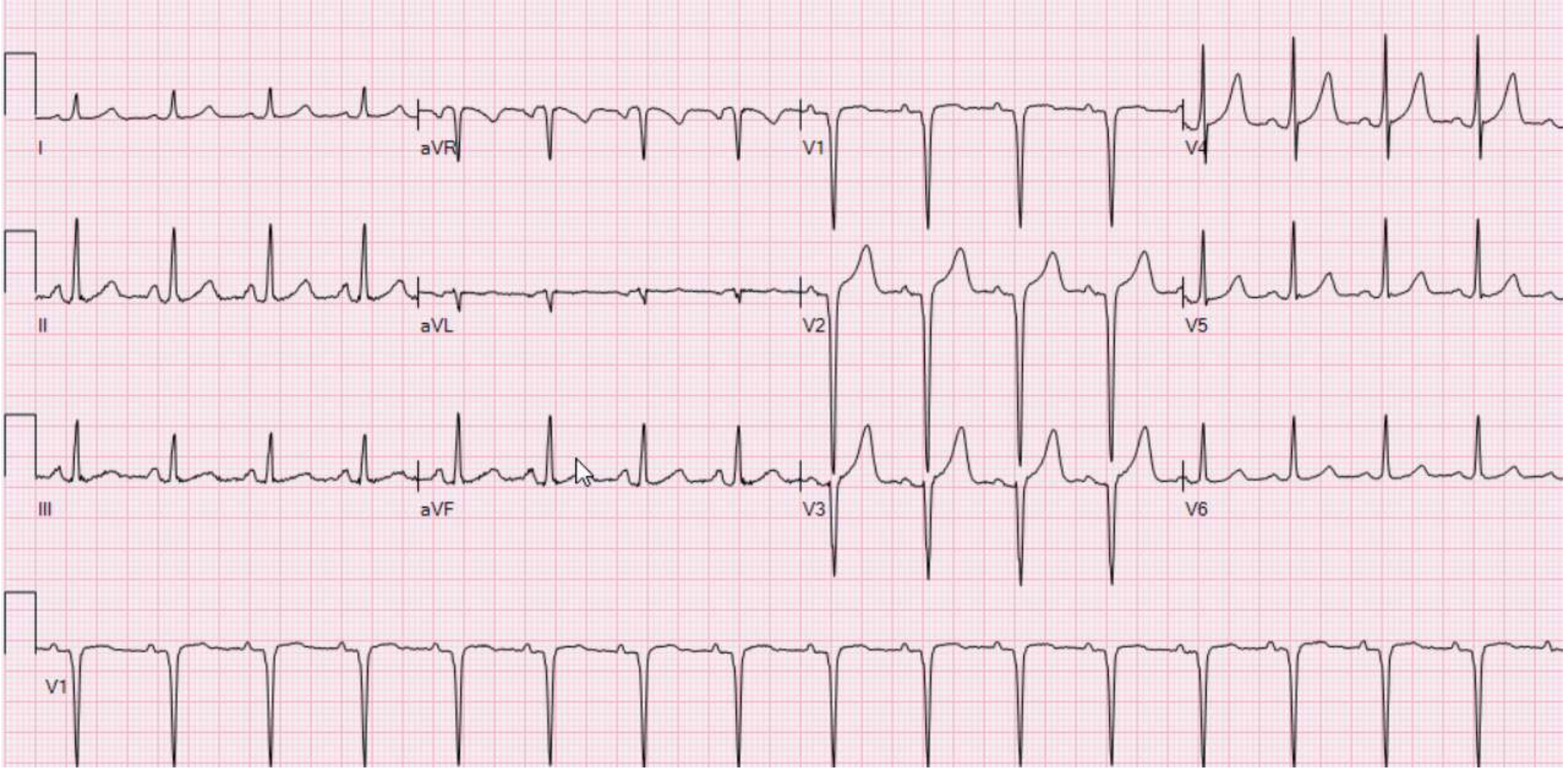
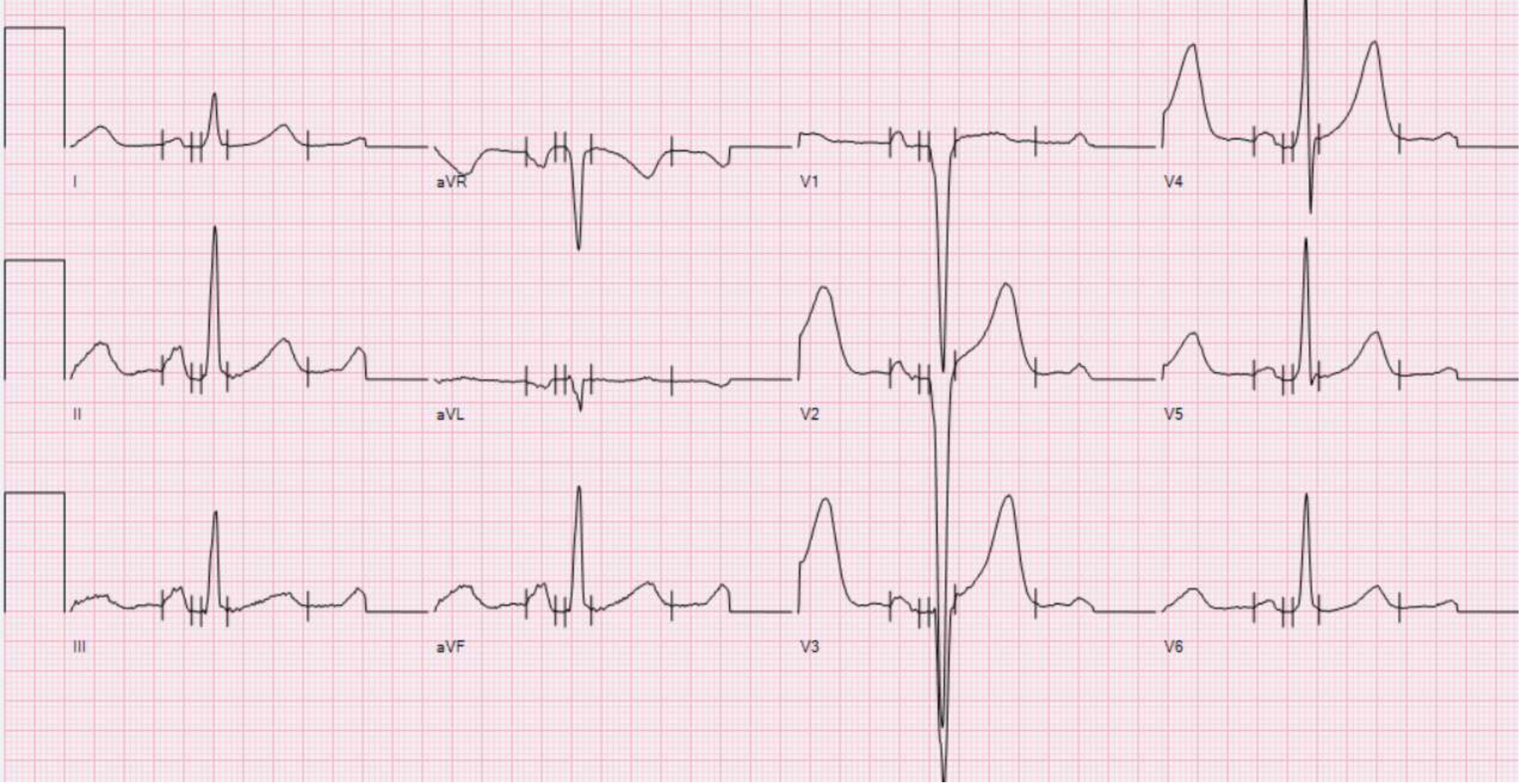
23-year old woman
Only palpitations
Electrophysiological study: manifest **right-sided Mahaim pathway**
No tachycardia's. What is your opinion about these ECG tracings?

