ATRIAL FIBRILLATION AND ITS FRIENDS

ASSESSMENT AND TREATMENT OF AFIB, AFLUTTER & SVT

BACKGROUND

Atrial Fibrillation is the most common arrhythmia in New Zealand. It is found in up to 1% of the population and can reach up to 5% prevalence in octogenarians.

It's a chaotic rhythm generated from the atria that is irregularly irregular. It can be narrow or wide complex. Normally there are no p waves seen on the ECG.

It is seen commonly in the ED.

A-Fib can be paroxysmal, intermittent or chronic.

One of the main concerns with atrial fibrillation is that it increases the risk of thromboembolic events (about 5% per year) about 3-5 x above the normal population.

We commonly see patients with afib with RVR in the emergency department

DISCUSSION TODAY

- Atrial fibrillation with rapid ventricular response (RVR)
- Atrial flutter with RVR
- Supraventricular tachycardia

We will talk about their presentation, ECG evaluation, work up and treatment

CASE 1

63 year old male presents complaining of a “fast heart rate”. He reports it started about 12 hours ago when he was at work.

Vitals:

Temp 37

HR 162-184

BP 163/92

02 sat 95%

Resp 16

He tells you further that he has noticed a high heart rate for at least 12 hours and has seen it on his watch monitor going in the 160s. He has never had this happen before.

What else do you want to know? He is having chest pain with this and feeling mildly short of breath. No recent illness, no medication changes, no drugs/alcohol

CASE 1

PMHx: HTN

Meds: Amlodipine

Sochx: Smoker

Physical exam: What specific findings may help you?

What do you need to consider with regard to the cause of the atrial fibrillation?

CASE 1: DIFFERENTIAL

ACS

Lone Atrial fibrillation

Sepsis from pneumonia, cellulitis, UTI, other cause

Hyperthyroidism

Hypokalemia, hypomagnesemia, other electrolyte abnormalities

Occult GI bleed; Other cause anemia

Recent medication change

Methamphetamine or other stimulant use

Heavy alcohol use (holiday heart)

Pulmonary Embolism

CASE 1

Initial evaluation includes monitor, ECG, iv

Determine:

- Stable vs. unstable
- Underlying cause?

This will guide your treatment

INITIAL ASSESSMENT

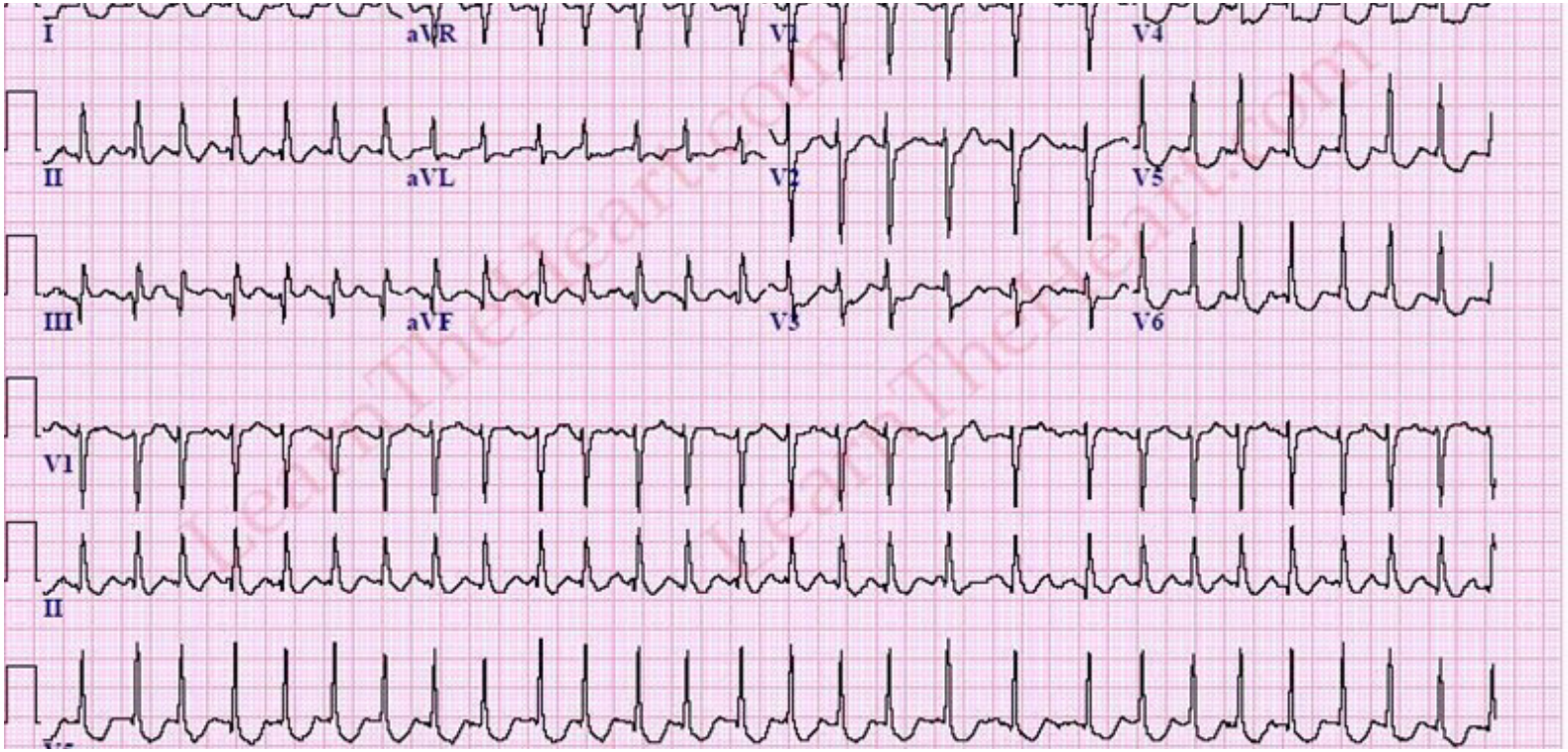
ECG: look for ACS, widened QRS, prolonged QTc.

