Your Arrhythmia Worst Nightmare

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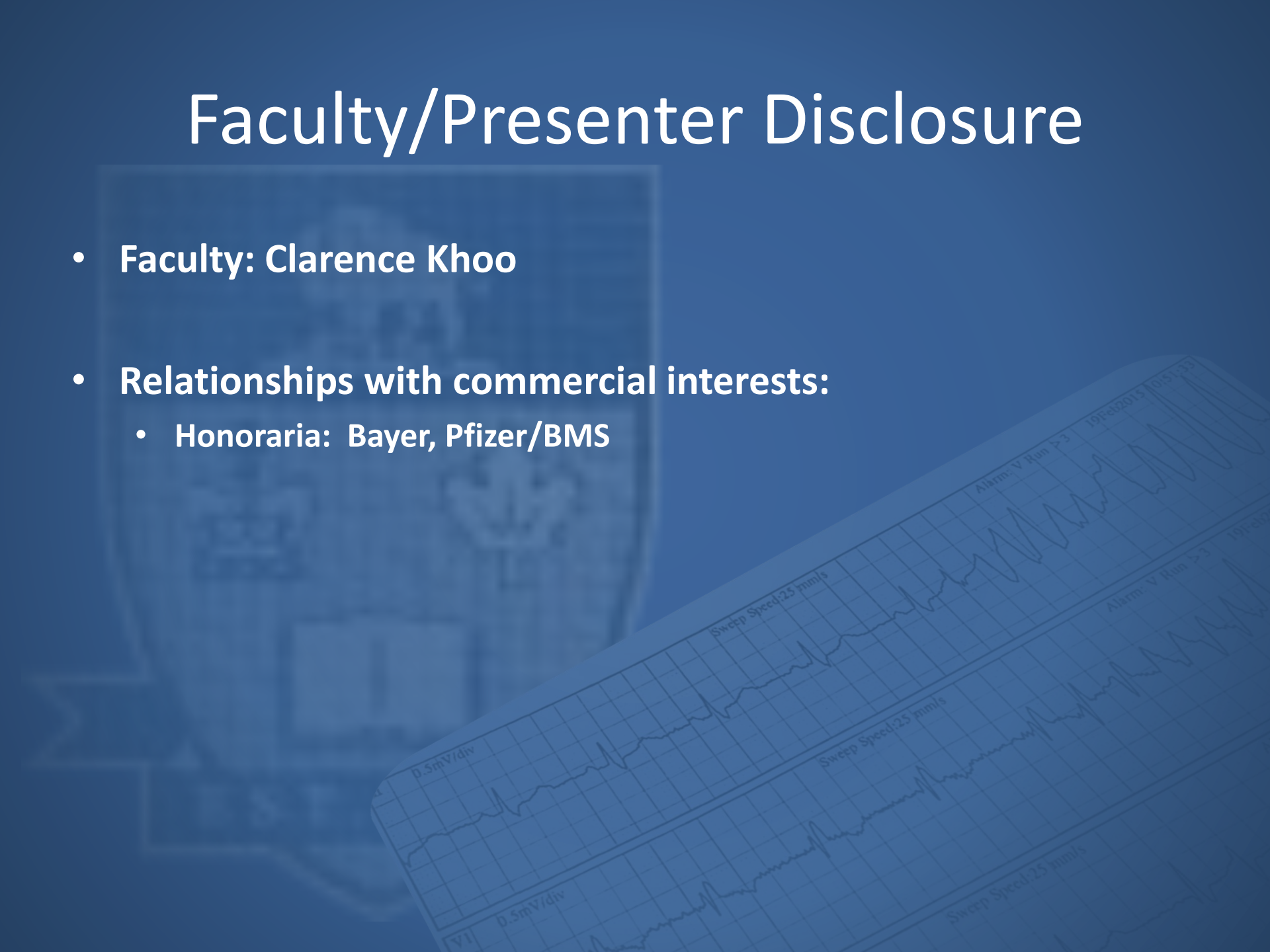
Faculty/Presenter Disclosure

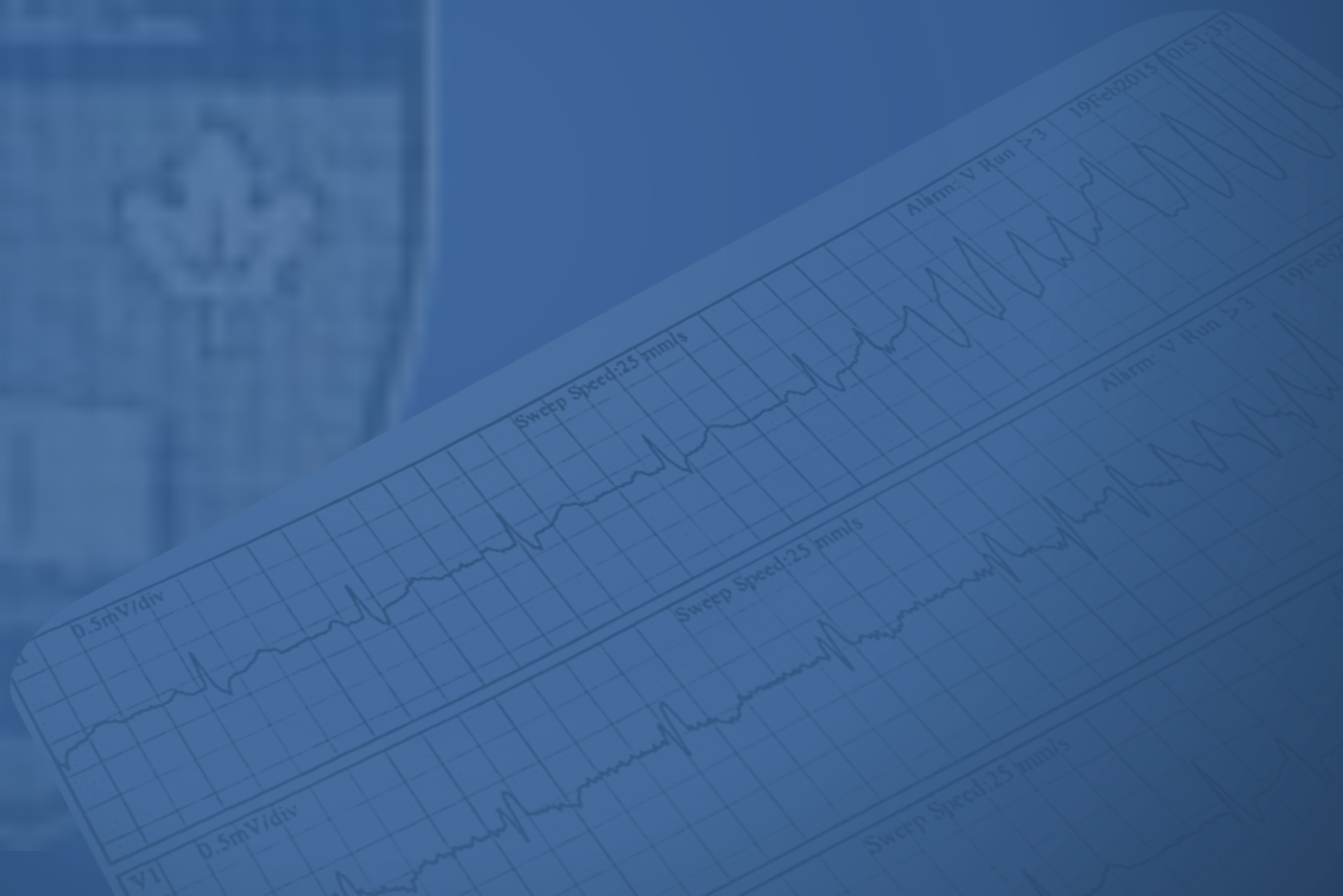
- **Faculty: Clarence Khoo**

- **Relationships with commercial interests:**
  - **Honoraria: Bayer, Pfizer/BMS**