Kjell Nikus, MD PhD
Heart Center, Tampere University Hospital and Faculty of Medicine and Health Technology, Tampere University, Finland
“The man from the icy country and the happiest people in the world!!”



Absent septal q waves, poor R-wave progression**.** **Observation:** **paper speed: 50mm/sec · 1mm (small square) = 20 ms · 5 mm (large square) = 100 ms.**


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6

Answer from

Prof. Dr. Andrés Ricardo **Pérez-Riera, MD PhD**
Clínica Médica e suas especialidades
Uninove - Universidade Nove de Julho - Campus Mauá

Dear friend Nikus:

Explanation about utility paper speed: 50mm/sec and the eponyms Mahaim fiber in next slides. The eponyms Mahaim fiber maybe should be abandoned

About paper speed: 50mm/sec

Doubling the standard rate will cause the ECG to appear drawn out or wider complex than 25mm/sec paper speeds

1mm (small square) = 0.02 sec (20ms)

5mm (large square) = 0.1 sec (100ms)

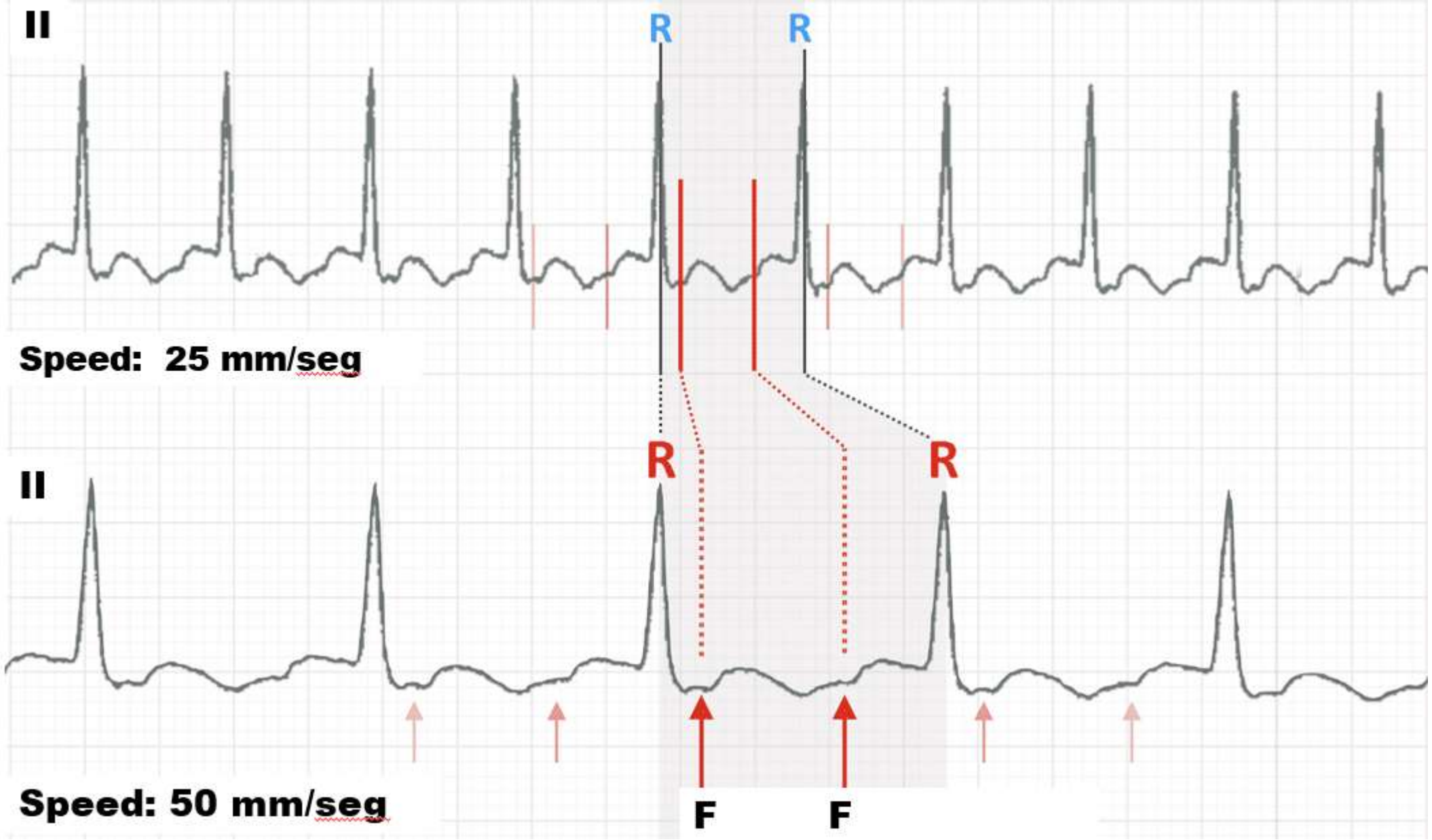
The rhythm strip will thus comprise 5 seconds total capture compared to the standard 10 seconds.