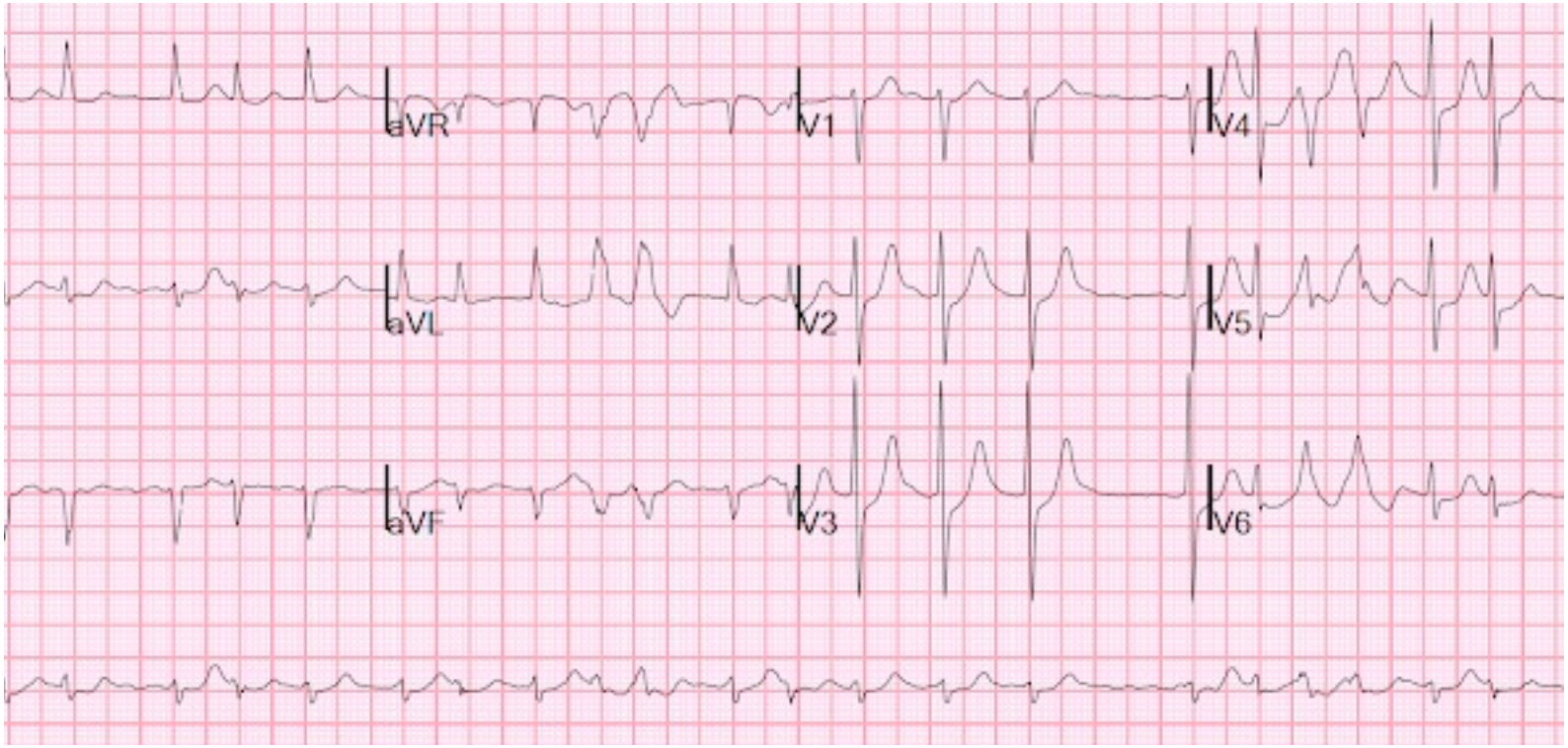
Blood work: Full Blood Count, Chemistry with electrolytes, Troponin, Thyroid Function Studies, Magnesium, D-dimer