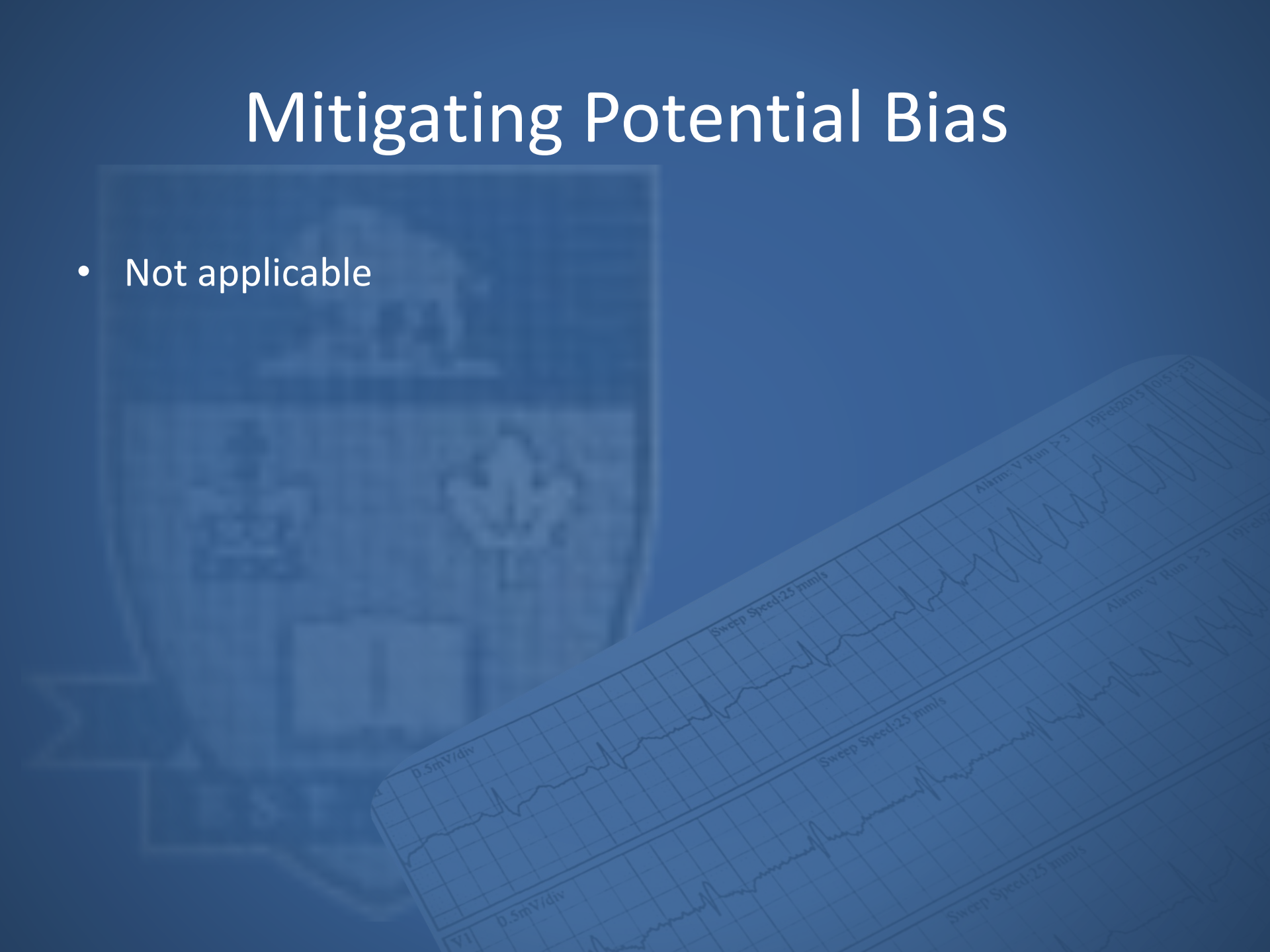
Disclosure of Commercial Support

- **No commercial support**



Mitigating Potential Bias

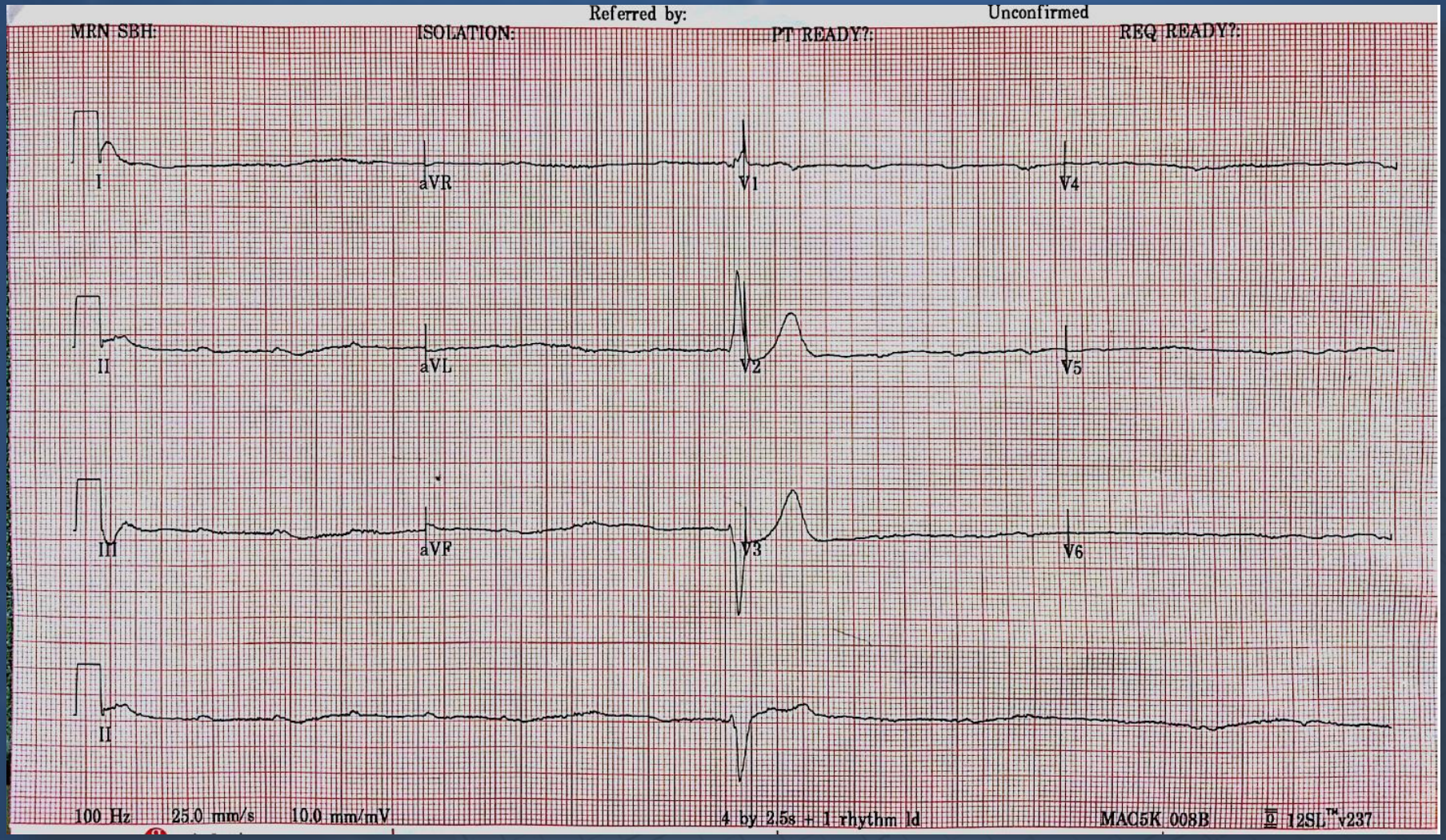
- Not applicable



Objectives

- Develop an approach to the diagnosis and management of common, yet problematic tachy- and brady- arrhythmias
- Appreciate that not all arrhythmias result in the sudden and horrific demise of your patient
- Identify when to keep calm, when to worry, and when to ask for help with an arrhythmic issue

Is this your worst nightmare?



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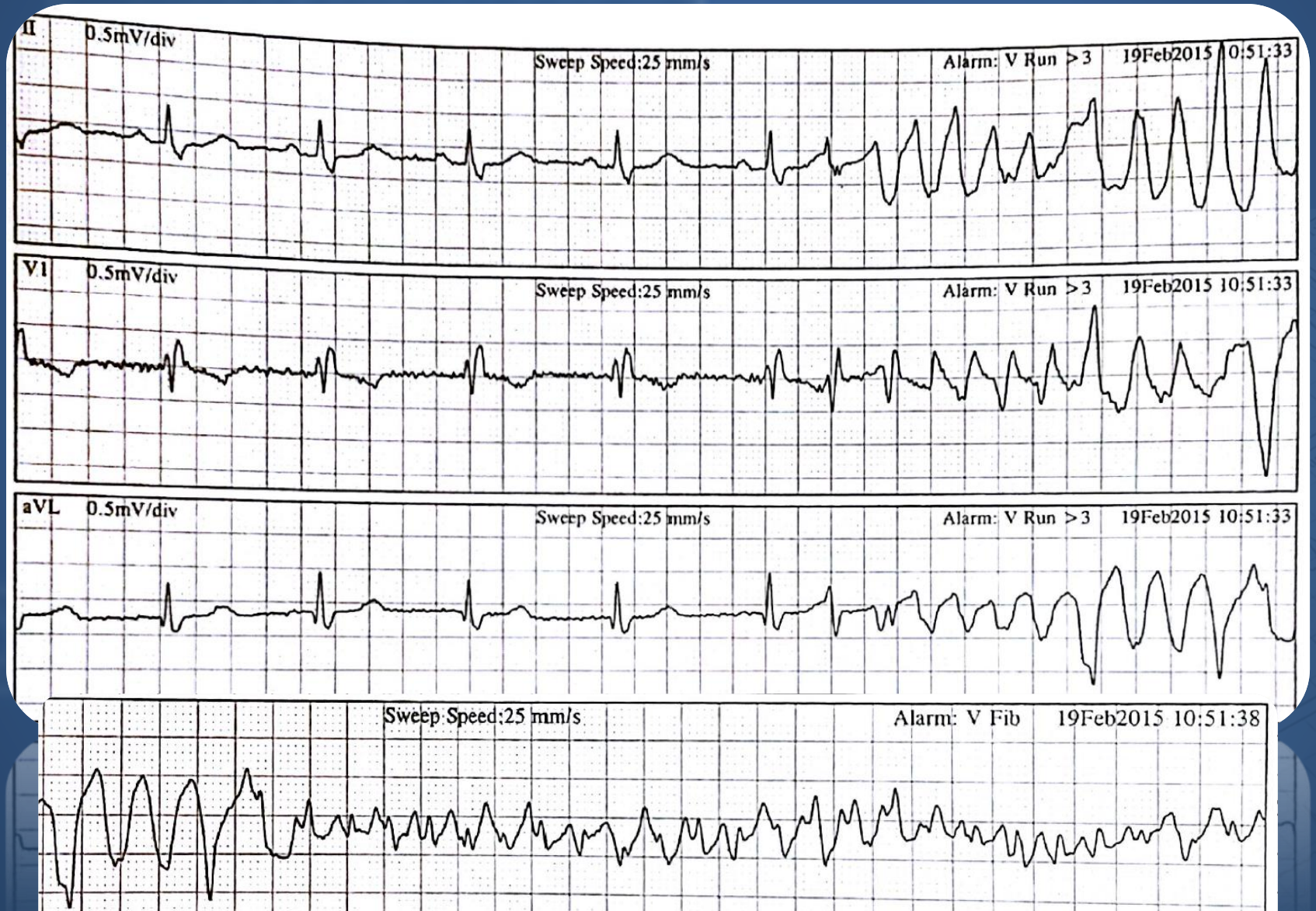

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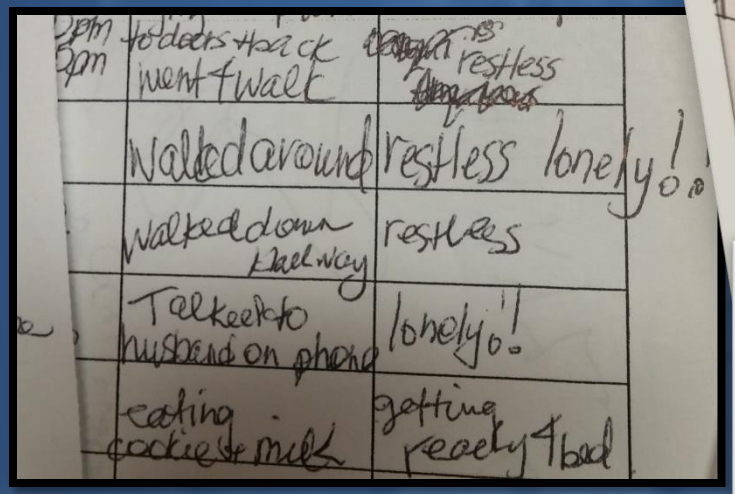
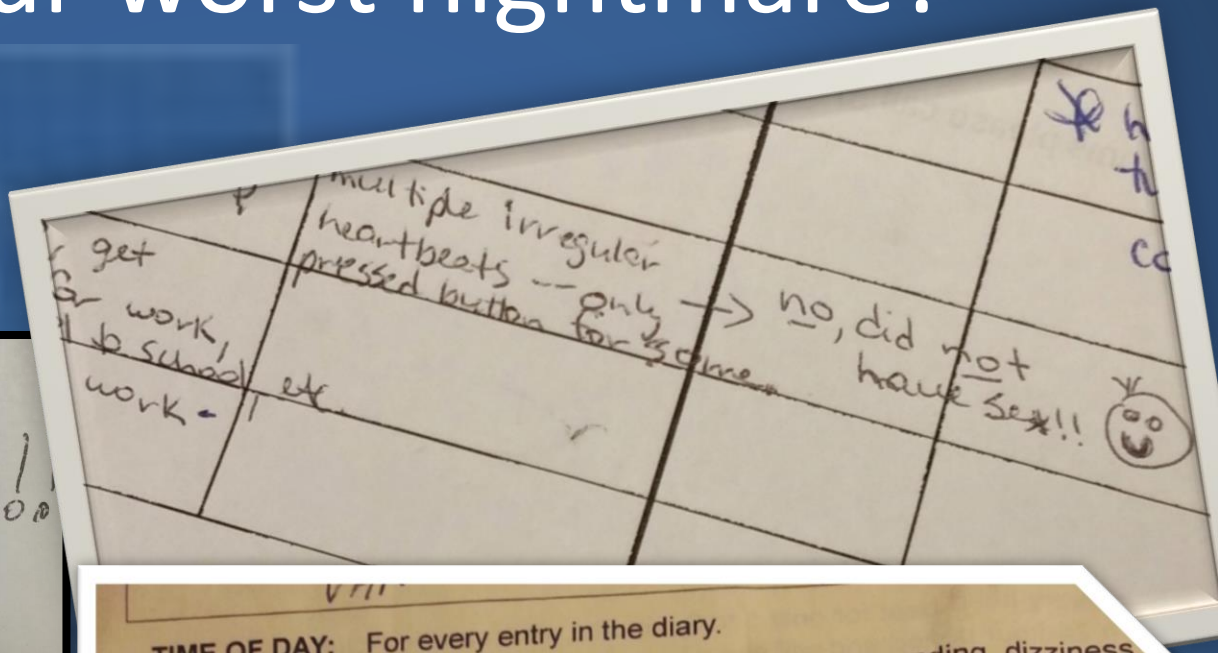
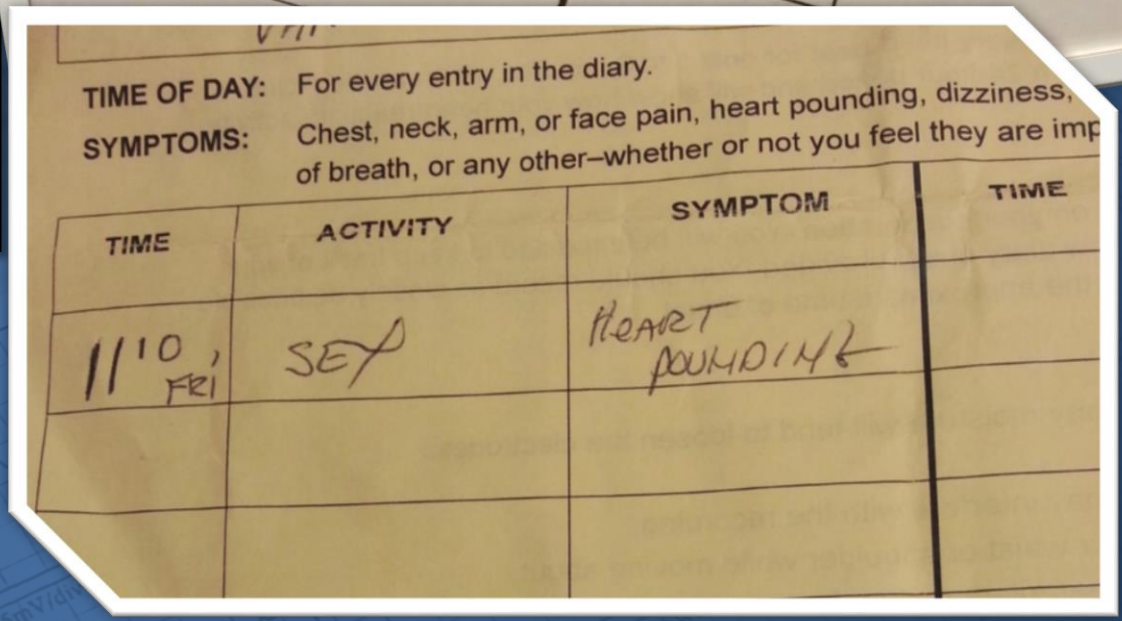


