintain sinus rhythm.
- Nonvalvular AF - AF in the absence of rheumatic mitral stenosis, a mechanical or bioprosthetic heart valve, or mitral valve repair.*

Lone AFib – not in guidelines

- Controversial term
- Don’t use it but know what it means
- Younger persons without:
  - Clinical or echocardiographic evidence of cardiopulmonary disease
  - Hypertension
  - DM

AFib – Random Facts

- Normal atrial myocardium has the following properties:
  - A short action-potential duration.
  - Cellular reactivation can occur rapidly due to the short refractory period (in contrast to Purkinje fibers and ventricular muscle).
  - Very rapid electrical conduction can occur.
  - The refractory period shortens with increasing rate.
- In the aggregate, these electrophysiologic properties permit the development of very complex patterns of conduction and an extremely rapid atrial rate as seen in AF.
- Select associated conditions:
  - Hypertension (80 percent)
  - CAD (60 percent)
  - Hyperlipidemia (62 percent)
  - HF (50 percent)
  - Anemia (42 percent)
  - Diabetes (36 percent)
  - Age (100 percent)

AFib – Random Facts

- Narrow complex tachycardia – Differential diagnosis
  - ***Irregular Narrow Complex Tachycardia = 1) A-fib, 2) A-flutter with variable conduction, or 3) MAT**
- Triggers:
  - AF is initiated (triggered) predominantly by rapid firing from the pulmonary veins.
  - Atrial tachycardia, atrial flutter, and other supraventricular tachycardias can initiate AF in predisposed patients.
  - Hypertension, valvular heart disease, congestive heart failure, coronary artery disease, physiologic stress, PE, COPD, infection, OSA, cardiac surgery.
- In patients with persistent AF, the prevailing understanding of the mechanism is that, once triggered, the arrhythmia is maintained (sustained) by one or more abnormalities in the atrial tissue.
  - Atrial remodeling
  - Electrical remodeling
  - Autonomic nervous system
  - Fibrosis
  - Inflammation and oxidative stress
  - Reentrant mechanism

AFib – Random Facts

- A ventricular rate below 60 beats per minute, in the absence of AV nodal blocking agents, suggests AV nodal disease that may be associated with the sinus node dysfunction.
- A ventricular rate above 170 beats per minute suggests thyrotoxicosis, catecholamine excess, parasympathetic withdrawal, or the existence of an accessory bypass tract in the preexcitation syndrome.
- Loss of atrial contraction may markedly decrease cardiac output, particularly when diastolic ventricular filling is impaired by mitral stenosis, hypertension, hypertrophic cardiomyopathy (HCM), or restrictive cardiomyopathy.

AFib – Considerations

- Main goal is to manage symptoms and potential complications
- Evaluate reversible causes
  - Hyperthyroidism
  - Pericarditis
  - Pulmonary embolism
  - Binge drinking
  - Valvular disease
- Management of risk factors
  - Hypertension
  - Obesity
  - Obstructive sleep apnea
- Rate vs Rhythm control
- Anticoagulation

Items to consider

- Is this really atrial fibrillation?
- Stable or unstable
- Fast or Slow
- Ongoing Ischemia
- Acute or Chronic
- Heart failure or not

Question

75-year-old female with hypertension and atrial fibrillation presented to your office for consultation. She is asymptomatic. Her medications include carvedilol 6.25 mg BID, lisinopril, and rivaroxaban.

On examination, her blood pressure was 105/70 mm Hg, her heart rate was 98 bpm, and she was afebrile.

Cardiac examination revealed irregularly irregular rhythm and the remainder of her examination was unremarkable.

EKG showed atrial fibrillation at approximately 100 bpm and left bundle branch block.

A nuclear myocardial perfusion scan performed 6 months prior showed a normal left ventricular ejection fraction (LVEF) without perfusion defects.

Which of the following is the most appropriate next step in the management of this patient?

A. Add dofetilide.
B. No change in therapy.
C. Add diltiazem.
D. Add digoxin.
E. AFib ablation.

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Trials

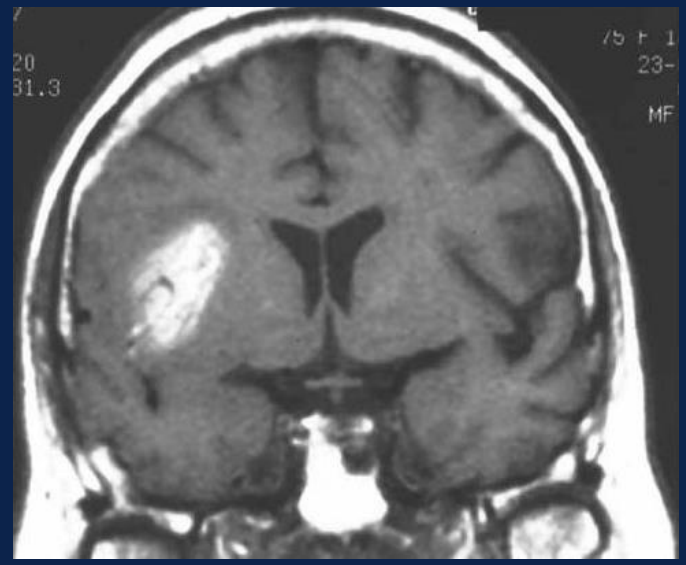
- AFFIRM Trial
  - Rate vs Rhythm
  - Nonvalvular atrial fibrillation, there is no ***survival*** benefit between rate and rhythm control. Rhythm had a tendency toward increased mortality.
  - Watch out for new information coming out showing improved morbidity with rhythm control, especially in younger patients.
- RACE II Trial
  - Strict or lenient control of atrial fibrillation rates.
  - Permanent atrial fibrillation patients, HR<110bpm is as effective as strict HR<80bpm in preventing cardiovascular events.
- ROCKET AF
  - Among patients with non-valvular Afib, rivaroxaban is noninferior to warfarin in preventing stroke and systemic thromboembolism.

Boring guidelines – Rate control

TABLE 9 Summary of Recommendations for Rate Control

Recommendations	COR	LOE	References
Control ventricular rate using a beta blocker or nondihydropyridine calcium channel antagonist for paroxysmal, persistent, or permanent AF	I	B	(267-269)
IV beta blocker or nondihydropyridine calcium channel blocker is recommended to slow ventricular heart rate in the acute setting in patients without pre-excitation. In hemodynamically unstable patients, electrical cardioversion is indicated	I	B	(270-273)
For AF, assess heart rate control during exertion, adjusting pharmacological treatment as necessary	I	C	N/A
A heart rate control (resting heart rate <80 bpm) strategy is reasonable for symptomatic management of AF	IIa	B	(269,274)
IV amiodarone can be useful for rate control in critically ill patients without pre-excitation	IIa	B	(275-277)
AV nodal ablation with permanent ventricular pacing is reasonable when pharmacological therapy is inadequate and rhythm control is not achievable	IIa	B	(278-280)
A lenient rate-control strategy (resting heart rate <110 bpm) may be reasonable when patients remain asymptomatic and LV systolic function is preserved	IIb	B	(274)
Oral amiodarone may be useful for ventricular rate control when other measures are unsuccessful or contraindicated	IIb	C	N/A
AV nodal ablation should not be performed without prior attempts to achieve rate control with medications	III: Harm	C	N/A
Nondihydropyridine calcium channel antagonists should not be used in decompensated HF	III: Harm	C	N/A
With pre-excitation and AF, digoxin, nondihydropyridine calcium channel antagonists, or amiodarone should not be administered	III: Harm	B	(281)
Dronedarone should not be used to control ventricular rate with permanent AF	III: Harm	B	(282,283)