Imaging: Chest X-ray

Bedside imaging: consider POCUS to assess for pericardial effusion, LV function

ATRIAL FIBRILLATION ECG

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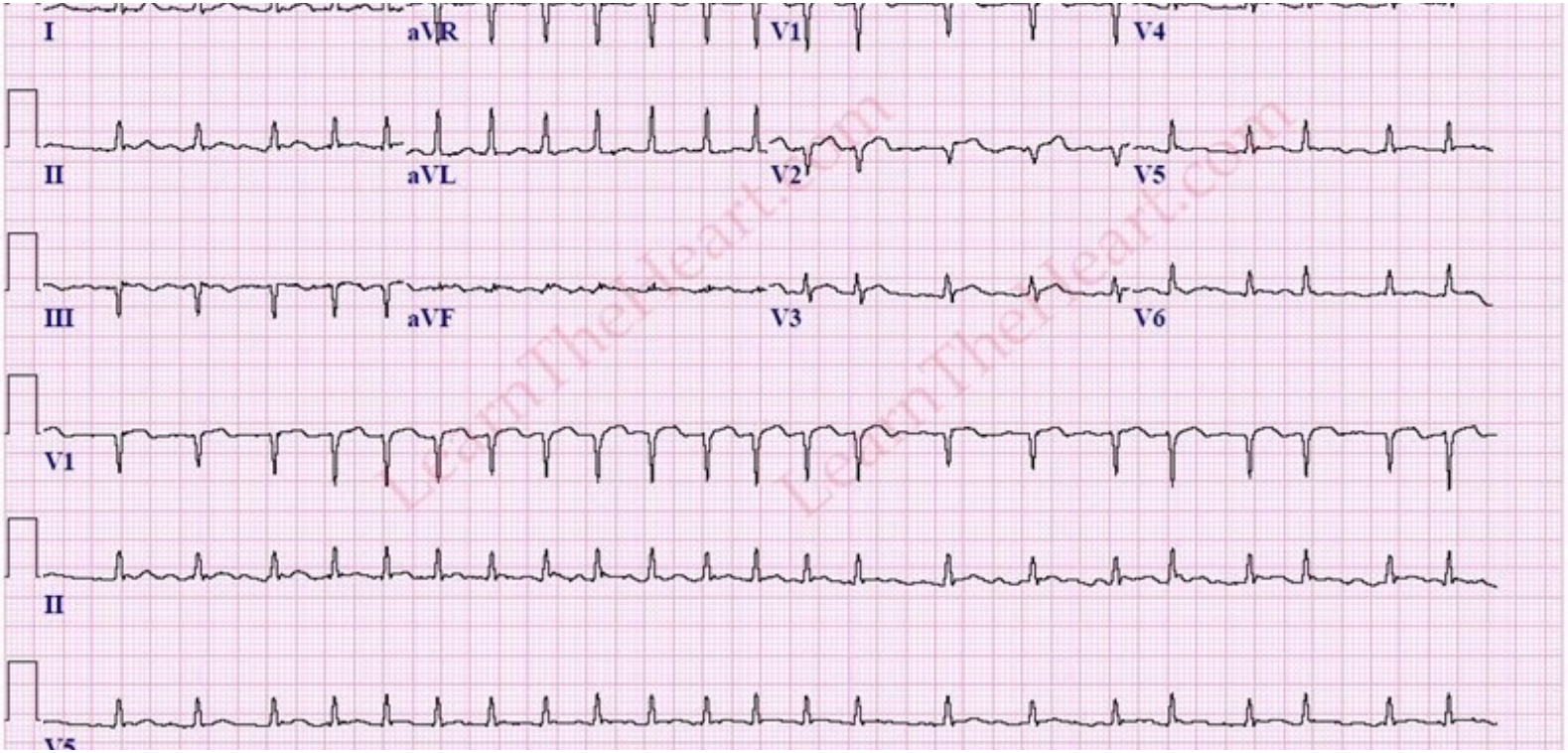
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CASE 1

Our patient has no concerning underlying conditions and other than his heart rate, his vitals are fine and he is texting on his phone and chatting comfortably.

What are our treatment options?

- Treat the underlying condition
- Rate control vs. rhythm control

If unstable – move them to Resus, consider cardioversion but realize there is a risk of thrombotic event/stroke if you do. So ask yourself, how unstable are they

Unstable would mean significant hypotension despite treatment of underlying condition, ischemia on the ECG and concerning findings for ischemia on physical exam (e.g. diaphoresis)

CASE 1: TREATMENT

Treat the underlying condition. In many patients, they tolerate a fairly fast heart rate. The older they are, the less well they tolerate faster heart rates. However, slowing the rate down when the underlying condition is the cause of the atrial fibrillation will likely worsen the patient's condition. They may need the tachycardia as compensation.

Fluids for sepsis or dehydration

IV antibiotics for sepsis

Blood for GI bleed with anemia or other cause anemia

Benzodiazepines for methamphetamine or other sympathomimetic drug

CASE 1: RATE VS. RHYTHM CONTROL

RHYTHM CONTROL - CARDIOVERSION:

Chemical cardioversion:

Flecainide 300 mg po x 1 if > 70 kg or 200 mg if < 70 kg. Do not use in people with structural heart disease or heart failure.

Ibutilide

Depends on local practice patterns. Some countries/areas do more chemical cardioversion, others tend to do more rate control.