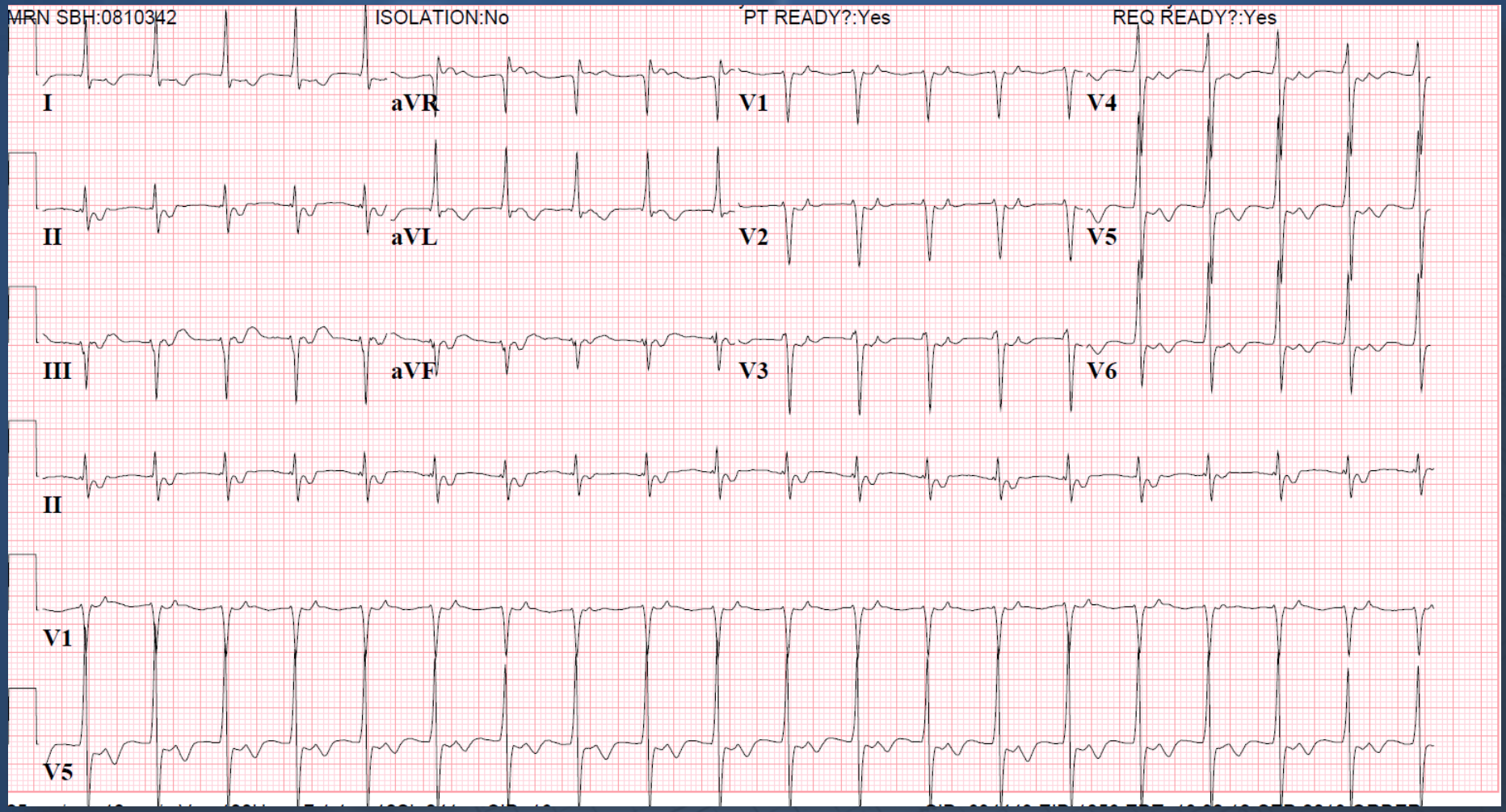




General Tips with Managing Arrhythmias

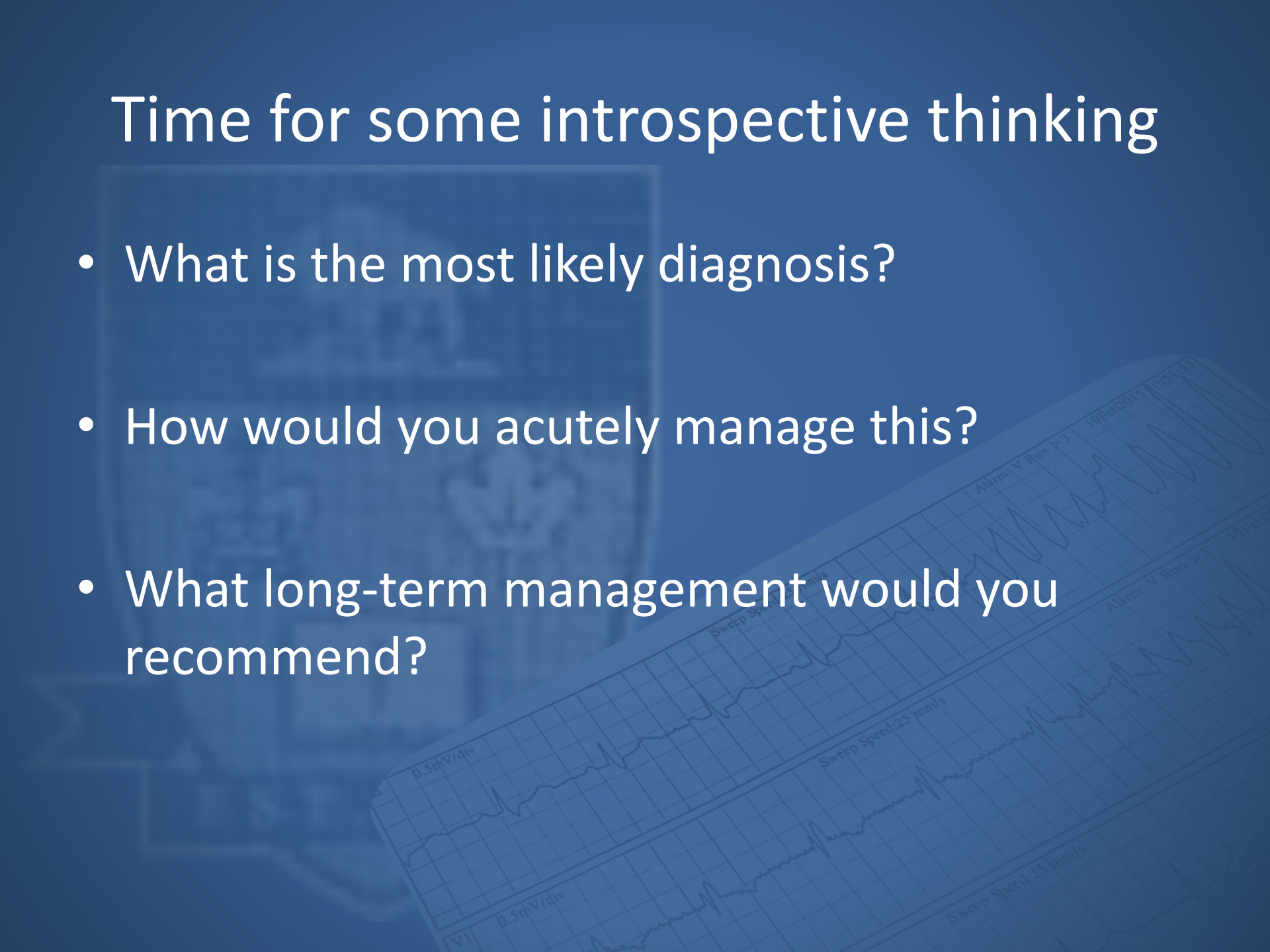
- Don’t panic (at least right away)
- Ensure that patient is haemodynamically stable first
- If possible, get a 12-lead ECG, not just a rhythm strip
- If SVT, try some basic manoeuvres
- Ask for help if unsure

37F from Rankin Inlet with palpitations



Time for some introspective thinking

- What is the most likely diagnosis?
- How would you acutely manage this?
- What long-term management would you recommend?



NARROW COMPLEX TACHYCARDIA - SVT

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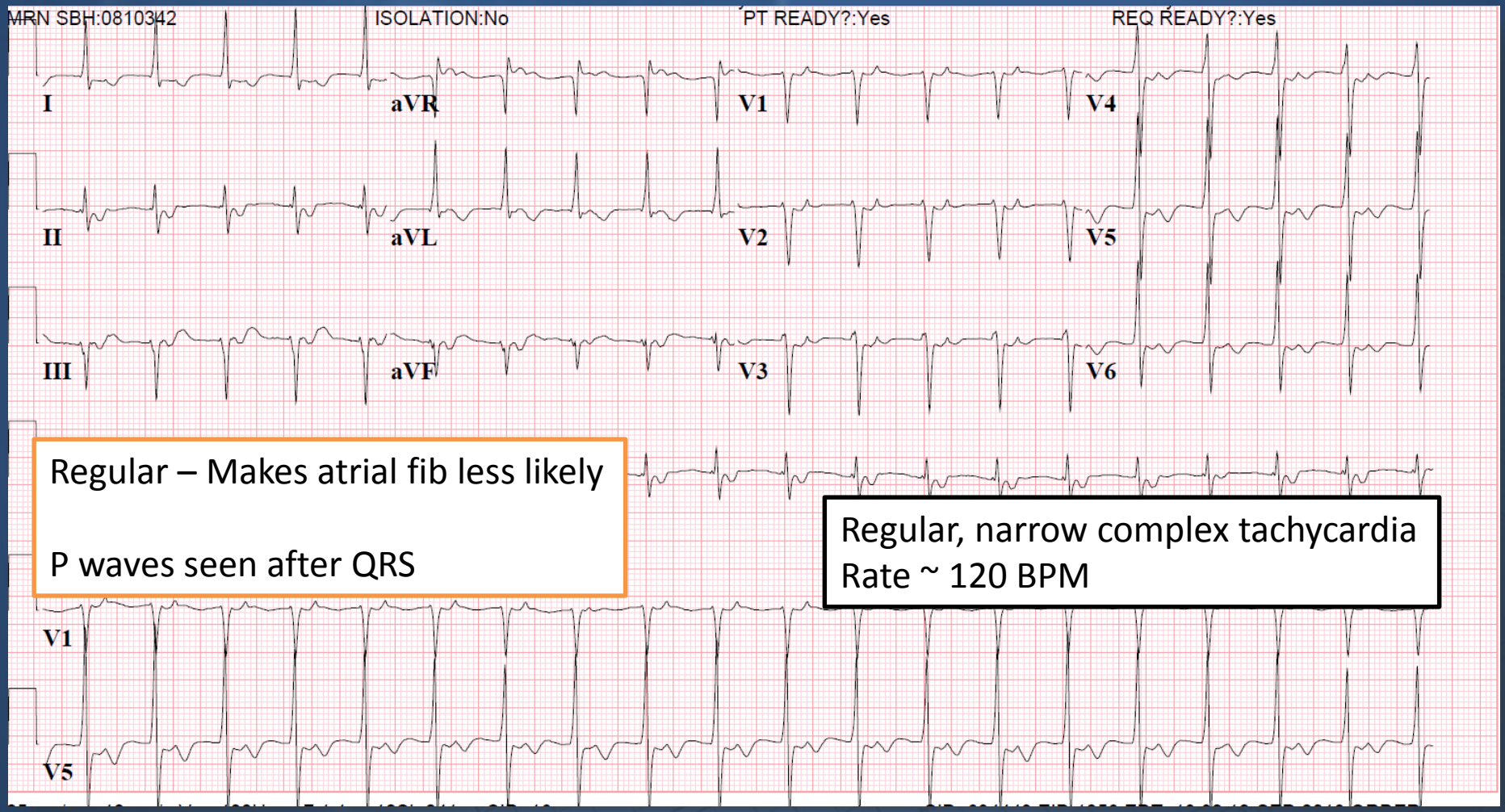
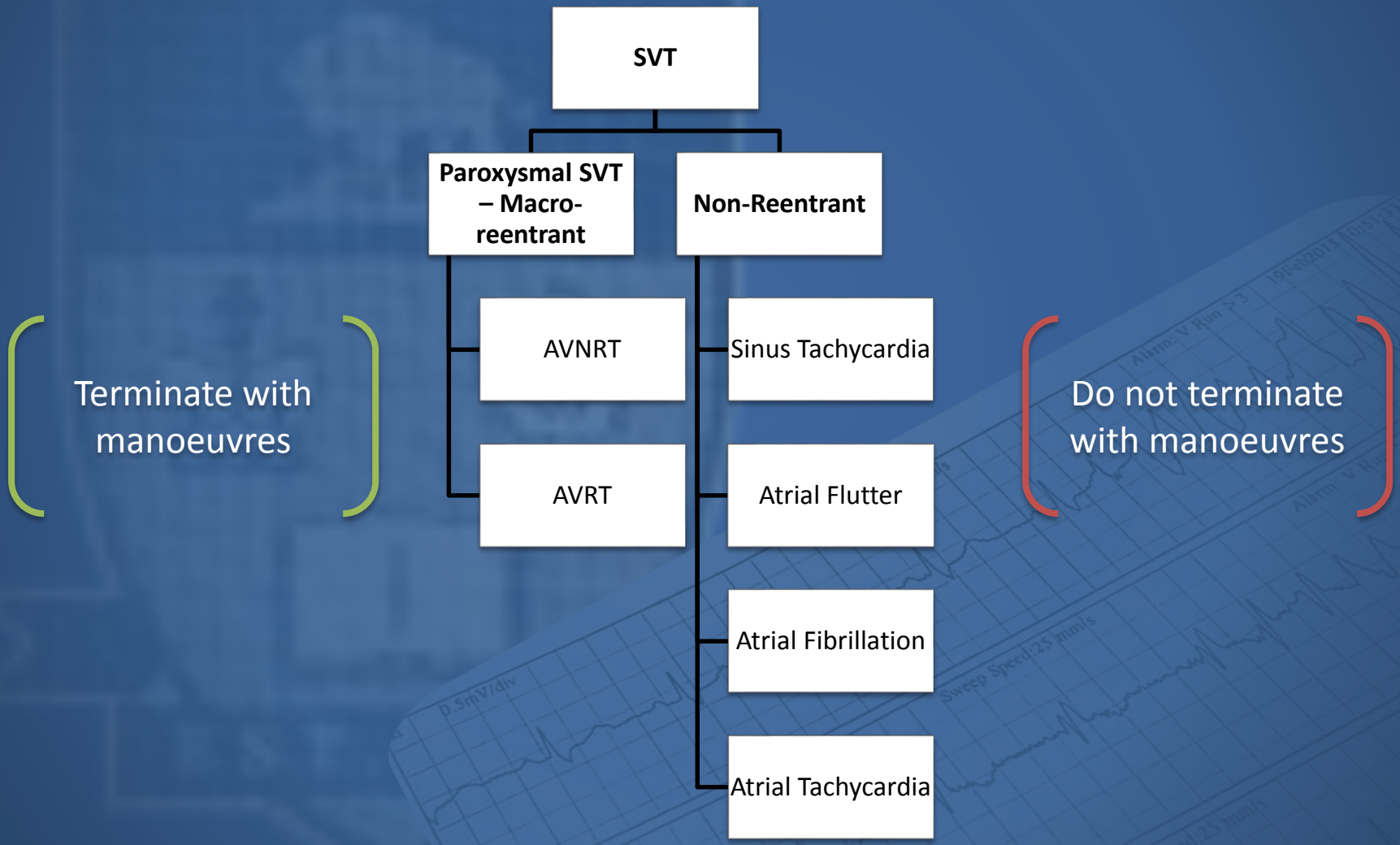


Supraventricular Tachycardia



General Tips about Narrow Complex SVT

- Invariably benign tachyarrhythmias, so do not panic!
- Non-reentrant SVT may not terminate readily, nor do they need to be acutely.
- Some are paroxysmal and may return even after you terminate them acutely → need longer term pharmacotherapy or ablation.