Anticoagulation



- What are we avoiding?
- Embolic stroke:
  - Most often occur suddenly (maximal at onset)
  - Rapid recovery
  - Can be precipitated by getting up at night to urinate or sudden coughing or sneezing
- The ratio of hemispheric events to retinal events was 25:1 with AF compared with 2:1 with carotid disease
- AF is also associated with silent cerebral infarctions and TIAs
- Neuroimaging
  - Simultaneous or sequential strokes in different arterial territories
  - Generally massive, superficial, single large striatocapsular or multiple infarcts in the middle cerebral artery
  - Predominate in the carotid and middle cerebral artery distributions

Anticoagulation

Antithrombotic therapy based on shared decision making, discussion of risks of stroke and bleeding, and patient's preferences	I	C	N/A
Selection of antithrombotic therapy based on risk of thromboembolism	I	B	(167–170)
CHA_2DS_2-VASc score recommended to assess stroke risk	I	B	(171–173)
Warfarin recommended for mechanical heart valves and target INR intensity based on type and location of prosthesis	I	B	(174–176)
With prior stroke, TIA, or CHA_2DS_2-VASc score ≥2, oral anticoagulants recommended. Options include:			
Warfarin	I	A	(171–173)
Dabigatran, rivaroxaban, or apixaban	I	B	(177–179)
For atrial flutter, antithrombotic therapy is recommended as for AF	I	C	N/A
With nonvalvular AF and CHA_2DS_2-VASc score of 0, it is reasonable to omit antithrombotic therapy	IIa	B	(183,184)
With CHA_2DS_2-VASc score ≥2 and end-stage CKD (CrCl <15 mL/min) or on hemodialysis, it is reasonable to prescribe warfarin for oral anticoagulation	IIa	B	(185)
With nonvalvular AF and a CHA_2DS_2-VASc score of 1, no antithrombotic therapy or treatment with oral anticoagulant or aspirin may be considered	IIb	C	??? N/A

Consider using GARFIELD-AF SCORE

- In the derivation of the $CHADS_2$ and $CHADS_2VASc$ risk scores, few very low to low risk patients were included (103 patients had a score of 0, and 162 patients a score of 1 out of 1084)
- Large-scale international registry programs demonstrate substantial divergence in clinical practice compared with guideline recommendations.

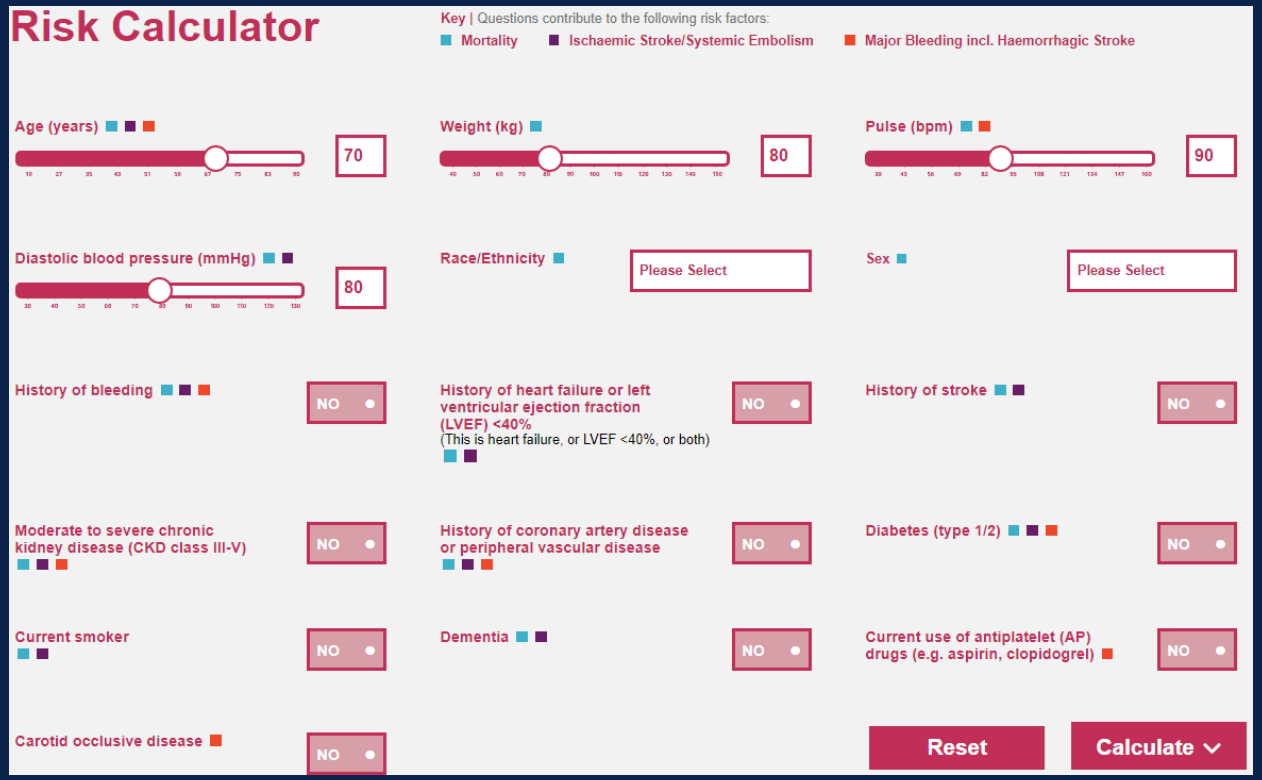
- 40,000 patients
- Externally validated
- Simultaneously calculates stroke, bleeding, and mortality risk.
- **Patients with a transient reversible cause of AF and those for whom follow-up was not completed were excluded.**
- Best used for males with $CHADS_2VASc$ 0-1 and females with $CHADS_2VASc$ of 0-2.

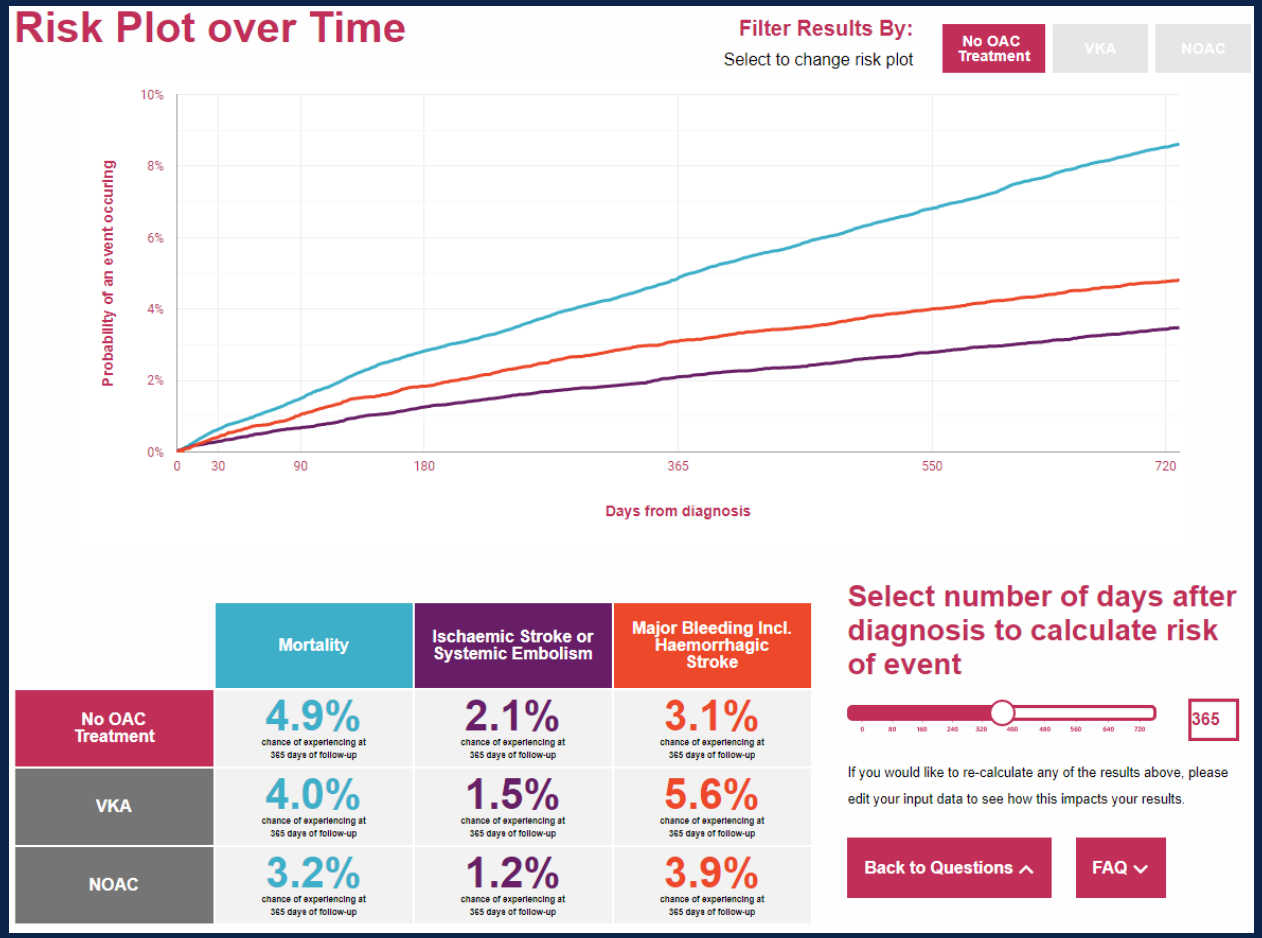
Results: Performance of $CHADS_2VASc$ and GARFIELD-AF risk models