Electrical cardioversion:

200 J (Biphasic). Again, consider whether the patient truly needs cardioversion because there is a risk of stroke and recent trials indicated that < 48 hours since onset is likely not safe and really it should be only < 12 hours

CASE 1

RATE CONTROL:

Diltiazem 2.5 mg- 20 mg iv bolus. Normally 10 mg, assess blood pressure and heart rate and then may repeat. Use caution with elderly, borderline hypotension. Diltiazem 30 mg po.

Metoprolol 2.5 – 5 mg iv bolus or Metoprolol 25 mg po

Amiodarone 150 mg bolus over 10 minutes

Esmolol bolus and drip. Good because fast on/off but not always available in the ED.

Digoxin. Okay to give bolus of digoxin but it is not helpful in hyperacute situation.

CASE 1: DISPOSITION

The patient was treated with a Diltiazem 10 mg iv bolus. His heart rate improved to 110-120 and he continued to have otherwise normal vitals. His chest pain resolved with treatment of the tachycardia.

Because this was his first episode and he had an elevated troponin, he was admitted to trend his troponin and for an echocardiogram and further work up.

Not all patients need to be admitted but if they are going home, you need to consider their thromboembolic risks and make sure they have excellent follow up and a plan for further work up.

Who would I send home? Younger, healthy, asymptomatic, patient is back in NSR (possibly HR controlled but still in A fib), no concerning underlying cause, low CHAD2S-VASC score and low HAS BLED score if considering anticoagulation. I would likely have discussion with GP and/or in patient team prior to discharging.

CASE 2

82 year old female presents feeling weak. She was brought in by St. John's around 11 pm from her home where she lives with her daughter. She started feeling unwell a few hours before.

Vitals:

Temp: 36.1

HR: 170

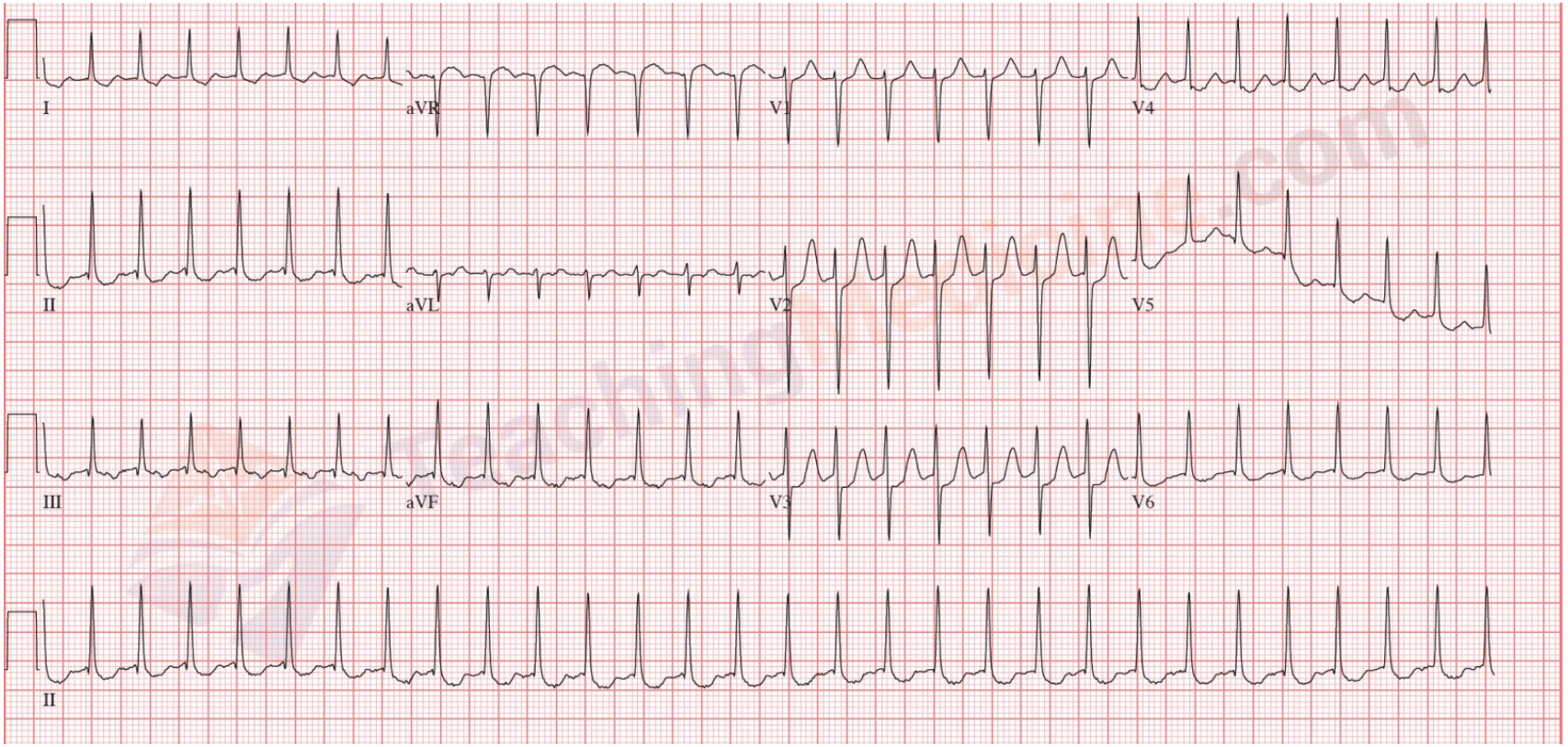
BP: 154/86

O2 sat: 96% RA

Resps: 18

What would you like to do first? Monitor, ECG, iv access

ECG



SVT

Regular, narrow complex tachycardia.