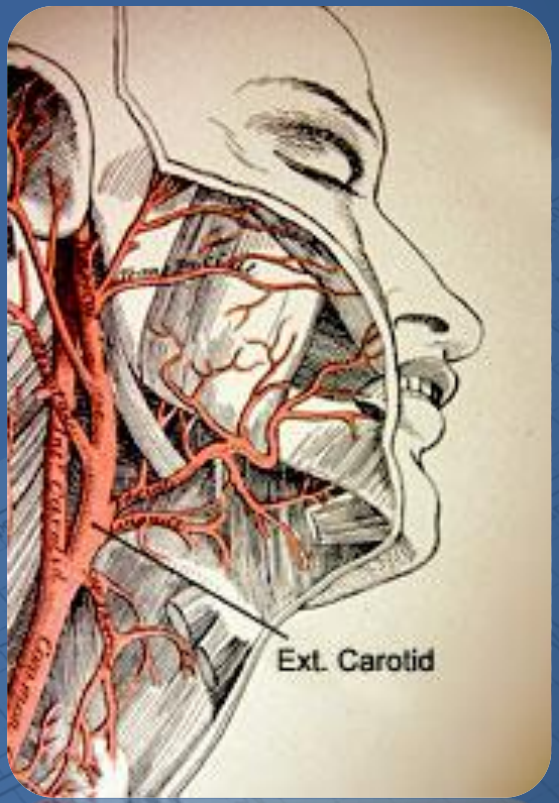
Vagal Manoeuvres

- Vagal manoeuvres are recommended for acute treatment in patients with regular SVT
  - Will terminate re-entrant SVT, help diagnosis of non-reentrant SVT

Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

Carotid Sinus Massage

- Perform in supine position
- Auscultate for carotid bruits before pressing
- Only push on one side at a time
- Push for approximately 5 – 10 sec
- Can pair it up with Valsalva manoeuvre – apply CSM after patient releases Valsalva
  - Use of both techniques sequentially has a 27.7% success rate



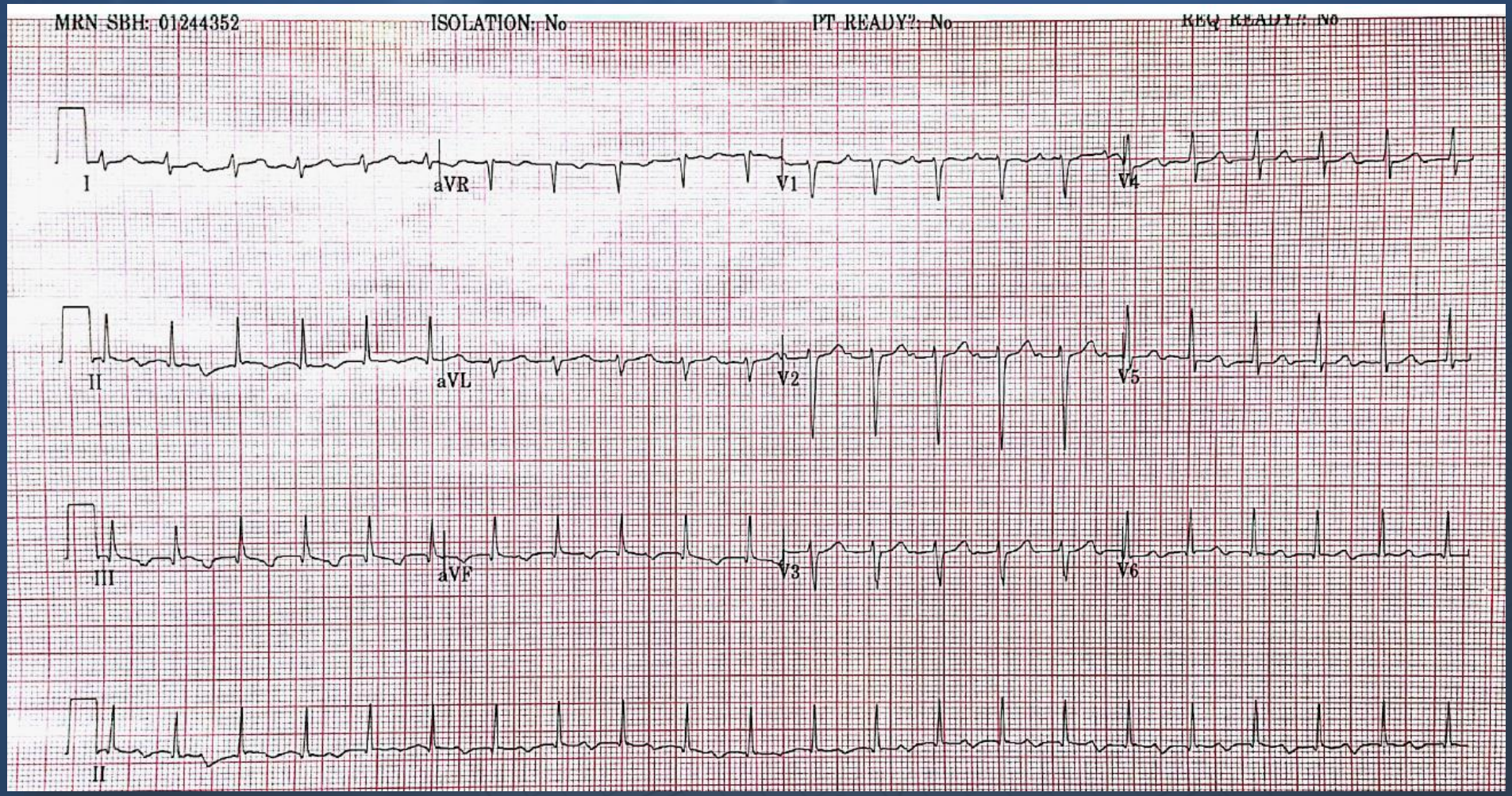
Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

Adenosine

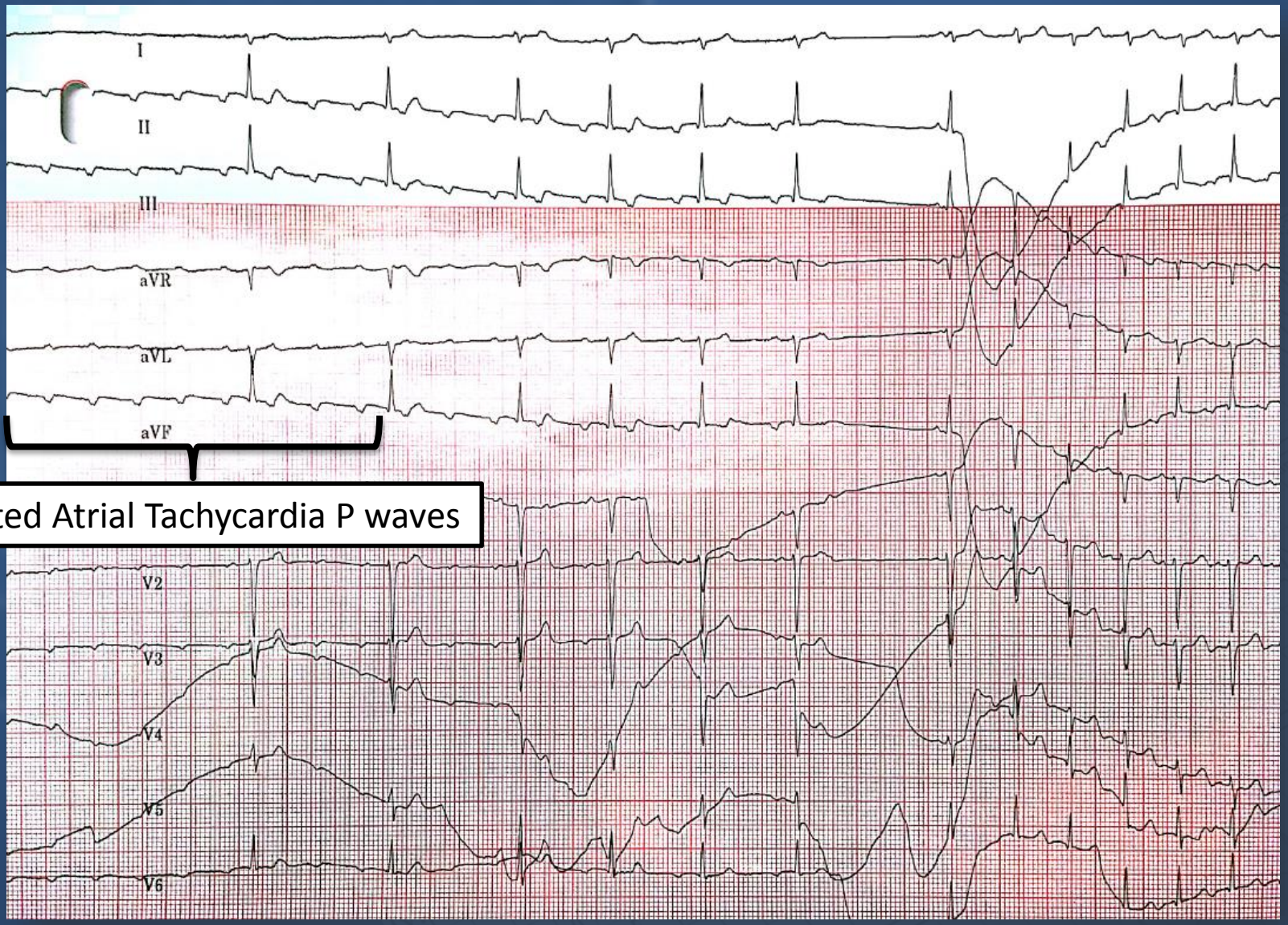
- Adenosine is recommended for acute treatment in patients with regular SVT
  - Success rates of 78 – 96%
  - AVNRT and AVRT will terminate
  - Helps unmask underlying atrial flutter or atrial tachycardia

Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

Regular SVT

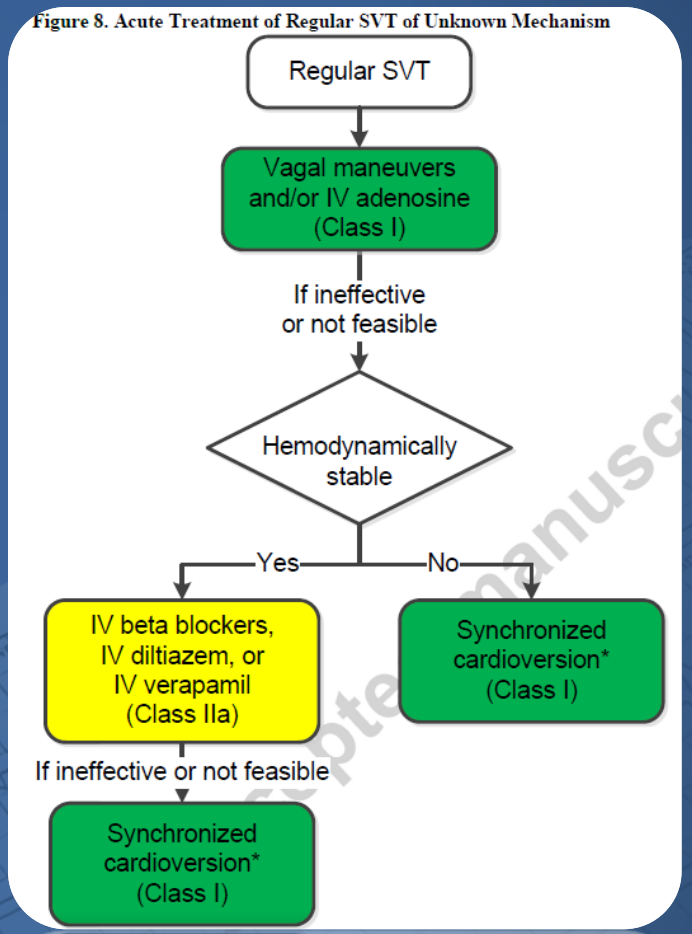

MRN SBH: 01244352
ISOLATION: No
PT READY?: No
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Adenosine Administered