Table 3 Comparison of the performance of the new GARFIELD-AF risk model with CHA_2DS_2-VASc (death, stroke or systemic embolism) or HAS-BLED* for major bleeding in all patients overall and by treated and untreated with anticoagulation and also for patients with lower stroke risk.

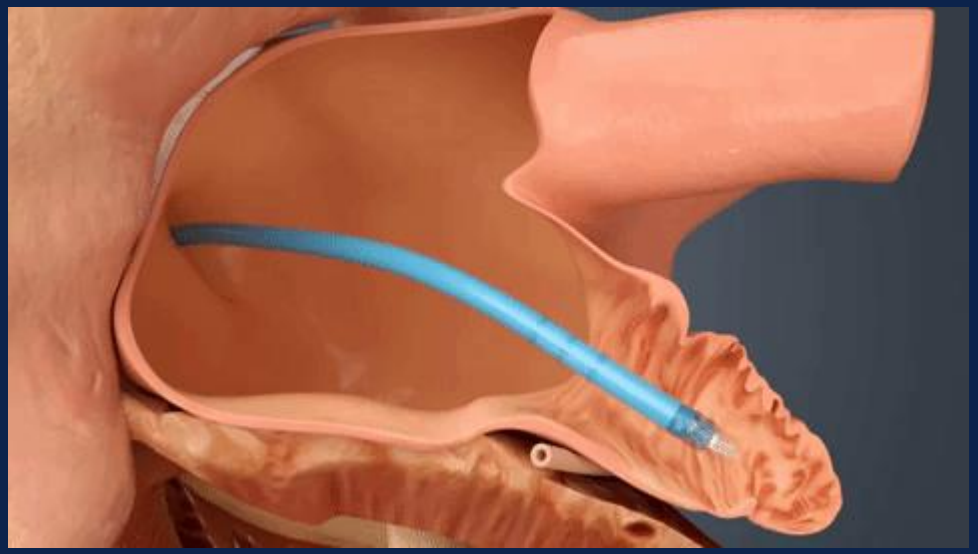
	C-index		P value of test for one risk model over the other	
Events	GARFIELD-AF risk model	CHA_2DS_2-VASc (HAS-BLED for bleeding)*	GARFIELD-AF risk model	CHA_2DS_2-VASc (HAS-BLED for bleeding)*
All patients				
All-cause mortality	0.77 (0.76–0.78)	0.66 (0.64–0.67)	<0.001	0.165
Anticoagulant treated	0.75 (0.73–0.77)	0.65 (0.63–0.66)	<0.001	0.186
Anticoagulant untreated	0.78 (0.77–0.80)	0.68 (0.66–0.70)	<0.001	0.507
Ischaemic stroke/systemic embolism	0.69 (0.67–0.71)	0.64 (0.61–0.66)	<0.001	0.006
Anticoagulant treated	0.67 (0.64–0.71)	0.64 (0.60–0.67)	<0.001	0.020
Anticoagulant untreated	0.69 (0.65–0.72)	0.65 (0.61–0.68)	<0.001	0.047
Major bleed (anticoagulant treated)	0.66 (0.62–0.69)	0.64 (0.61–0.68)*	<0.001	0.001*
Very low to low risk patients CHA_2DS_2-VASc score of 0 or 1 (men) and 1 or 2 (women); HAS-BLED 0 for bleeding				
All-cause mortality	0.69 (0.64–0.75)	0.50 (0.45–0.55)	<0.001	0.383
Ischaemic stroke/systemic embolism	0.65 (0.56–0.73)	0.59 (0.50–0.67)	0.004	0.108
Major bleed (anticoagulant treated)	0.60 (0.47–0.73)	0.55 (0.53–0.56)*	0.299	0.403*
Low to intermediate or higher risk patients (sensitivity analysis) CHA_2DS_2-VASc score 0, 1 or 2 (men) and 1, 2 or 3 (women); HAS-BLED score 0 or 1 for bleeding				
All-cause mortality	0.72 (0.70–0.75)	0.56 (0.54–0.59)	<0.001	0.377
Ischaemic stroke/systemic embolism	0.67 (0.63–0.72)	0.58 (0.54–0.62)	<0.001	0.087
Major bleed (anticoagulant treated)	0.64 (0.58–0.71)	0.62 (0.58–0.65)*	0.001	1.000*

Consider using GARFIELD-AF SCORE





What about Watchman????



- I will attempt to refrain from personal bias
- Key facts from trial:
  - Noninferior compared to warfarin (not DOAC)
  - Noninferiority was set to within 40%
  - Still requires 45 days of uninterrupted anticoagulation
- Side note:
  - HAS-BLED with questionable validation
  - HAS-BLED is very commonly miscalculated

Anticoagulation and Cardioversion

- < 48 hours – Elective DCCV and decide on AC if high risk.*
- > 48 hours, TEE with at least 4 weeks of anticoagulation afterwards.
- Unknown, TEE with at least 4 weeks of anticoagulation after.
- Unknown, 3 weeks of anticoagulation prior to cardioversion.*

Question

A 54-year-old female presents to your office regarding management of atrial fibrillation. Her PCP recommended catheter ablation due to palpitations and exercise intolerance.

Other medical problems include hypertension, diabetes mellitus, dyslipidemia, and obstructive sleep apnea.

Her medications include HCTZ, metoprolol succinate, metformin, rivaroxaban, atorvastatin, and zolpidem.

She reported that she wears a continuous positive airway pressure (CPAP) mask nightly. She drinks 1 alcoholic beverage nightly and admitted to smoking 1/2 pack cigarettes daily, although she is trying to quit.

On examination, BMI 30 kg/m^2. Vitas WNL, exam otherwise normal.

Echocardiogram showed normal ventricular and valvular function with no pulmonary hypertension.

A treadmill stress test showed no ischemia at adequate heart rate. She would like to avoid catheter ablation and asked what other interventions might help.

What intervention do you recommend?

A. Switch HCTZ to valsartan.
B. Change CPAP to BiPAP.
C. Weight loss of 15-20 lbs.
D. Smoking cessation.
E. Switch metformin to glyburide 5 mg daily.