Usually rate is 150-180.

It is very regular and doesn't vary hardly at all. This is compared to a sinus tachycardia which should vary a fair amount.

Causes can be similar to Atrial fibrillation/atrial flutter but SVT is a more benign rhythm usually.

TREATMENT: SVT

Non invasive:

Vagal maneuvers

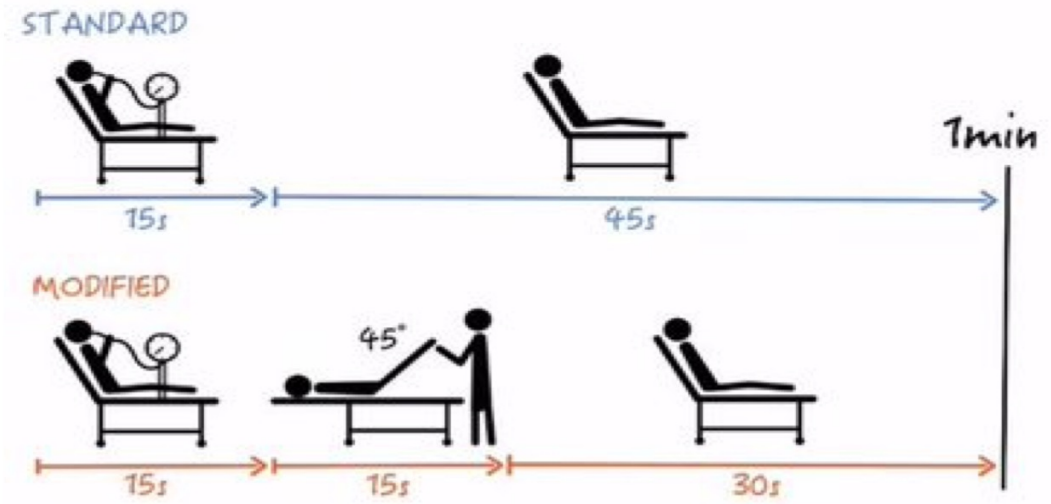


Figure 3. The Modified Valsalva Maneuver
From: medmastery.com/magazine/modified-valsalva-maneuver-video-review

TREATMENT: SVT

IV medications:

Adenosine 6 mg, then 12 mg. Avoid in patients with heart transplant. Be very cautious in patient's with a central line because normal doses can cause prolonged asystole. Also use a lower dose (3 mg)when on dipyridamole or carbamazepine.

It's important to talk to your patient about how Adenosine will make them feel if they haven't had it before. It gives them a horrible pressure in the chest, an impending sense of doom and they feel awful for about 15 seconds.

How to give adenosine? It should be through a line in the AC fossa if at all possible. IV push then quickly flush with 20 ml of saline, hold arm up.

Diltiazem can be second line if Adenosine not working.