Beta blockers and Calcium Channel Blockers

- IV diltiazem or verapamil (IIa B) or IV beta blockers (IIa C) may be used
  - Adenosine is still preferable since it is not only therapeutic, but diagnostic



Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

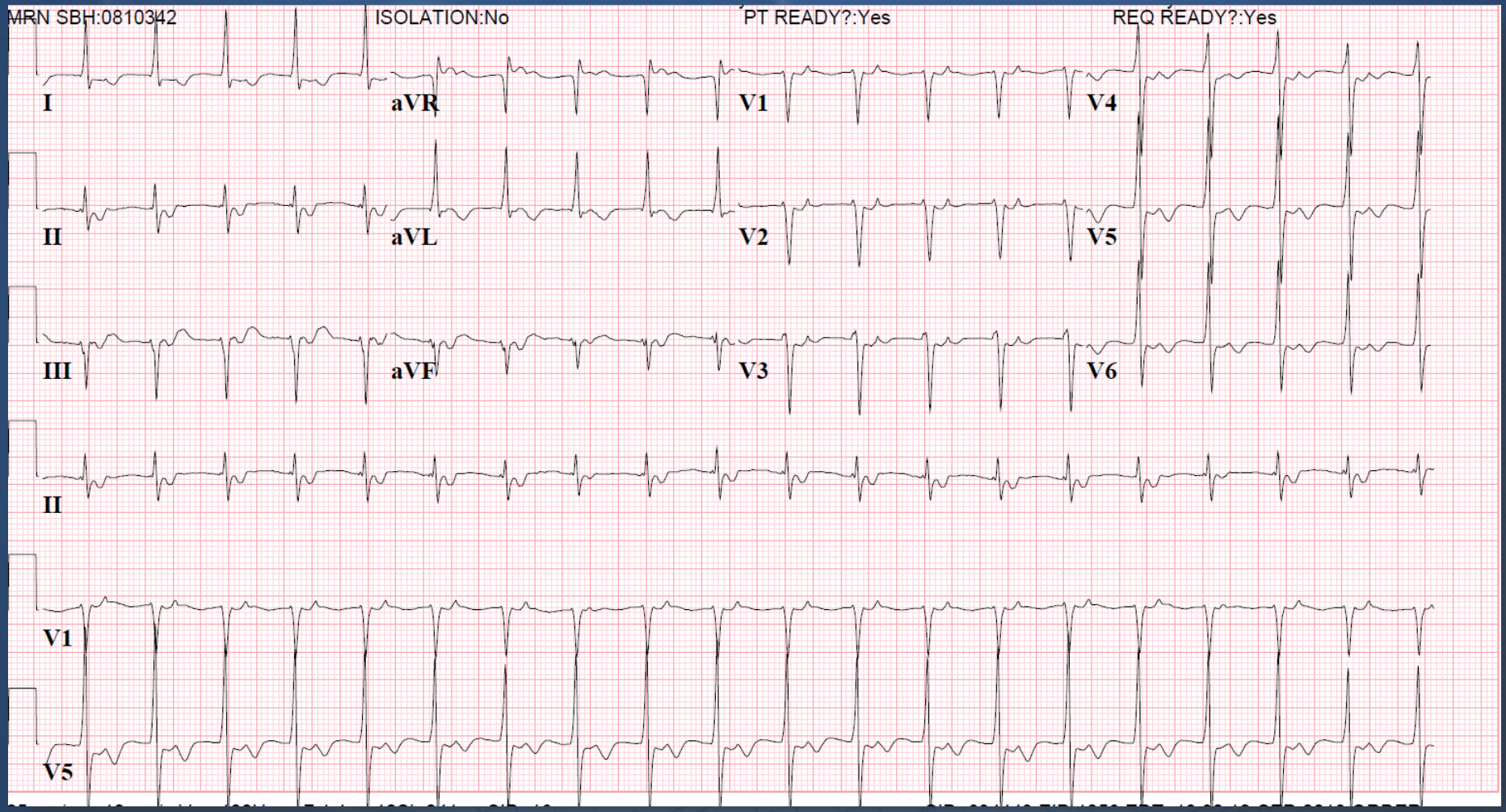
Long-Term Management

- Re-entrant SVT are benign so reassure patients of this

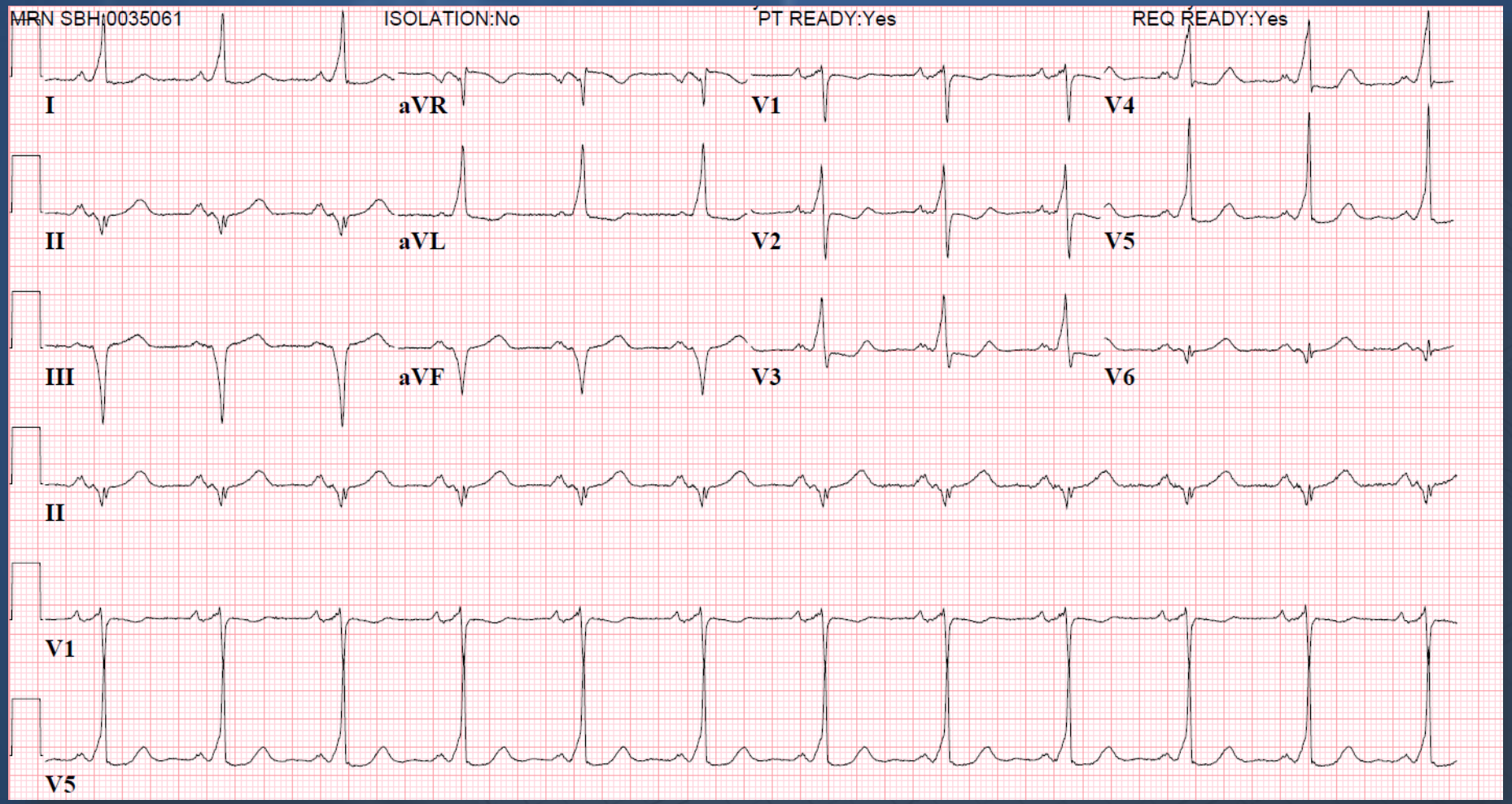
- Options for long-term management include:
  1. **Conservative:** Valsalva prn
  2. **Daily pharmacotherapy:** Beta blockers or Verapamil/Diltiazem
  3. **Referral for EP study & ablation**
     - Potential cure for young patients, or those with +++ symptoms or episodes

Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

37F from Rankin Inlet with palpitations

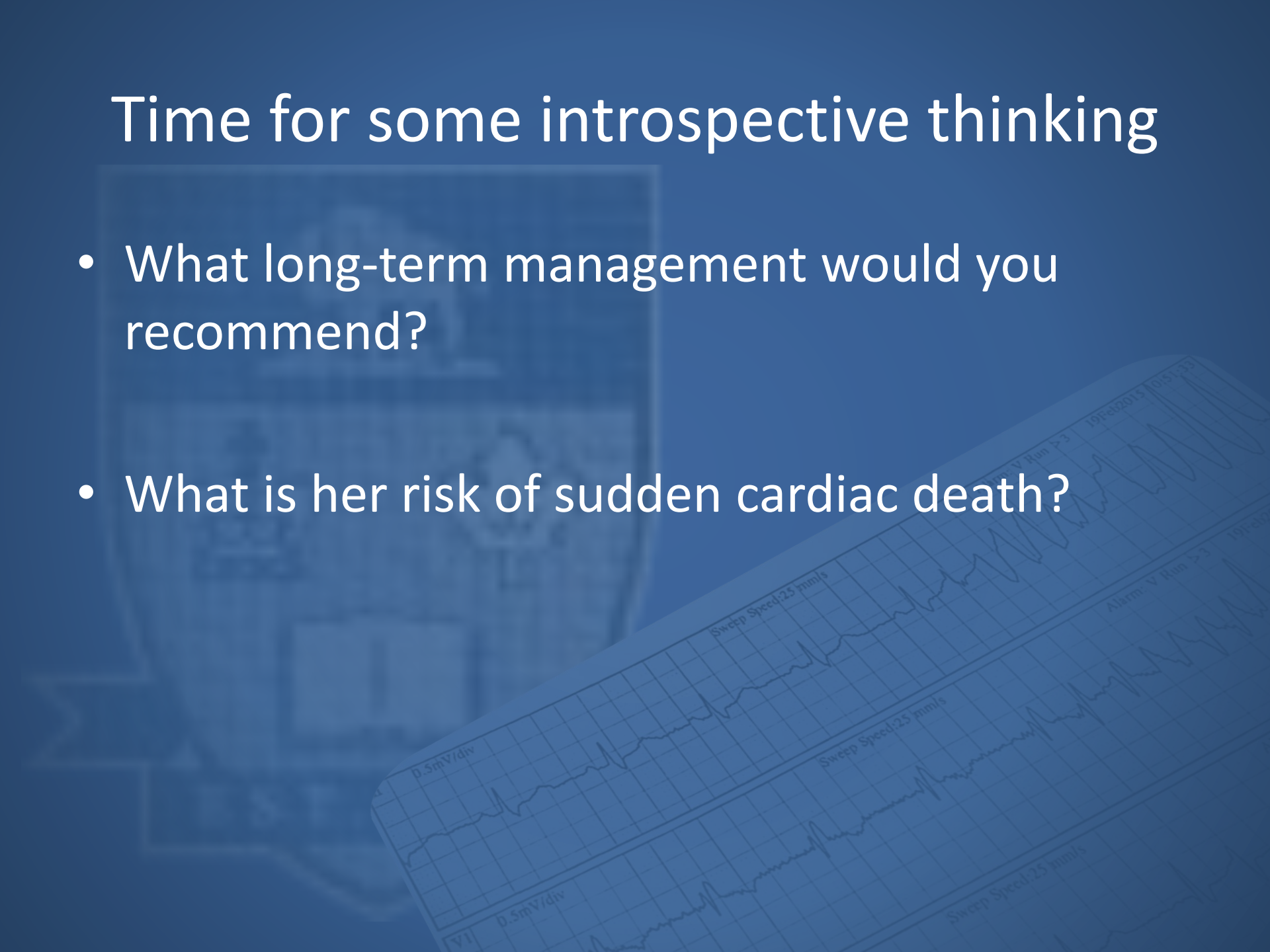


Adenosine 6mg IV x 1 is administered



Time for some introspective thinking

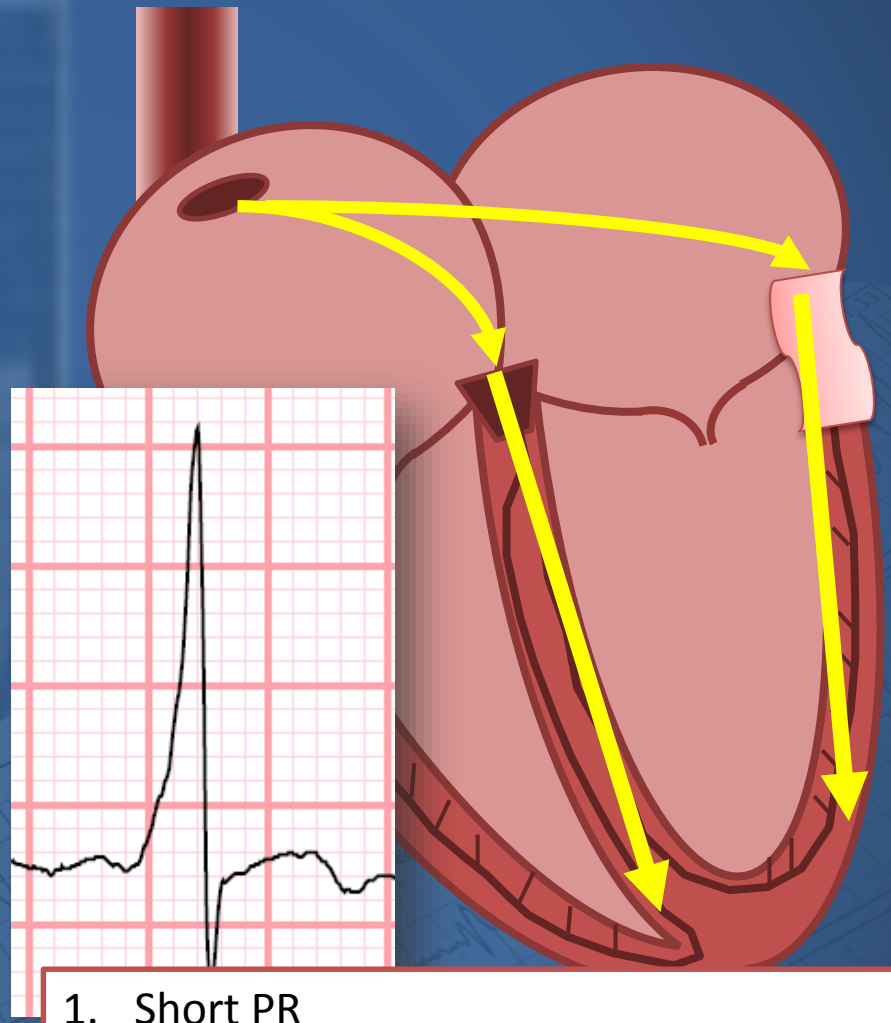
- What long-term management would you recommend?
- What is her risk of sudden cardiac death?



ACCESSORY PATHWAYS & WPW

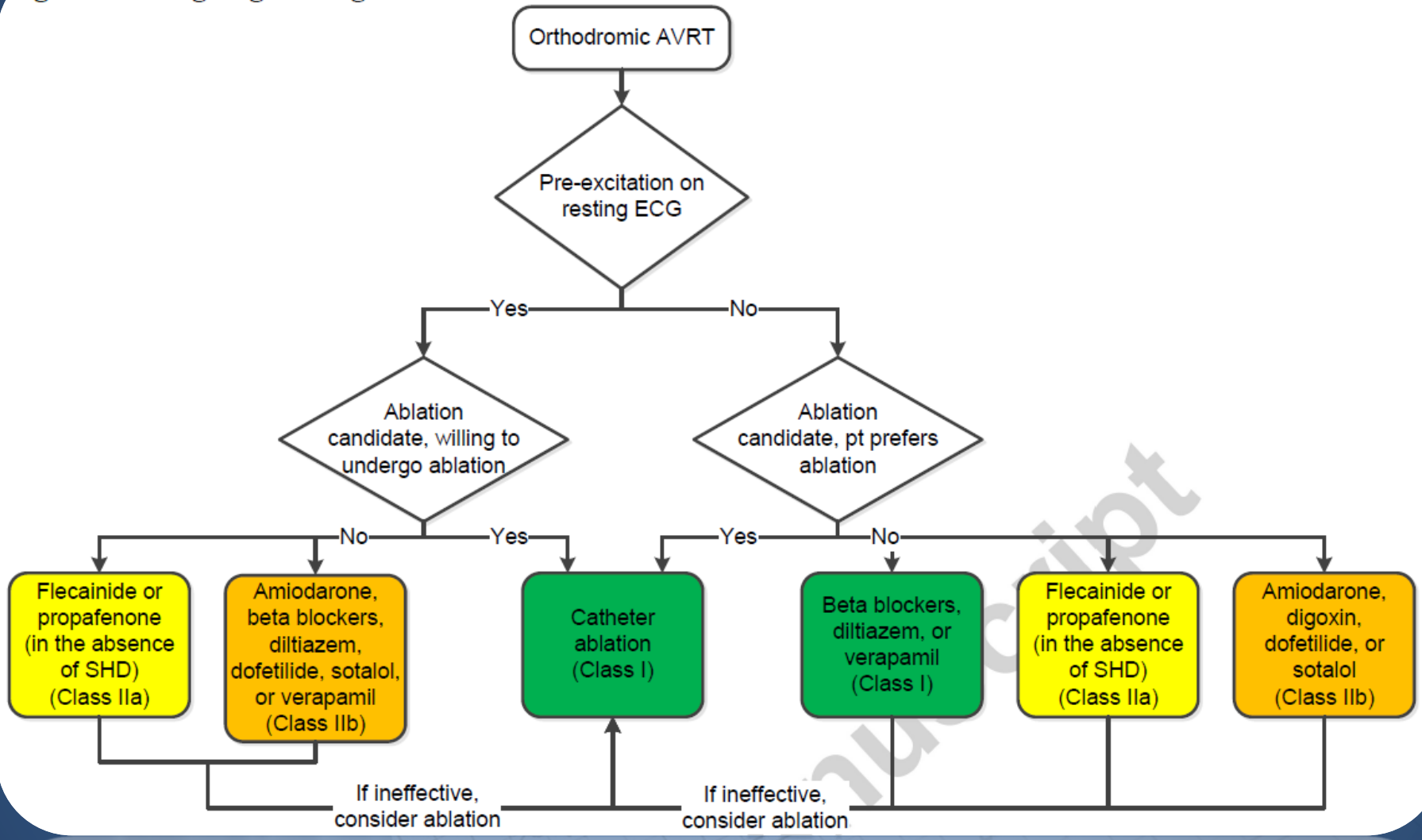
Manifest Accessory Pathways

- During sinus rhythm, conduction occurs through *both AV node and accessory pathway* simultaneously
- Conduction down the accessory pathway produces a slur to the upstroke of the QRS = **"Delta wave"**



1. Short PR
2. "Delta" wave = Accessory pathway
   "Pre-excitation"

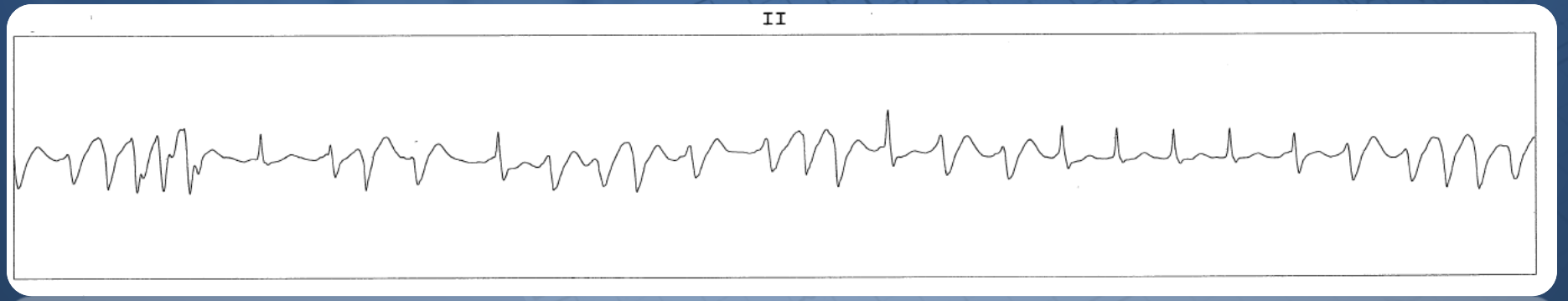
Figure 15. Ongoing Management of Orthodromic AVRT



Page et al., 2015 ACC/AHA/HRS SVT Guidelines.

Atrial Fibrillation and WPW

- If an accessory pathway is present, it may conduct rapidly during AF, at times causing ventricular rates exceeding 200 – 300 BPM.
- This can lead to **Ventricular Fibrillation** and sudden cardiac death
- Irregularly irregular wide complex tachycardia +/- previous ECG's showing manifest pre-excitation



Management of AF with WPW

- In patients with AF and a rapidly conducting accessory pathway **DO NOT GIVE AV NODAL BLOCKING AGENTS** (i.e. beta blockers, verapamil/diltiazem, digoxin)
  - They will allow for preferential conduction down the accessory pathway and precipitate badness

- *Acutely:* Safest therapy is cardioversion (alternative is IV procainamide)

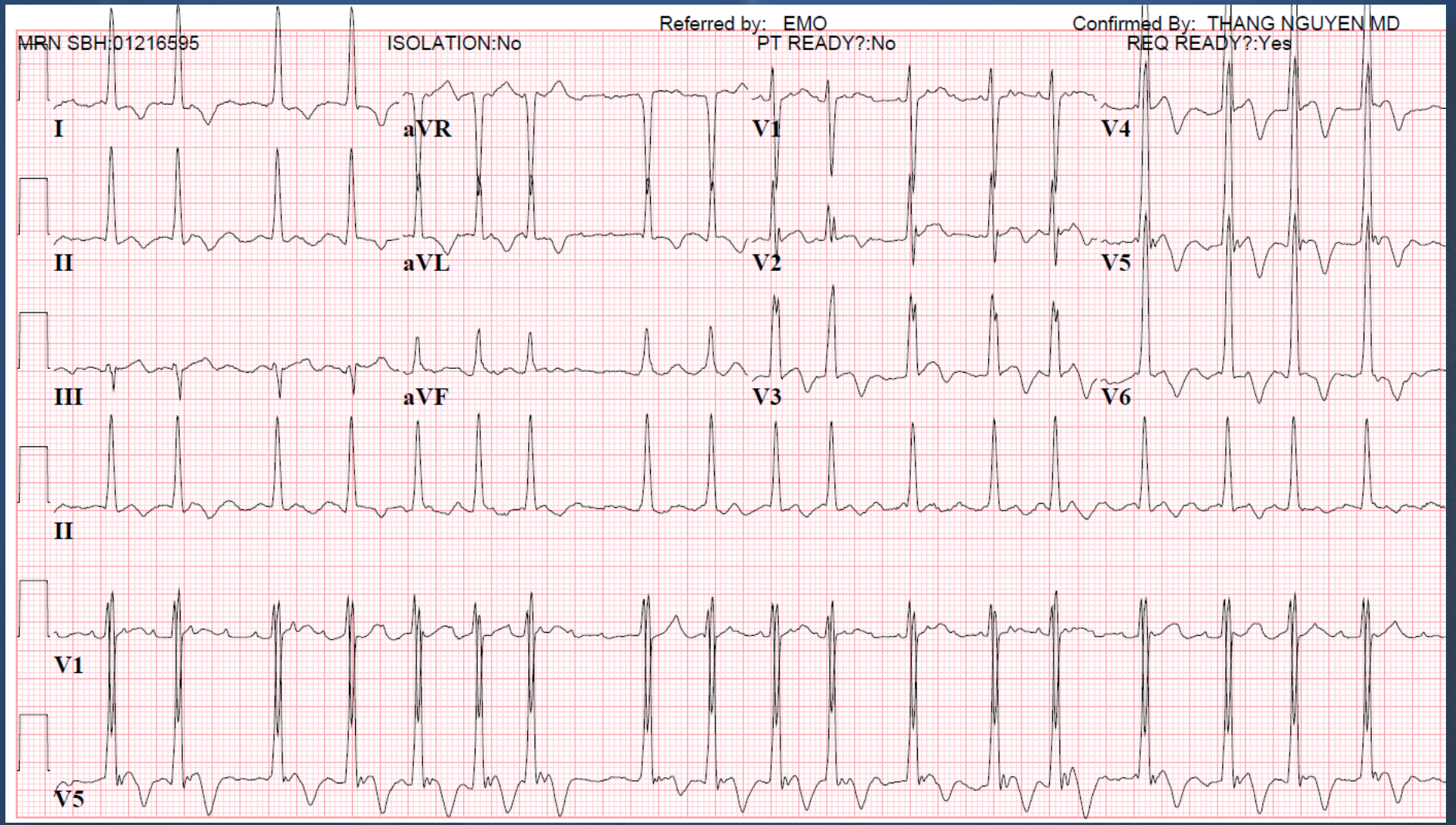
- *Chronically:* Electrophysiology study with ablation of accessory pathway is paramount

Should I worry about my asymptomatic patient manifest pre-excitation?

- If any palpitations, syncope etc., then refer to cardiology/EP for assessment
- First symptom in rare subset of patients may be unheralded syncope or cardiac arrest
  - Risk of sudden death in truly asymptomatic patients is low, in the range of 0.1% annual risk
- May refer to cardiology/EP for risk stratification or ablation in high risk occupations (athletes, pilots, etc.)

Cohen et al., Heart Rhythm. 2012. 9(6): 1006 – 23.
Klein et al., Circulation: Arrhythmia and Electrophysiology. 2009; 2: 97-99.