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LEGACY Trial

Long-Term Effect of Goal-Directed Weight Management in an Atrial Fibrillation Cohort trial

- 335 patients with BMI >29 kg/m2 were offered weight management assistance and followed for 5 years
- Weight loss of ≥10% of body weight resulted in a six-fold greater probability of arrhythmia-free survival at 5 years

Boring Guidelines – 2019 update

- NOACs are recommended over warfarin except in patients with moderate to severe mitral stenosis or a prosthetic heart valve (COR I, LOE A).
- The decision to use an anticoagulant should not be influenced by whether the AF is paroxysmal or persistent (COR I, LOE B).
- Renal and hepatic function should be tested before initiation of a NOAC and at least annually thereafter (COR I, LOE B-NR).
- In AF patients with a CHA_2DS_2-VASc score ≥2 in men or ≥3 in women and a creatinine clearance <15 ml/min or who are on dialysis, it is reasonable to use warfarin or apixaban for oral anticoagulation (COR IIb, LOE B-NR).
- Idarucizumab is recommended for the reversal of dabigatran in the event of a life-threatening bleed or urgent procedure (COR I, LOE B-NR).
- Andexanet alfa (recombinant factor Xa) can be useful for the reversal of rivaroxaban and apixaban in the event of life-threatening bleeding (COR IIa, LOE B-NR).
- Percutaneous left atrial appendage occlusion may be considered for at-risk AF patients with AF at increased risk of stroke who have contraindications to long-term anticoagulation (COR IIb, LOE B-NR).
- AF catheter ablation may be reasonable in symptomatic patients with heart failure and a reduced ejection fraction to reduce mortality and heart failure hospitalizations (COR IIb, B-R).
- In at-risk AF patients who have undergone coronary artery stenting, double therapy with clopidogrel and low-dose rivaroxaban (15 mg daily) or dabigatran (150 twice daily) is reasonable to reduce the risk of bleeding as compared to triple therapy (COR IIa, B-R).
- Weight loss combined with risk factor modification is recommended for overweight and obese patients with AF (COR I, LOE B-R).
- In patients with cryptogenic stroke in whom **external ambulatory monitoring is inconclusive**, implantation of a cardiac monitor is reasonable for detection of subclinical AF (COR IIa, B-R).

Misc - Boring Guidelines

- Special populations
  - HOCM – always use AC, may need more rhythm control
  - ACS – Urgent cardioversion of new-onset AF in the setting of ACS is recommended for patients with hemodynamic compromise, ongoing ischemia, or inadequate rate control.
  - Hyperthyroid
  - Pulm diseases – A nondihydropyridine calcium channel antagonist is recommended to control ventricular rate with AF and COPD
  - WPW
  - CHF – Avoid IV nondihydropyridine calcium channel antagonists, IV beta blockers, and dronedarone should not be given with decompensated HF. Digoxin is effective to control resting heart rate with HFrEF.
  - Post-op – BB before CaChB

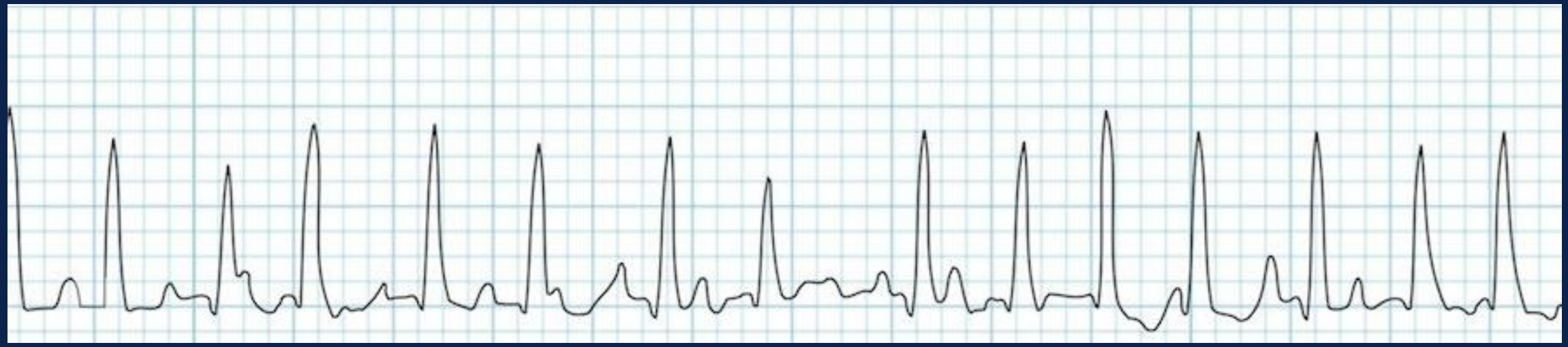
AFib = Sinus Tach*

Cases:

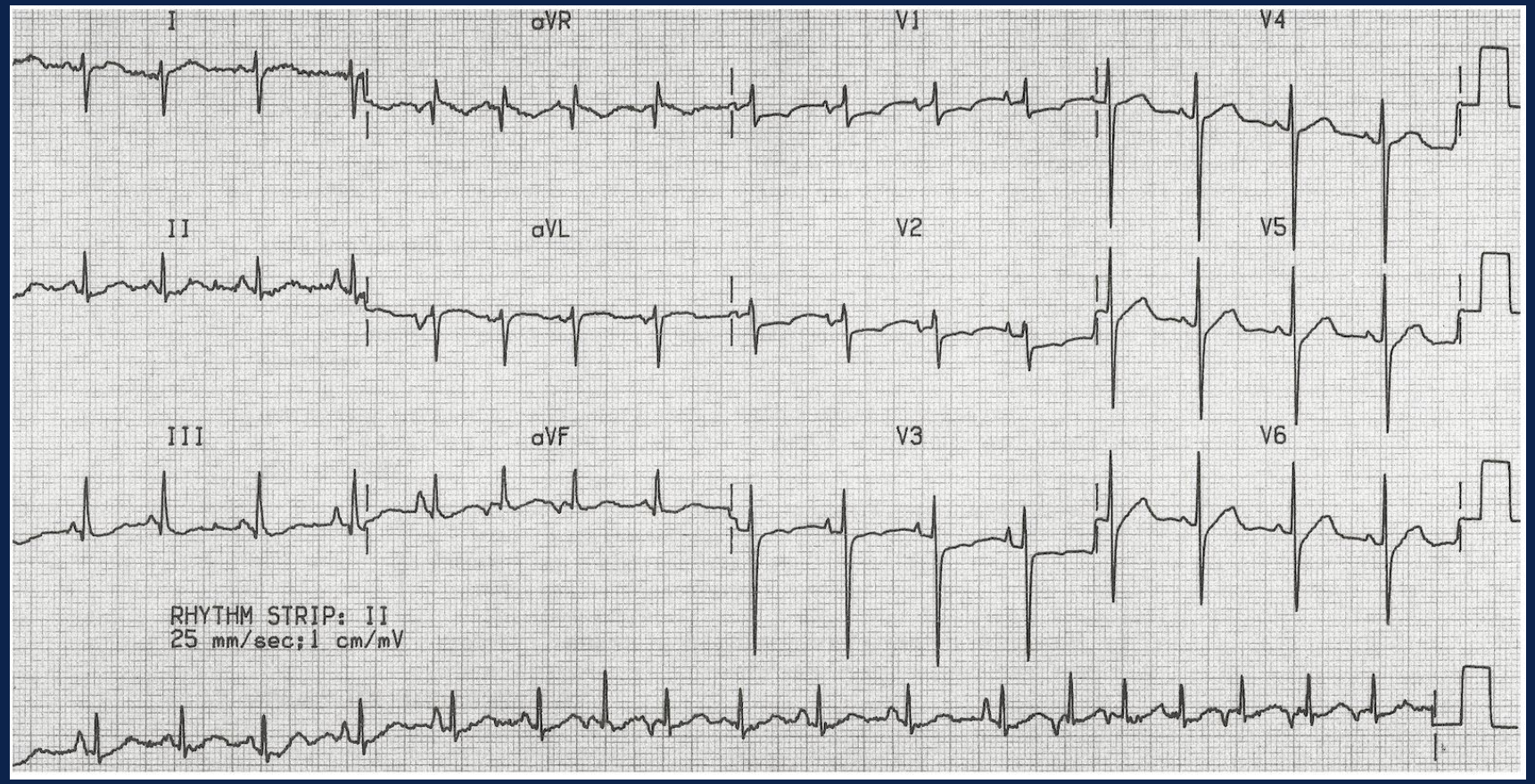
Question – Cross cover delight

- Close your eyes and picture the VA call room
- PAGE FROM 4C – PATIENT WITH FAST HEART RATE, TELE SAYS AFIB.
- CROSS COVER SIGN OUT
  - 66 YEAR OLD WITH COPD EXACERBATION …… NTD on signout
  - You decide this warrants a bedside eval
  - Let’s check out the tele strip…