Annotated example

Why use 50 mm/second?

Doubling the standard rate can reveal subtle ECG findings hidden at the slower rates, in particular atrial flutter waves in a 2:1 block:

Correct diagnosis of difficult narrow complex tachycardia's Is improved when ECGs **at both 25 mm/s and 50 mm/s were used for interpretation**. It appears that the simple technique of increasing the ECG paper speed, and thus effectively spacing out the rhythm, enhances the diagnostic ability of the observer. Best evidence topic reports (BETs) summarize the evidence pertaining to particular clinical questions. They are not systematic reviews, but rather contain the best (highest level) evidence that can be practically obtained by busy practicing clinicians. The search strategies used to find the best evidence are reported in detail in order to allow clinicians to update searches whenever necessary. Each BET is based on a clinical scenario and ends with a clinical bottom line, which Indicates, in the light of the evidence found, what the reporting clinician would do if faced with the same scenario again. The BETs published below were first reported at the Critical Appraisal Journal Club at the Manchester Royal Infirmary or placed on the BestBETs website. Each BET has been constructed in the four stages that have been described elsewhere. The BETs shown here together with those published previously and those currently under construction can be seen at http://www.bestbets.org Four BETs are included in this issue of the journal. 1) Diagnostic utility of ECG for diagnosing pulmonary embolism.; 2) Lignocaine as a pretreatment to rapid sequence intubation.; 3) in patients with status asthmatics Steroids in sudden sensorineural hearing



At 50mm/sec, **F** waves are more clearly discernible in a 2:1 ratio to QRS complexes

loss..; 4) **Differential diagnosis of narrow complex tachycardia's by increasing electrocardiograph speed. (2).**

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In **1938,** Mahaim and Benatt **1** described the pathological findings of fibers connecting the atrioventricular (AV) node and His bundle to the ventricular septum. The role of such fibers in causing arrhythmias is controversial. In **1981**, Gallagher et al wrote an article**2** on variants of Wolff-Parkinson-White (WPW) syndrome and wide QRS tachycardia's. They described 12 cases of “Mahaim” fibers, **6** with nodoventricular fibers (NVFs) and 6 with fasciculoventricular fiber (FVFs).**2** All those with NVFs had normal or prolonged PR intervals in sinus rhythm and left bundle branch block (LBBB) morphology with left axis deviation during tachycardia. They theorized that the ventricular insertion of these fibers was in the posteroinferior right ventricle (RV) or septum. The 6 patients with FVFs had a short PR interval, and none had an arrhythmia causally related to the FVF. Citing Anderson et al**3** (whose pathological work had suggested using the terms NVF and FVF fibers), they concluded that patients with a certain type of LBBB