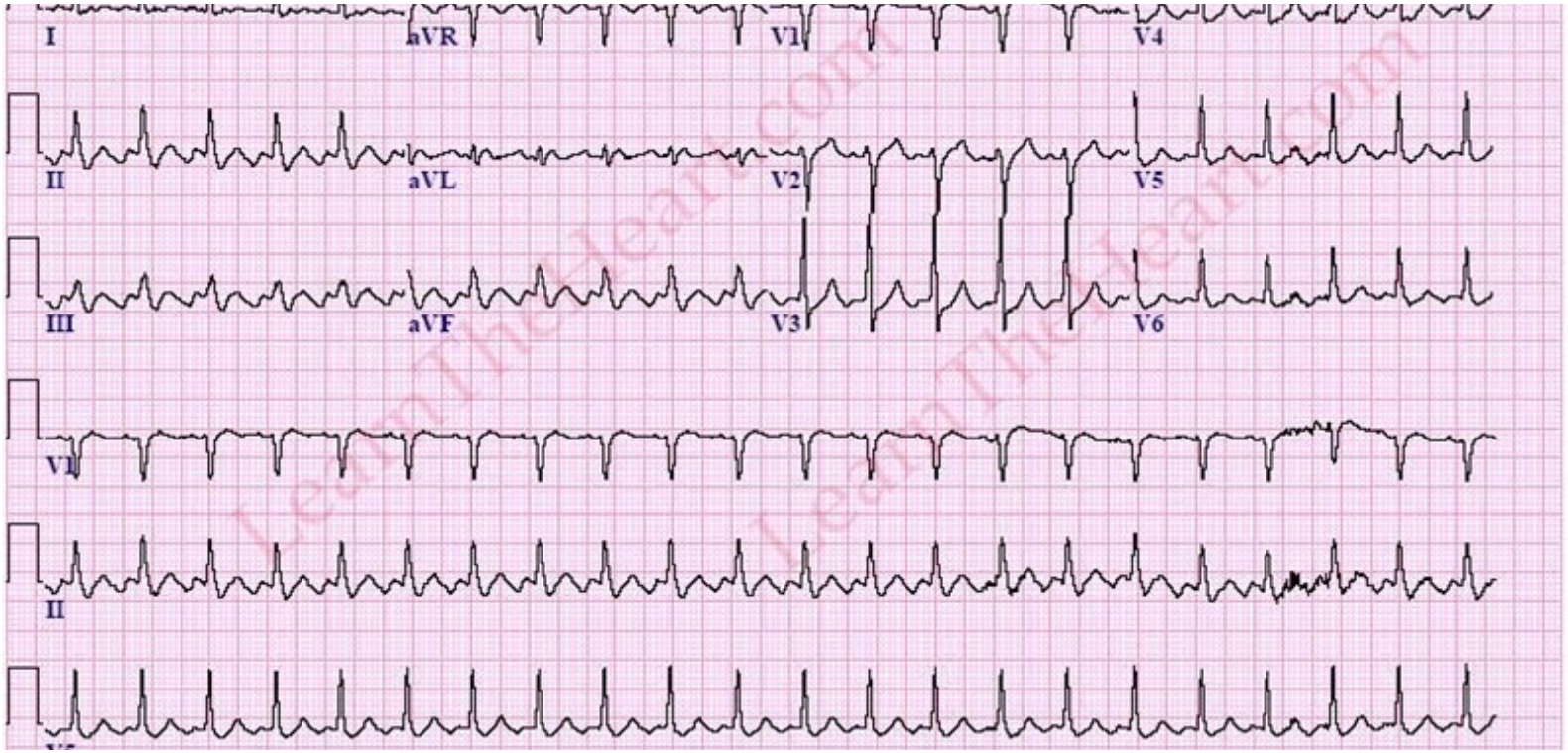
DISPOSITION

Who can go home?

Patients with a history of SVT, whose symptoms have resolved and who have no underlying cause that needs treatment can go home.

Younger, good follow up, asymptomatic after treatment, even if it is first event can go home if there are no concerns for structural heart disease but they should have close outpatient follow up and may need a cardiology referral.

This patient's SVT converted with the modified vagal maneuver but she was admitted because her troponin was elevated and she had a UTI and mild AKI which probably contributed to her going into SVT.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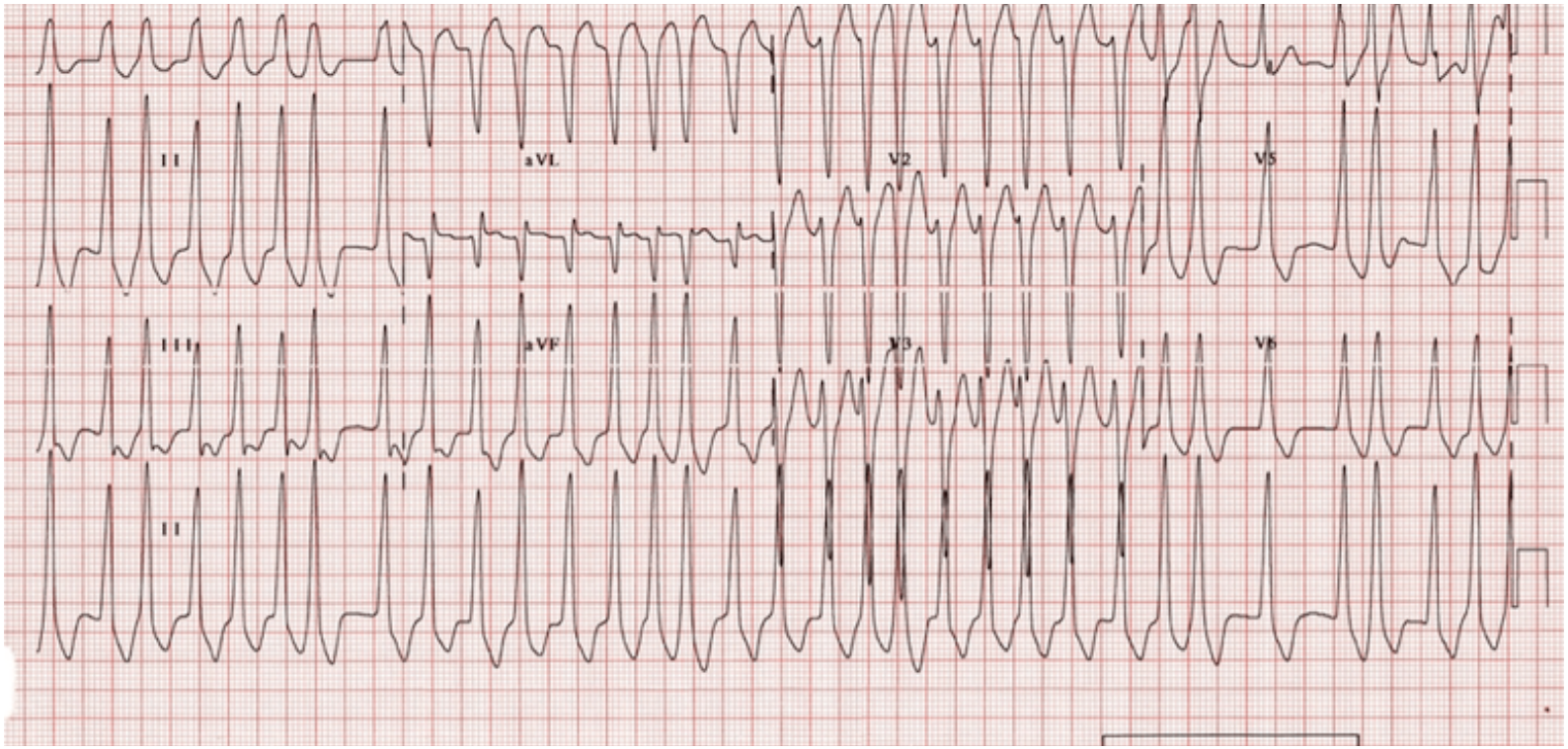
ATRIAL FLUTTER