Question – Cross cover delight



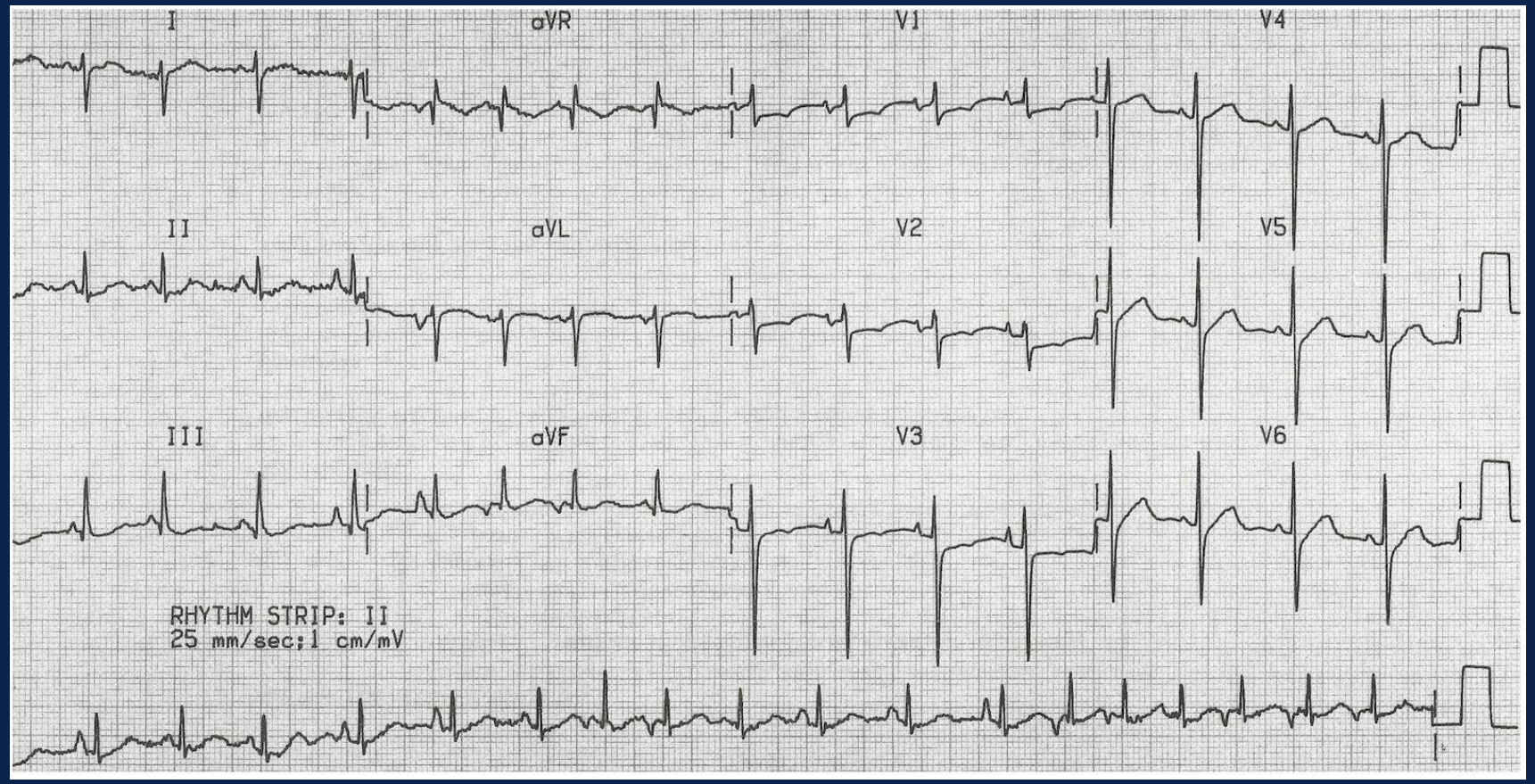
12-Lead EKG



- Diagnosis?

A. AFib
B. Sinus
C. MAT
D. Aflutter
E. SVT

12-Lead EKG



- Diagnosis?

A. AFib
B. Sinus
C. MAT
D. Aflutter
E. SVT

Multifocal atrial tachycardia

- Rapid irregular atrial rhythm arising from multiple ectopic foci within the atria
- Most commonly seen in severe COPD
- Heart rate >100 BMP
- Varying PP, PR, and RR intervals
- At least 3 distinct P wave morphologies in the same lead
- Isoelectric baseline between P waves
- Treatment: Treat underlying disease process/Consult Pulm ***STAT***

Question

- 62 year old gentleman who develops acute onset of chest pain.
- Classic story. He read the same AHD article the night before. It sounds legit.
- EKG – NO STEMI
- Chest pain order set done!
- Troponin is cooking in the lab.
- Clicked on the heparin gtt 3x. Cancelled the order 2x so far and now they just say pending.
- Clicked on labs/refreshed about 5x so far.

Hmm, look back at the EKG

- It’s atrial fibrillation with RVR.
- He’s going fast because of his chest pain.* I would be too.
- Troponin is back --- 4
- Repeat bedside eval
  - Mostly moans and groans
  - Blood pressure is 75/40
  - Called respiratory therapy to get STAT EKG

Next step?

A. Initiate pressors
B. Call cards for TEE
C. Start Amio
D. DCCV
E. IV Metoprolol

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Unstable Atrial Fibrillation

- Emergent Cardioversion
- Place the electrode pads
  - Preferable along left anteroposterior fashion.
    - Better for atrial arrhythmias, small interelectrode distance and less interposition of lung parenchyma.
    - Though has not been verified in a randomized trial and studies differ.
  - Synchronized to the QRS complex
  - Defibrillation – random delivery of shock during the cardiac cycle