morphology during the tachycardia had an underlying NVF or nodofascicular fiber. They went on to add that “an alternative explanation would be an accessory AV node or an AP with “nodal” properties inserting in the right ventricle.” In 1982, Gillette et al[4] described 4 patients with “prolonged and decremental conduction properties in right anterior accessory connections: wide QRS antidromic tachycardia of LBBB pattern without WPW in sinus rhythm.” They showed that “the endocardial ventricular insertion of this fiber was at the RV apex or the RV anterior surface near the position of the moderator band.” By successfully treating 3 patients with an atrial incision remote from the AV conduction system, they showed that “the weight of evidence in these patients suggests that they did not have a Mahaim fiber.” Hence, Gillette et al were the first to demonstrate cardiac APs that had AV nodal (decremental) properties, an atrial insertion, an insulated ventricular that “an alternative explanation would be an accessory AV node or an AP with “nodal” properties inserting in the right ventricle.” Gallagher p 178.2

in right anterior accessory connections: wide QRS antidromic tachycardia of LBBB pattern without WPW in sinus rhythm.” They showed that “the endocardial ventricular insertion of this fiber was at the RV apex or the RV anterior surface near the position of the moderator band.” By successfully treating 3 patients with an atrial incision remote from the AV conduction system, they showed that “the weight of evidence in these patients suggests that they did not have a Mahaim fiber.” Hence, Gillette et al were the first to demonstrate cardiac accessory pathways that had AV nodal (decremental) properties, an atrial insertion, an insulated ventricularFollowing this, others have described similar pathways on the left side and from the aortic cusps.7–9 In 1994, Klein et al10 published an editorial in Circulation. They said: “The time has come to abandon the vague and inappropriate jargon applied to entities involving APs with long conduction times and rate-dependent conduction, including ‘Mahaim tract’ and ‘Mahaim physiology.’” As shown above, Mahaim did not describe AFF and the fibers that Mahaim had described, although of historical interest, do not correspond to the clinically significant AFF as currently understood. Hence, it does not appear appropriate to apply the name “Mahaim fiber” to these pathways. For historical context, inaccuracy in eponymous designation is replete in the electrophysiology world. Indeed, histological

structures believed by Kent11 in 1913 to be the normal conduction pathways at the AV junction were later attributed to him to be the substrate for the WPW pattern. In fact, even that is probably inaccurate, as these structures had an AV node–like appearance and, as pointed out by Becker et al,12 probably represented the first description of the proximal components of AFF.

AFF Atriofascicular Fiber

ART Antidromic Reciprocating Tachycardia;

AVF Atrioventricular Fiber

dcAVF decrementally conducting Atrioventricular Fiber

FVF Fasciculoventricular Fiber

NFF Nodofascicular Fiber;