The differential diagnosis, work up and treatment of atrial flutter is very similar to atrial fibrillation. You may find that atrial flutter is easier to cardiovert both chemically and electrically if you need to cardiovert.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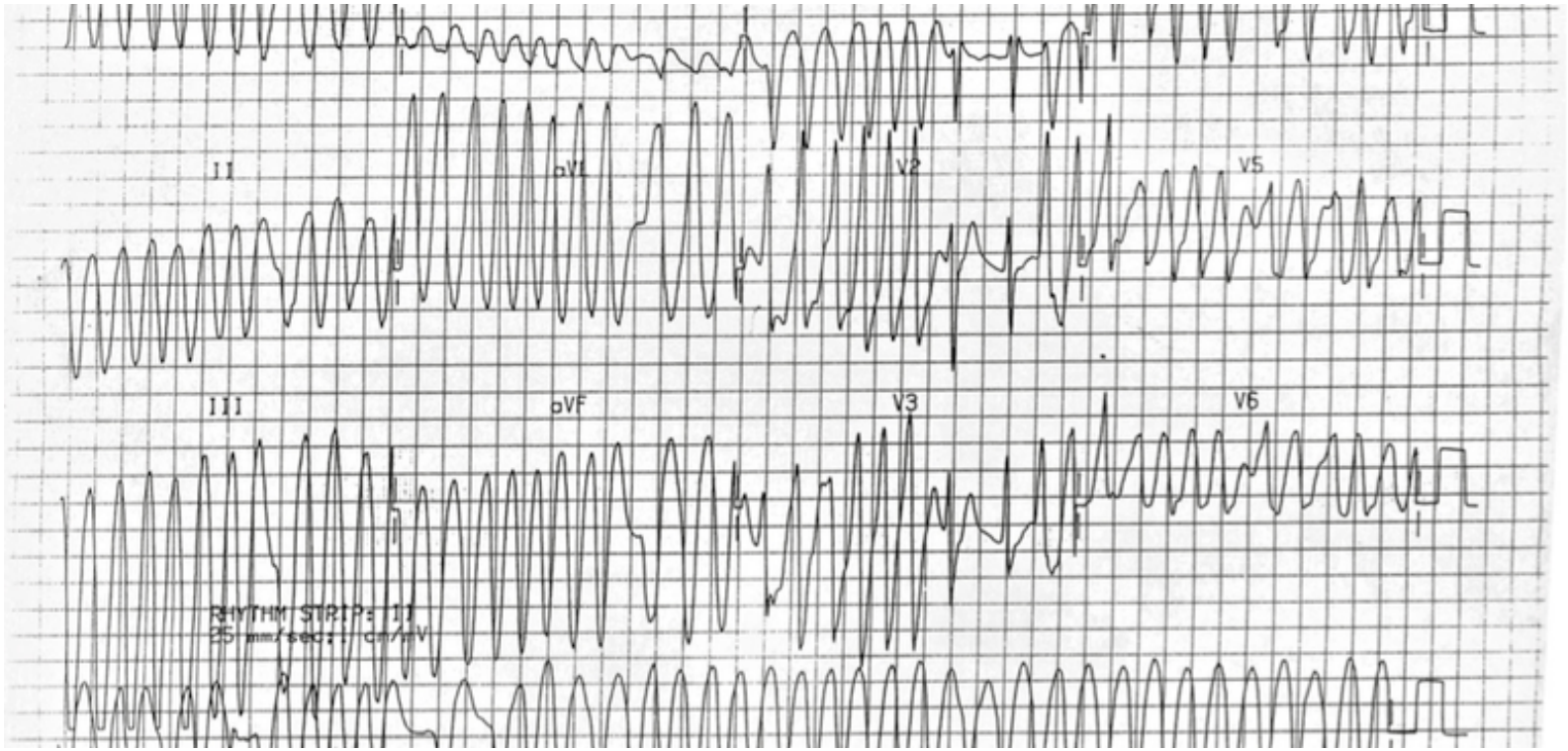
ATRIAL FIBRILLATION WITH WPW



ATRIAL FIBRILLATION WITH WPW

This is really important. A typical patient with this would usually be younger, possibly have a family or personal history of “abnormal ECG”. If you have an old ECG, make sure to look at it.