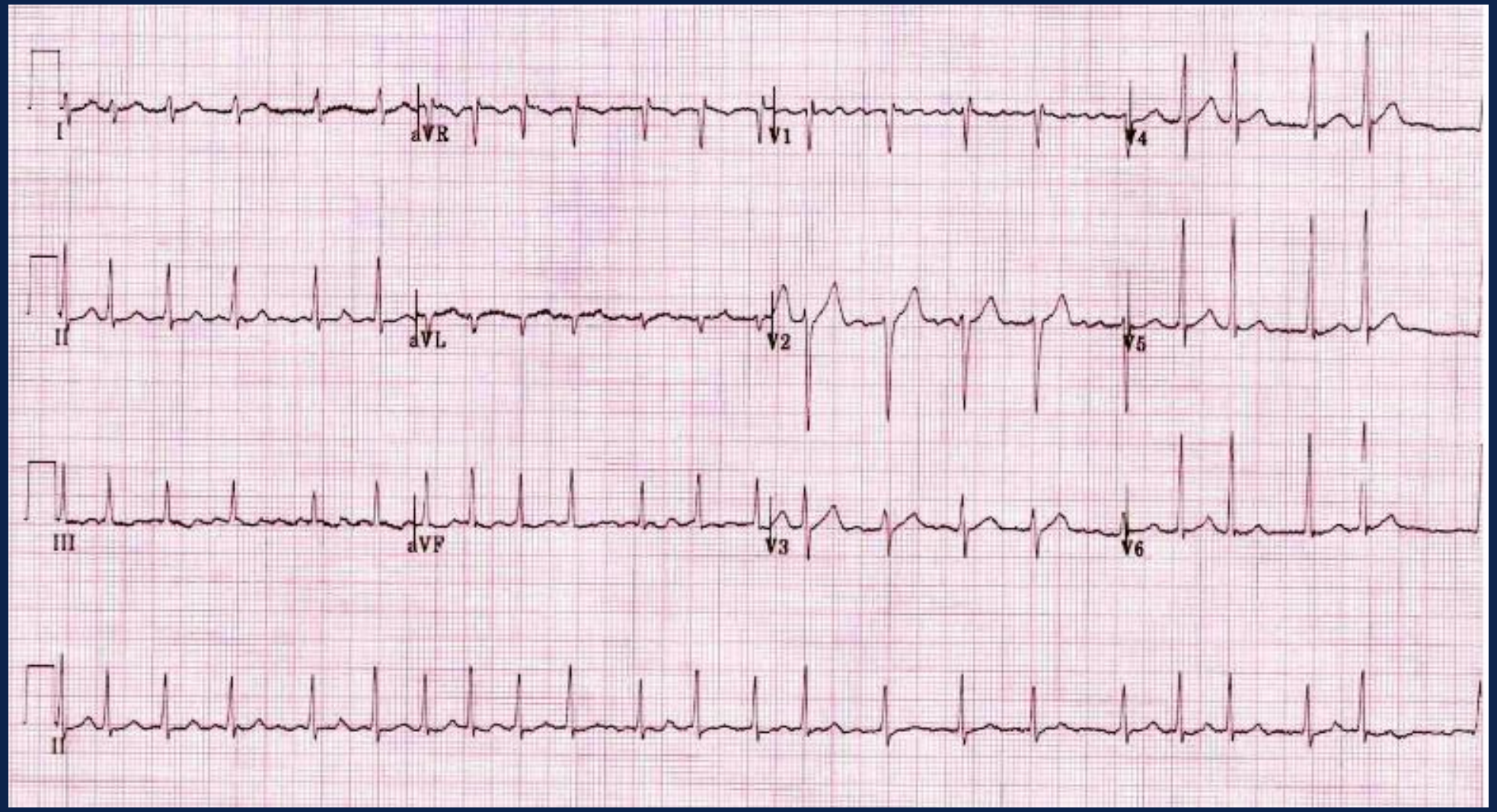
Shock

1. Place pads
2. Oxygen/airway/RT
3. Energy selection 120-200J (biphasic) (Best-AF trial)
4. Synchronization
    - This is to prevent the R on T phenomenon.
5. Clear/Shock

Case – 2:30 AM New Onset AFib

- PAGED BY 3B – Patient needs to be made a full admit and a renew Foley order….. Oh, and also has new onset atrial fibrillation
- Vitals are stable 3 hours ago

12 lead EKG



Treatment options

- Blood pressure 120/80, Normal mentation
- Physical exams show no signs of heart failure
- Medications
  - IV 5mg Metoprolol x 3, q 5 minutes

Now what do you do?

A. DCCV
B. More IV Metoprolol
C. Dilt ggt
D. Dig
E. Let it ride
F. Call cards
G. Amiodarone
H. Call Distler no matter what time it is
I. Hospice

IV Metoprolol doesn't work

- What now?!
- Diltiazem gtt
  - Call pharmacy?
  - Start off with a 10mg Diltiazem bolus (remember, stable BP) followed by running gtt at anywhere from 5 to 15mg/hr
  - Rates are better controlled
  - 4:00 AM success story

Diltiazem gtt

- Hmm, patient is on diltiazem gtt at 15mg/hr. Rate controlled. All is good.
- Leave it on until AM crew gets in.
- Standard Rates of Conversion
  - 5mg/hr – 180mg/day
  - 10mg/hr - 330mg/day
  - 15mg/hr – 480mg/day
- Divide by 4 to get the short acting dose.
- Give first dose about an hour prior to stopping the gtt.
- If this works, then you can convert to long acting diltiazem.
- Calcium channel blockers (except amlodipine) should generally be avoided in patients with heart failure with reduced ejection fraction (HFrEF) since they provide no functional or mortality benefit and some first generation agents may worsen outcomes. Will your patient immediately code if given dilt if the have a history of CHF? Probably not, but in general we avoid non-dihydropyridine CaChB in HFrEF.

Currently rate controlled but still in AFib

- Patient is highly symptomatic at a heart rate of 67 BMP
- What do you do?
  1. Go up on the PO dilt dose until heart rate is 60BPM
  2. Dismiss it, rate control is all you get
  3. Consult cardiology
  4. Slap the pads on him and lets get him in sinus rhythm
- This is good scenario where rate control is not enough. He clearly is symptomatic in mere presence of atrial fibrillation (A-V dyssynchrony). Rhythm control will be ideal for this patient.
- Good case for cardioversion, anti-arrhythmics, or even to go visit Dr. Su.

Case – MICU resident

- 2:30 AM – New admission arrives
  - 62 year old veteran from the AZ Veterans home.
  - Previous CVA, no history obtained, no family to call.
  - But 10,000 notes in CPRS to review.
  - Admitted to ICU for sepsis and hypotension.

Clues

- WBC 25k, Lactic acid 7, BNP 1,000
- Echo from 2001 shows an EF of 20%
- Vitals
  - 105/76, RR 18, Pulse 138

Treatment options

- Remember that tachycardia has its role in sepsis
- Low EF/marginal blood pressure
  - IV Metoprolol
  - IV Diltiazem
  - IV Sotalol
  - IV Amiodarone
  - Cardioversion

Amiodarone

- The gift from the Gods.
- Dose
  - 150mg over 10 minutes; 1mg/min for 6 hours, and then 0.5mg/min for 18 hours.
  - Who knows the total loading dose of amio?
  - Bonus* When do you give 300mg rapid bolus?
    - Pulseless VT/VF

Fear of Amiodarone

- I’m afraid to use Amio.
- I don’t want to cause a stroke. What if he/she converts on me?
  - Document why we need rate control now
- Should I just start a heparin gtt while I give amio?
- What about that lumbar puncture I still need to do?

Converting and throwing left atrial thrombus in the appendage

- Sepsis – Afib with RVR
  - May not be the ideal time for anticoagulation.
  - Guidelines state you should start AC as soon as possible but keep in mind any reason that the patient should not be on AC during their stay (need surgery?, LP?, central line?)