Nightmare?

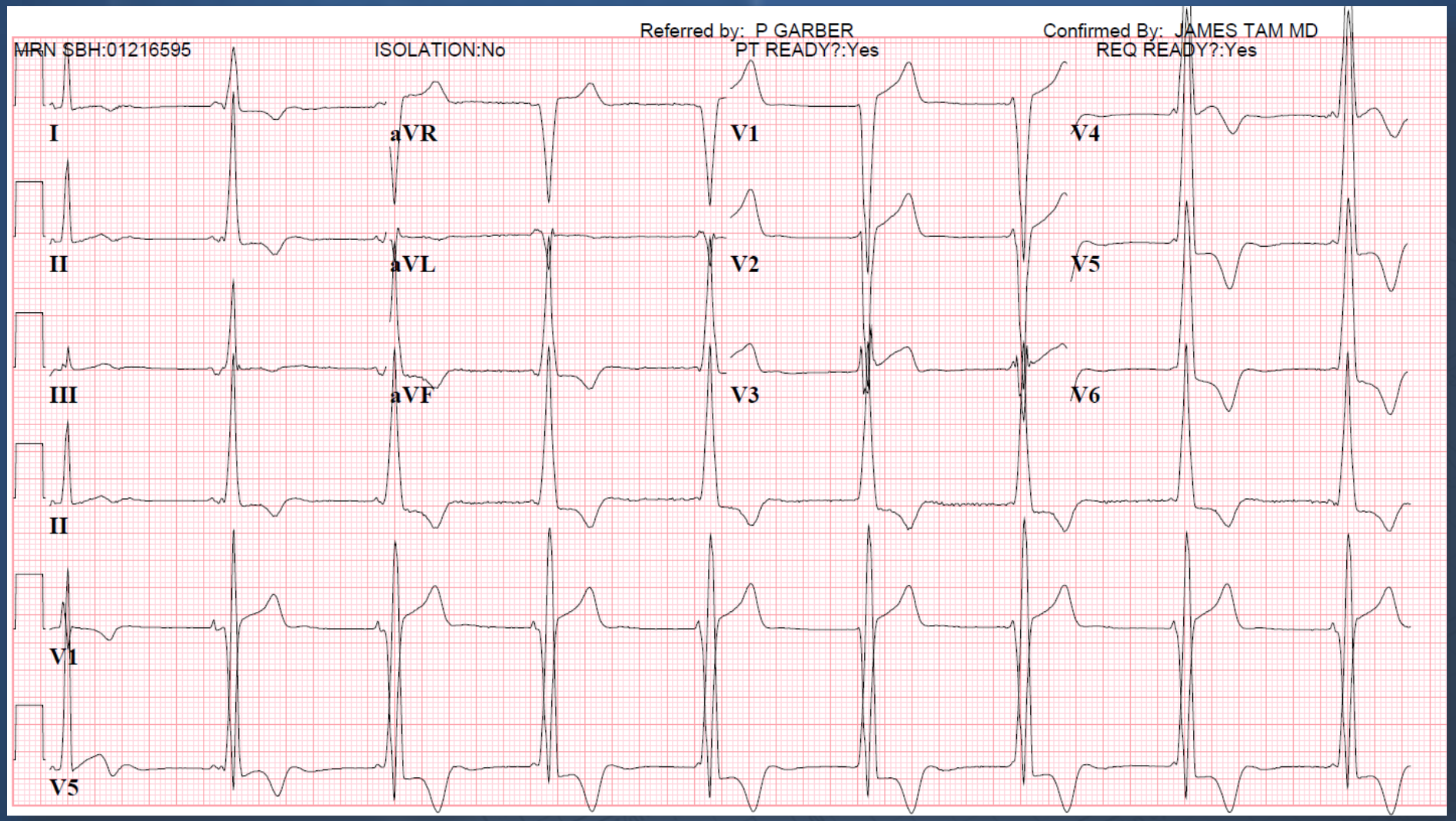

MRN SBH:01216595
ISOLATION:No
Referred by: EMO
PT READY?:No
Confirmed By: THANG NGUYEN MD
REQ READY?:Yes
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II
V1
V5

How not to treat AF…

- 73F managed for AF as an outpatient on sotalol, receiving elective cardioversion in ER

- Returned into AF while still in ER
  - Given several metoprolol IV boluses
  - Metoprolol 100 mg PO x 1
  - Amiodarone 400 mg IV x 1
  - Diltiazem 30 mg PO x 1
  - All within a few hours of each other

Patient subsequently on IV dopamine and transferred for temporary pacemaker…



TIPS FOR RATE CONTROL OF ATRIAL FIBRILLATION

General Pointers

- If haemodynamically stable, you can take your time with controlling the ventricular rate. In fact, if patient has decent outpatient follow-up, can be done largely in the office.
- Ideally want to start PO meds with a reasonably long half-life so that the rate stays down as opposed to short effect of IV meds.
- If in a rush, use PO meds with a slightly shorter half-life (i.e. metoprolol as opposed to bisoprolol) to allow for more rapid up-titration.
- The target HR is fairly liberal – **less than 100 BPM**. It does not need to be perfect!

Beta blockers or Calcium Channel Blockers?

- Beta blockers likely safe most of the time. Beware of the use of calcium channel blockers in CHF or known LV dysfunction.

- Calcium channel blockers often result in more prompt rate control in ER setting, but may be associated with more hypotension

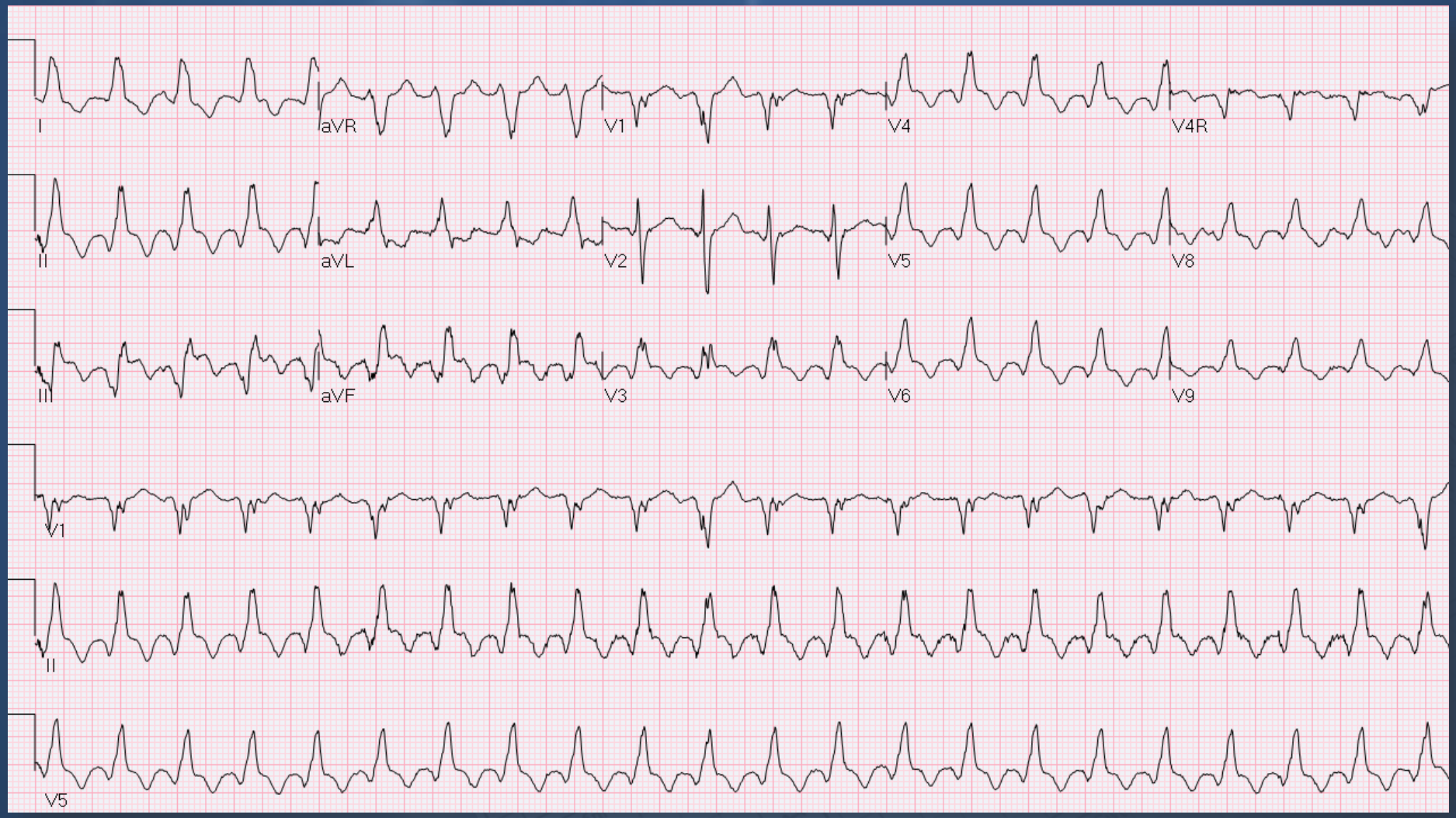
Martindale et al., Eur J Emerg Med. 2015. 22: 150 – 4.

What if ventricular rate is still rapid on a single agent?

- **Digoxin**
  - use in normal renal function at lowest dose possible – 0.0625 – 0.125 mg daily may be sufficient for most and will act synergistically with other rate control agents.
  - Do not titrate to a digoxin level!
- **Combined beta blocker / CCB**
  - Use with caution as may cause marked sinus bradycardia, heart block in some patients
- **Amiodarone**
  - May occasionally be used for its rate control effects in select patients
- **Pacemaker & AV node ablation**
  - Reasonable option in older patients with difficult to control rates or significant intolerance to medications

Edoute et al., J Cardiovasc Pharmacol. 2000. 35(4): 556 – 9.

66M with chest pressure, SBP 106 mmHg



VENTRICULAR TACHYARRHYTHMIAS

Initial Management

- If patient is unstable, then will need to start ACLS.
  - Synchronised DC cardioversion / defibrillation.
- If patient is stable, then there is no urgency to act or to rush straight to cardioversion.
  - Try to determine if rhythm is truly VT or not.
  - Obtain a 12 lead ECG if possible as opposed to just a rhythm strip.

Ways to differentiate SVT vs. VT

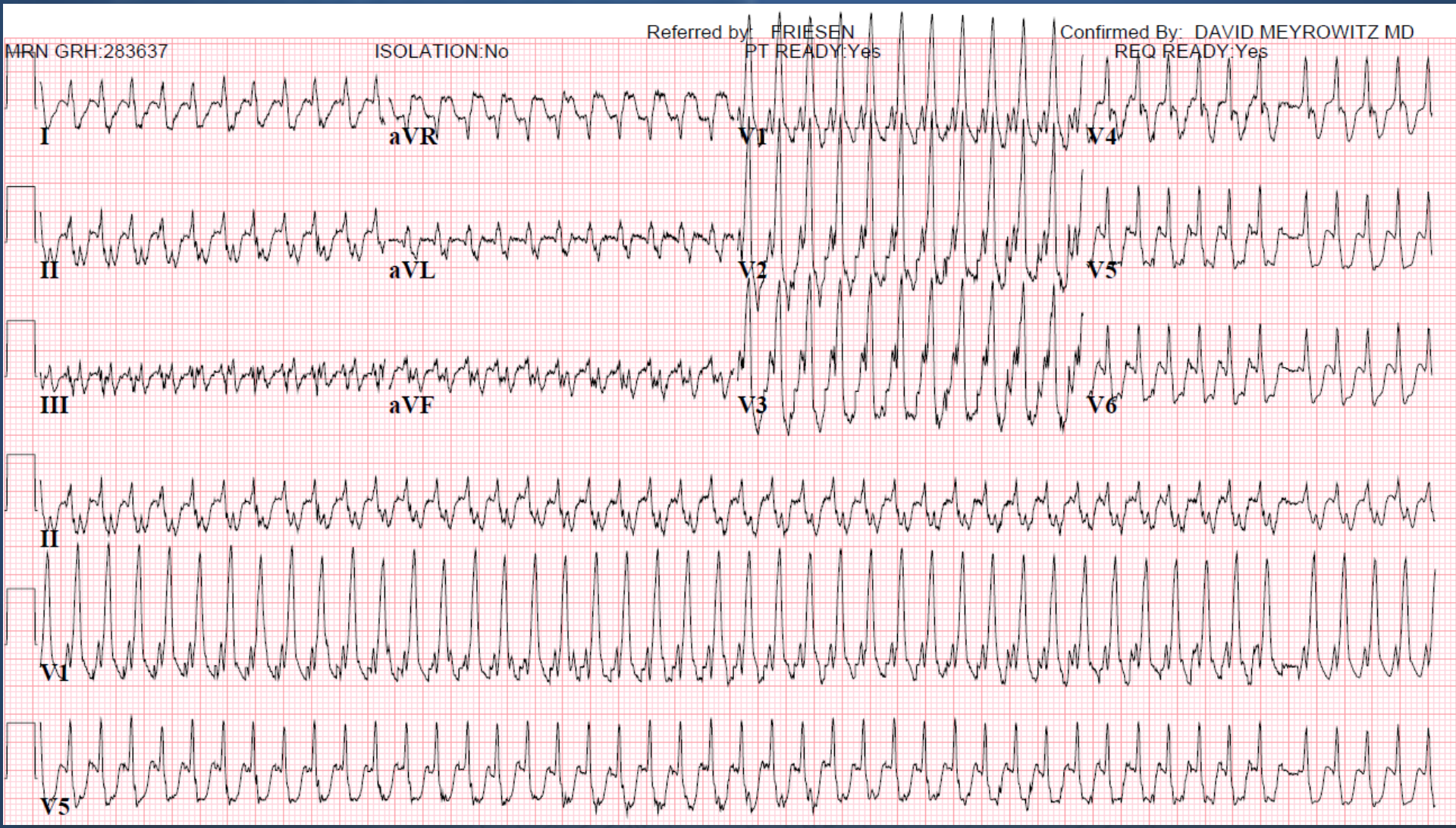
- Patient characteristics
- Baseline ECG
- Clues on ECG:
  - AV dissociation, capture/fusion beats, morphology criteria
- Adenosine
  - As per AHA ACLS guidelines
  - Monomorphic, regular, haemodynamically tolerated WCT – may help to clarify diagnosis