If you give them a beta blocker, calcium channel blocker, adenosine they could rapidly worsen because you block conduction through the AV node. This has to do with the fact that in WPW they have an accessory pathway and if the atrial fibrillation is conducted down this when the normal pathway is blocked, it will accelerate even more. Be very cautious in these situations and review ECG and case with someone

The ECGs can be tricky and sometimes difficult to determine whether it’s atrial fibrillation with a wide complex or Torsade or ventricular tachycardia.

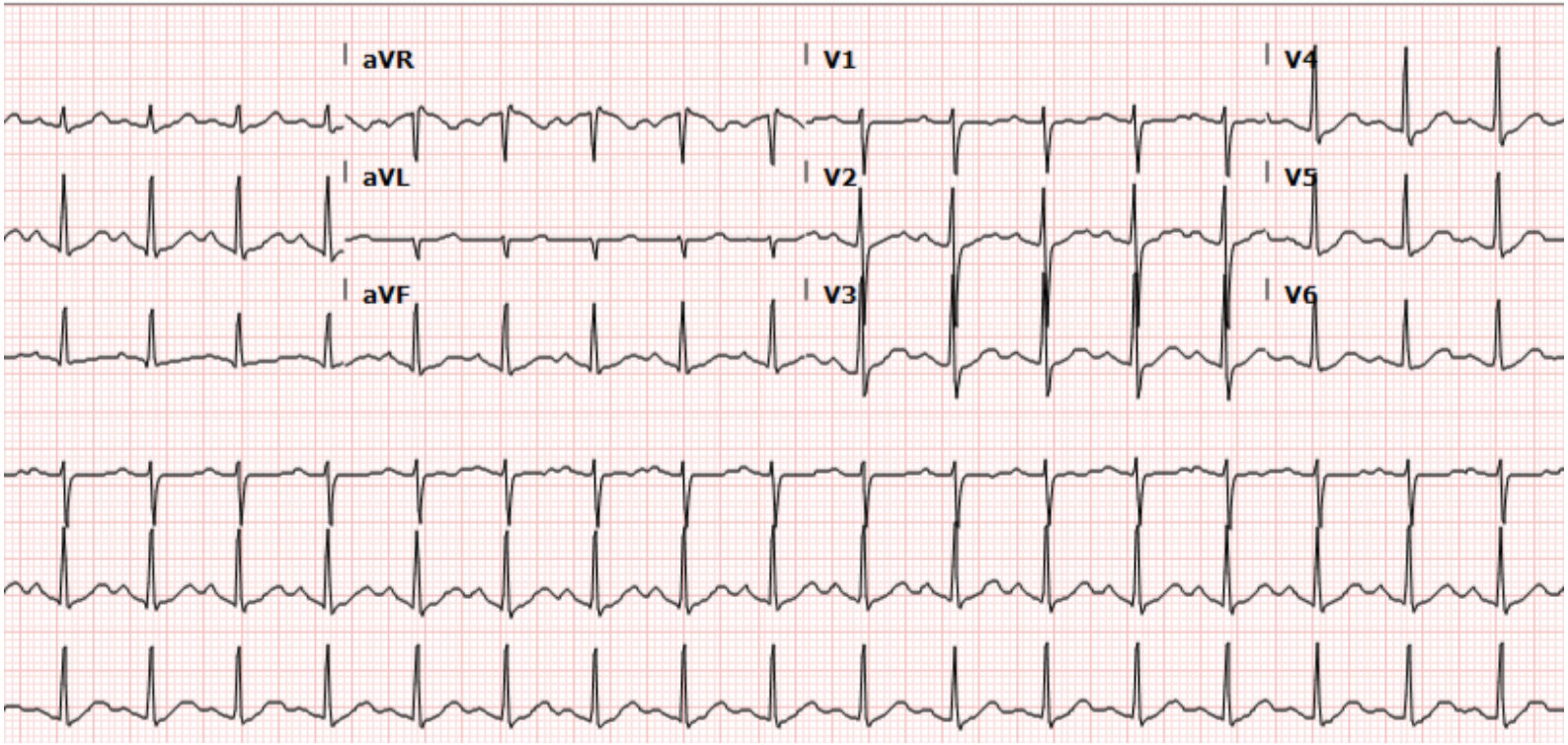
TREATMENTS: AFIB WITH WPW

Electrical cardioversion (preferred)

Procainamide

Amiodarone

Ibutilide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SINUS TACHYCARDIA

Sometimes it can be difficult to differentiate between sinus tachycardia versus atrial flutter versus SVT.

One clue can be that sinus tachycardia will vary with the respiratory cycle while aflutter or SVT will not.

It doesn't hurt to try the modified valsalva maneuver to see if you note p waves or changes in the rate

Repeat the ECG

PEARLS

Always consider the underlying cause and use your history and physical exam to help you find clues.

Treat the underlying cause.

Remember to ask yourself, how is the patient tolerating the rate? You may not need to emergently slow them down even if they need to be admitted for further work up. You could consider oral medications.

Try the modified vagal maneuver for SVT. It can be up to 47% effective and helps you avoid extra monitoring and time, especially in otherwise healthy patients who might be able to go home after they convert.

Remember to watch out for wide complex atrial fibrillation with RVR. Be very concerned about WPW and avoid beta blockers, calcium channel blockers and adenosine.