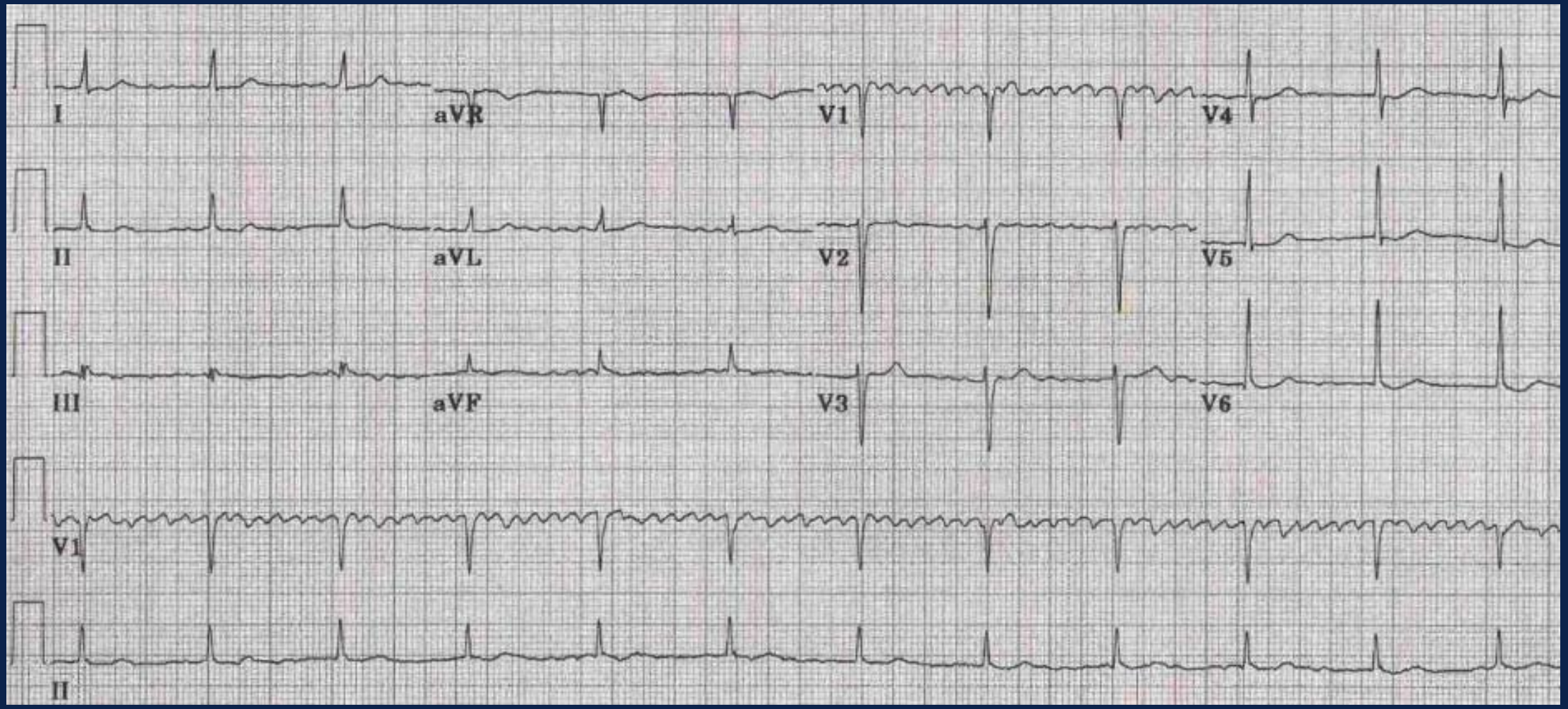
Critical care/AFib

- Esmolol
  - Beta block
  - Greater short half life, negative inotropic effect
- Diltiazem
  - Longer half life in as esmolol
  - Negative inotropic effects
  - Stay away in the (non-VA) ICU setting
- Amiodarone
  - Safe in heart failure
- Digoxin
  - Positive inotropic effect
  - Reduce dose in renal dysfunction

Case – History of AFib

- 84 year old male admitted for nausea, vomiting, and possible syncope.
  - History of heart failure, EF of 30%
  - Doesn’t remember his meds but has been on same ones “forever.”

12-Lead



RHYTHM ANALYSIS

- Ventricle -> Regular rate
- How does Afib give you a regular rate?
  - Not classic for flutter (rate, no sawtooth)
- It’s ok, the patient is rate controlled. My work is done.

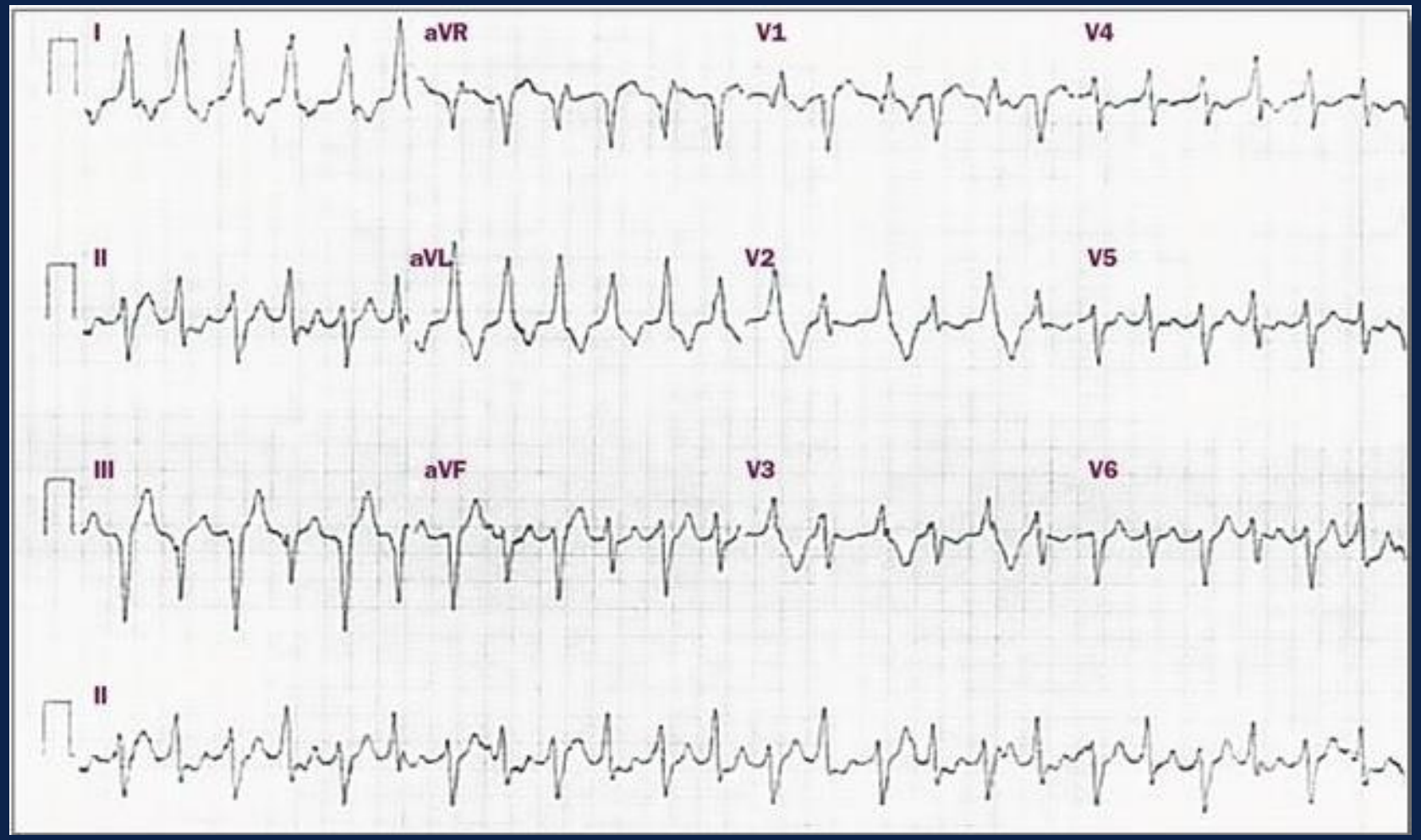
Digoxin toxicity

- This is complete heart block.
- We have a regularized ventricular rate in the setting of atrial fibrillation.
- Always be suspicious of some form of block when encountering a slow atrial fibrillation.

Treatment of Digoxin toxicity

- Any clinically significant arrhythmia from digitalis, can produce hypotension and will need digoxin specific antibody fragments.
- Contact poison control and the local adults.
- Symptomatic bradycardia can be treated with atropine until digi-bind is available.
- Any ventricular arrhythmia can be treated with standard ACLS.