Diagnosing Monomorphic VT

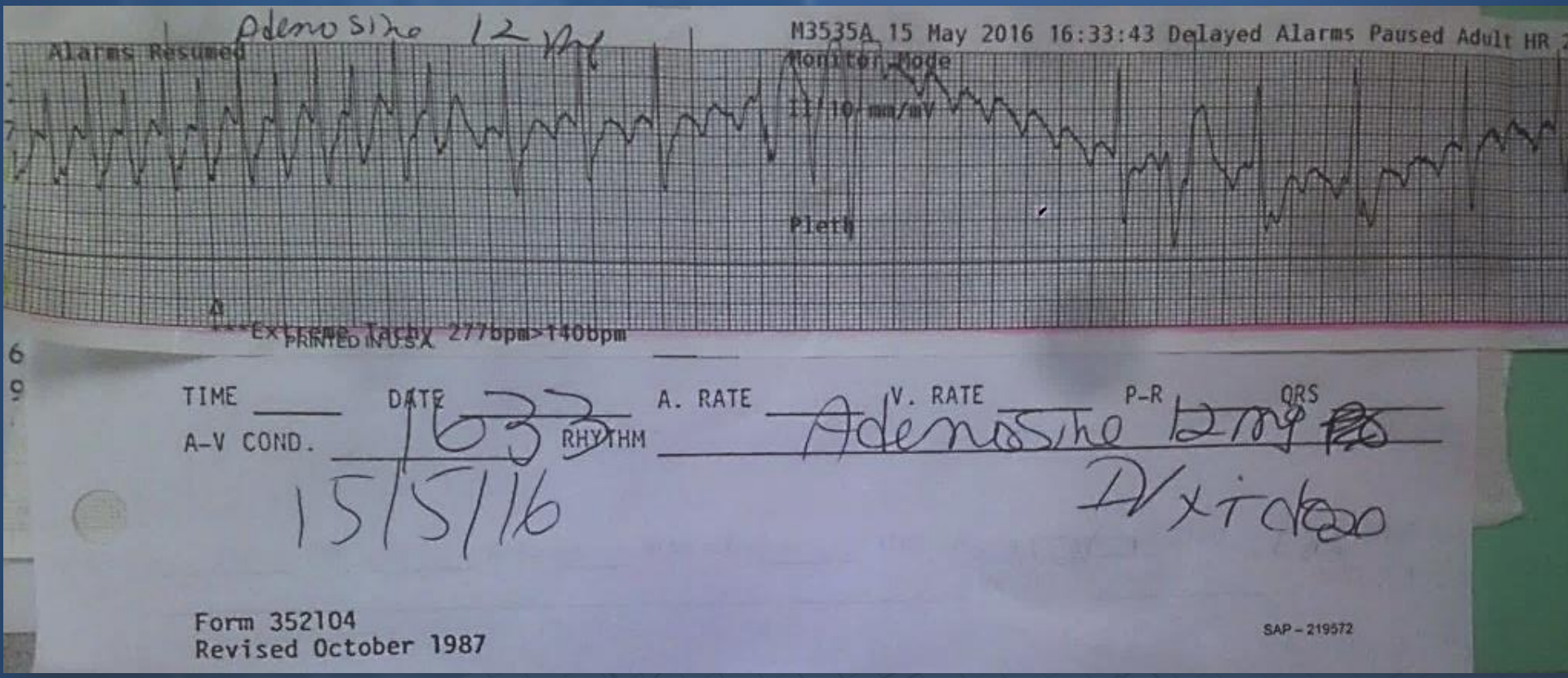
- Clinical history of patient can provide clues as to whether it is more likely VT or SVT
  - History of structural heart disease, MI, etc. makes VT much more likely
  - The absence of syncope or hemodynamic instability *does not* rule out VT

More likely SVT	More likely VT
Young	Older
Previously healthy	Previous history of MI, cardiac disease

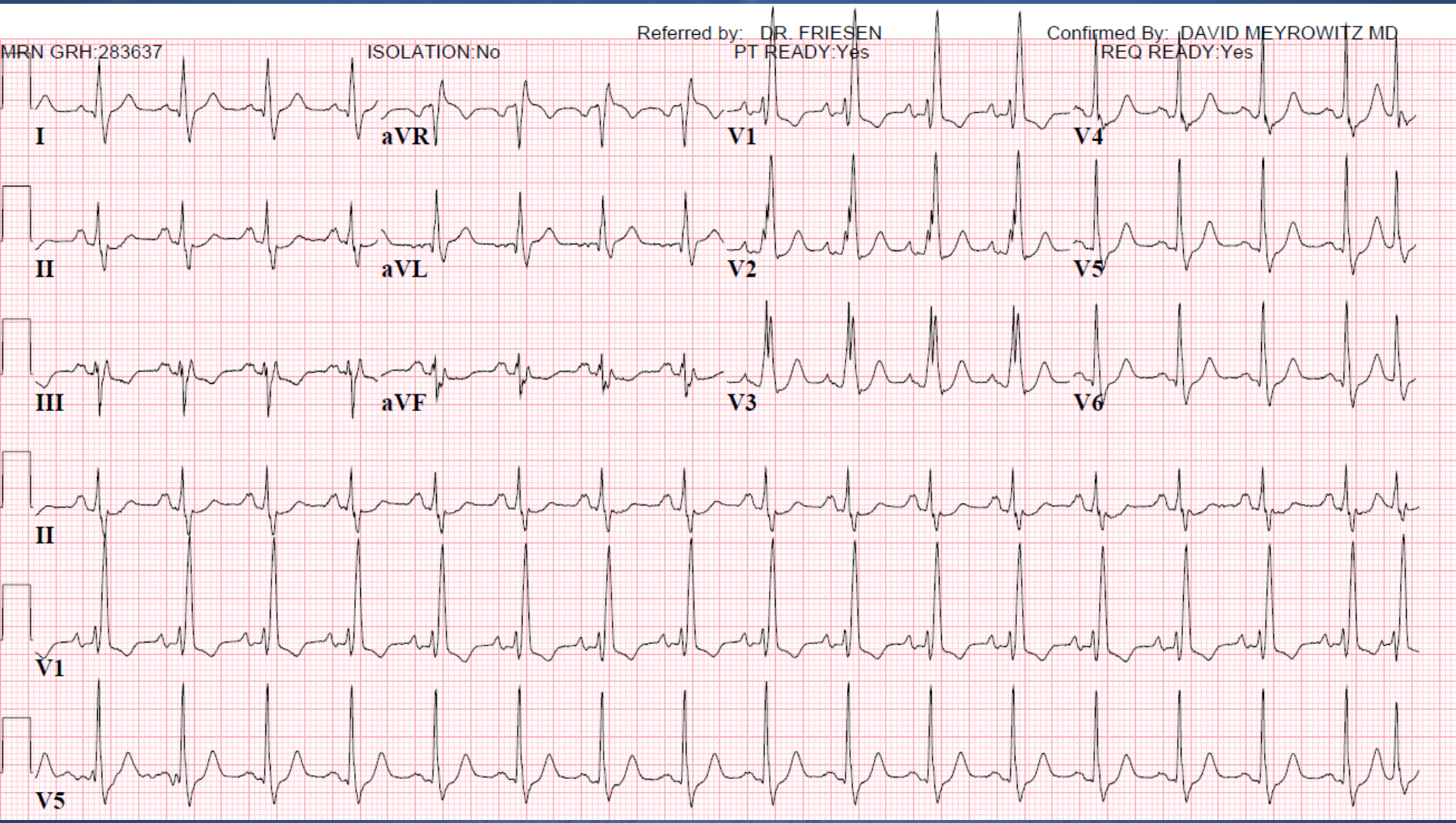
Differential?



Adenosine 12 mg x 1 IV

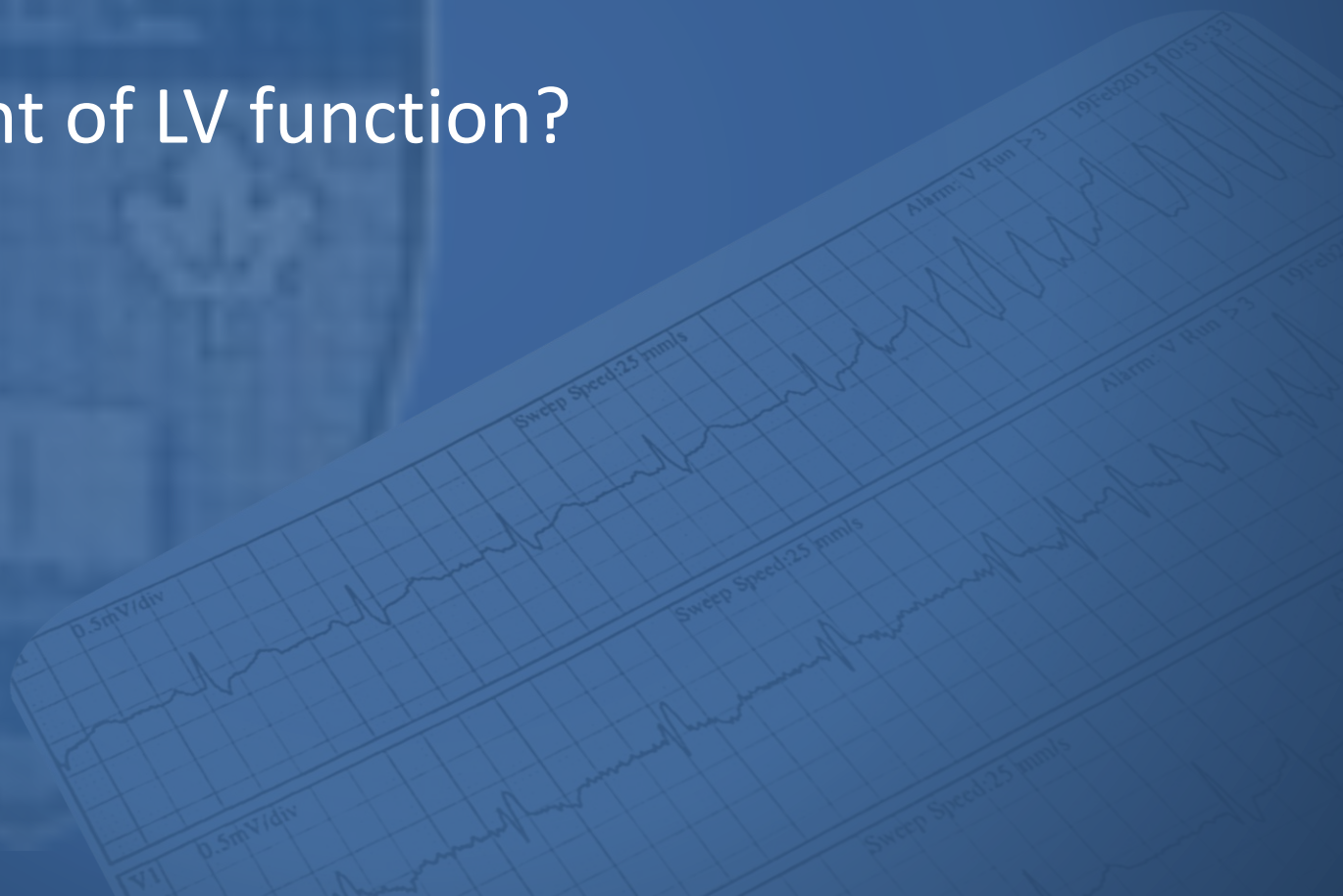


Same person post-cardioversion



What is the next step to work-up monomorphic VT?

- Coronary angiography?
- Assessment of LV function?
- EP study?



The answer is in fact not coronary angiography!

- In one study of 57 patients with MMVT receiving coronary angiography.
  - 4 patients (7.1%) needed revascularisation, of which 3 patients continued to have MMVT.

- Assessment of LVEF is more important to identify aetiology and determine management.
  - Pharmacotherapy or ablation may be considered for VT in structurally normal hearts.
  - Structurally abnormal hearts will likely require an ICD.

Skaf et al., Circulation. 2012. 126(Suppl 21): Abstract 17286.

Summary

- Like all nightmares, arrhythmias may invoke a primordial yet undeserved sense of fear in you
- If you have time to think, use it to collect your thoughts and not over-react
- Knowledge is always half the battle, and hopefully now you know!

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