Digoxin toxicity



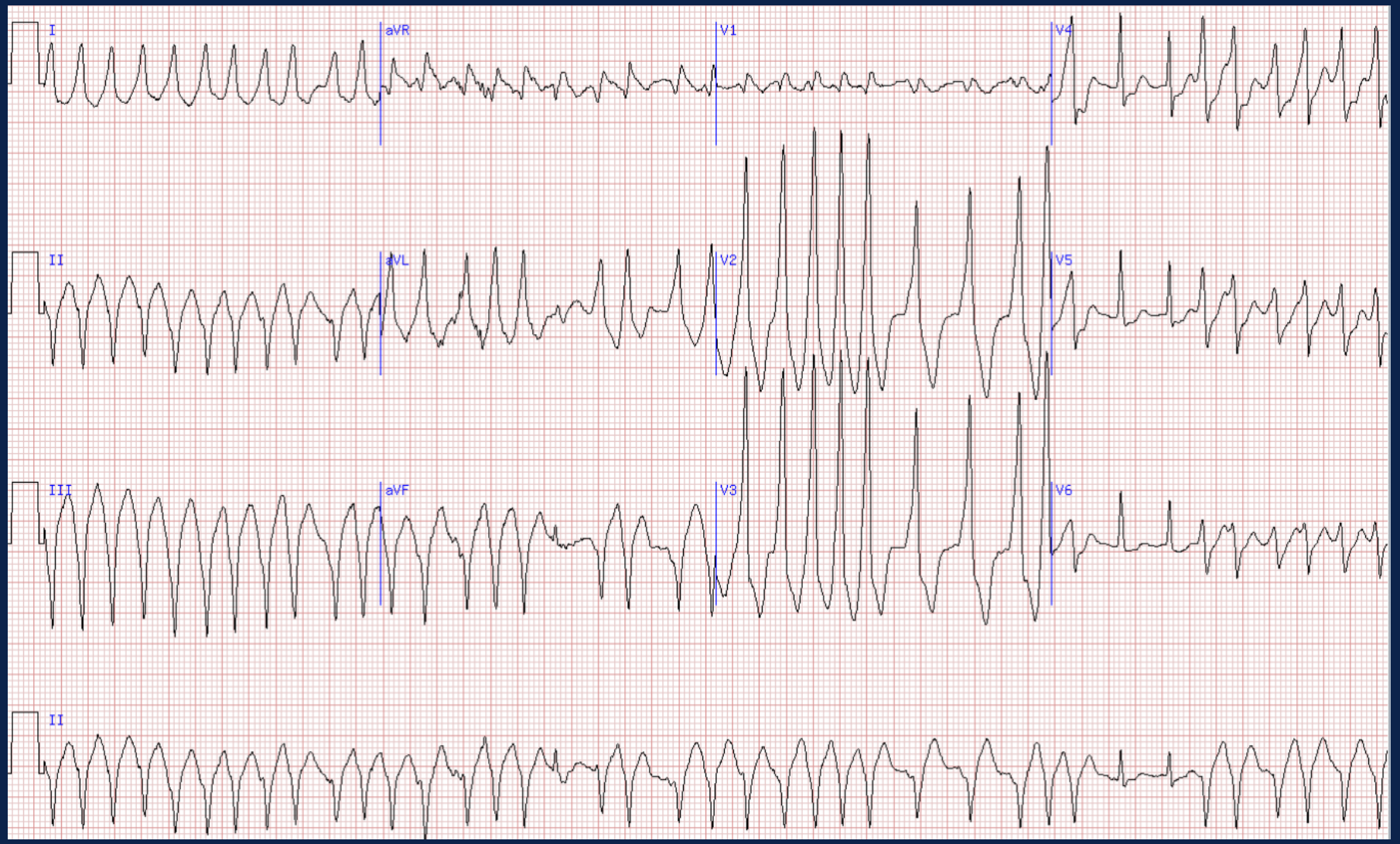
A few fun fact of popular AFib drugs

- Flecanide, Propafenone, and Sotalol – used for maintenance of sinus rhythm.
  - NO LVH, CAD, or Heart Failure
  - If your patient is on this drug, he/she shouldn't have the above conditions
- Heart Failure
  - Amiodarone
  - Dofetilide
- Patient has coronary artery disease
  - Options are Dofetilide, Sotalol, Amiodarone

Case

- 49 year old lady with a history of “palpitations” that is now short of breath at a rapid response.

12 lead ECG



WPW and AFib

- Can occur in up to 20% of patients with WPW.
- The accessory pathway allows for rapid conduction directly to the ventricles bypassing the AV node.
- Rapid ventricular rates may result in degeneration to VT or VF.
- Complexes vary in shape and width
- May approach 250-300 bpm or higher (hint that its going too fast for the AVN, therefore not a Afib with LBBB)
- Avoid all AV nodal blockers…including Amiodarone!
  - Use Procainamide, Flecainide(?), or electrical cardioversion

JAMA | Original Investigation

Effect of a Home-Based Wearable Continuous ECG Monitoring Patch on Detection of Undiagnosed Atrial Fibrillation
The mSToPS Randomized Clinical Trial

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IMPORTANCE Opportunistic screening for atrial fibrillation (AF) is recommended, and improved methods of early identification could allow for the initiation of appropriate therapies to prevent the adverse health outcomes associated with AF.

OBJECTIVE To determine the effect of a self-applied wearable electrocardiogram (ECG) patch in detecting AF and the clinical consequences associated with such a detection strategy.

DESIGN, SETTING, AND PARTICIPANTS A direct-to-participant randomized clinical trial and prospective matched observational cohort study were conducted among members of a large national health plan. Recruitment began November 17, 2015, and was completed on October 4, 2016, and 1-year claims-based follow-up concluded in January 2018. For the clinical trial, 2659 individuals were randomized to active home-based monitoring to start immediately or delayed by 4 months. For the observational study, 2 deidentified age-, sex- and CHA_2DS_2-VASc–matched controls were selected for each actively monitored individual.

INTERVENTIONS The actively monitored cohort wore a self-applied continuous ECG monitoring patch at home during routine activities for up to 4 weeks, initiated either immediately after enrolling (n = 1364) or delayed for 4 months after enrollment (n = 1291).

MAIN OUTCOMES AND MEASURES The primary end point was the incidence of a new diagnosis of AF at 4 months among those randomized to immediate monitoring vs delayed monitoring. A secondary end point was new AF diagnosis at 1 year in the combined actively monitored groups vs matched observational controls. Other outcomes included new prescriptions for anticoagulants and health care utilization (outpatient cardiology visits, primary care visits, or AF-related emergency department visits and hospitalizations) at 1 year.

RESULTS The randomized groups included 2659 participants (mean [SD] age, 72.4 [7.3] years; 38.6% women), of whom 1738 (65.4%) completed active monitoring. The observational study comprised 5214 (mean [SD] age, 73.7 [7.0] years; 40.5% women; median CHA_2DS_2-VASc score, 3.0), including 1738 actively monitored individuals from the randomized trial and 3476 matched controls. In the randomized study, new AF was identified by 4 months in 3.9% (53/1366) of the immediate group vs 0.9% (12/1293) in the delayed group (absolute difference, 3.0% [95% CI, 1.8%-4.1%]). At 1 year, AF was newly diagnosed in 109 monitored (6.7 per 100 person-years) and 81 unmonitored (2.6 per 100 person-years; difference, 4.1 [95% CI, 3.9-4.2]) individuals. Active monitoring was associated with increased initiation of anticoagulants (5.7 vs 3.7 per 100 person-years; difference, 2.0 [95% CI, 1.9-2.2]), outpatient cardiology visits (33.5 vs 26.0 per 100 person-years; difference, 7.5 [95% CI, 7.2-7.9), and primary care visits (83.5 vs 82.6 per 100 person-years; difference, 0.9 [95% CI, 0.4-1.5]). There was no difference in AF-related emergency department visits and hospitalizations (1.3 vs 1.4 per 100 person-years; difference, 0.1 [95% CI, −0.1 to 0]).

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Video and Supplemental content

CME Quiz at jamanetwork.com/learning and CME Questions page 199

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