NVF Nodoventricular Fiber

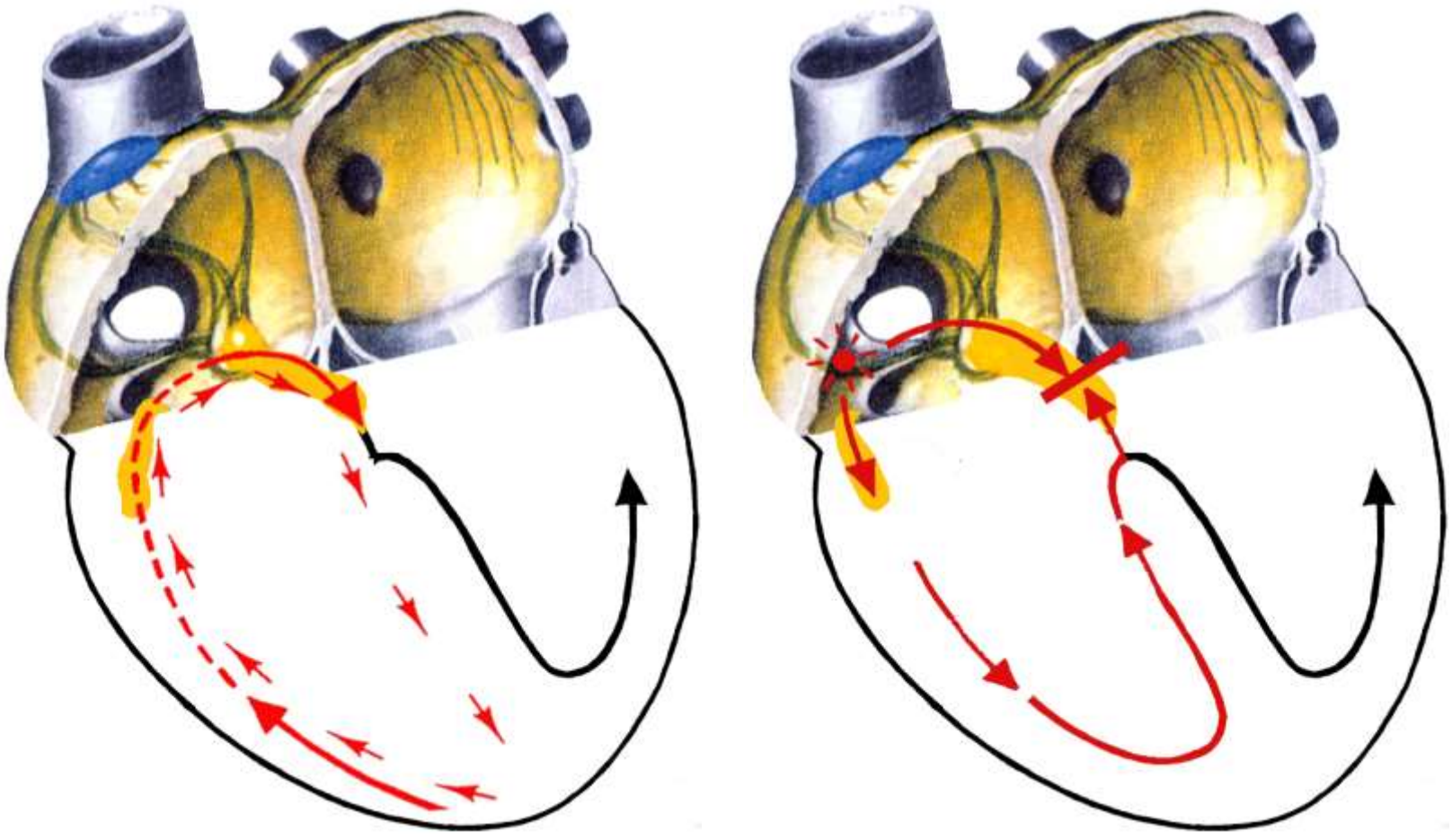
Table 1: Schema to distinguish atypical accessory pathways (APs) on next slide

Table 1: Schema to distinguish atypical accessory pathways

Sinus rhythm shows:	Regular, wide complex tachycardia shows:	The nonelectrophysiologist should consider:	The electrophysiologist should consider:
No preexcitation	LBBB, LAD, and VA association	▪ VT, ▪ NC SVT with LBBB, ▪ Preexcited SVT	▪ VT ▪ NC SVT with LBBB ▪ ART using AFF and NFF ▪ NC SVT with/bystander AFF and NFF
No preexcitation	LBBB, LAD, and VA dissociation (V.A)	▪ VT	▪ VT ▪ ART using NFF ▪ Orthodromic AVN-NFF reentry with LBBB
Atypical preexcitation	Wider version of sinus QRS onset and VA association	▪ Preexcited SVT ▪ VT	▪ ART using dcAVF and NVF ▪ NC SVT w/bystander dcAVF, NVF ▪ VT
Atypical preexcitation	Wider version of sinus QRS onset and VA dissociation (V.A)	▪ VT ▪ Preexcited SVT	▪ ART using NVF ▪ VT
Atypical preexcitation	Identical QRS to sinus	▪ Preexcited SVT	▪ NC SVT w/bystander FVF
Typical preexcitation	Wider version of sinus QRS onset (delta wave) and VA association	▪ Preexcited SVT ▪ VT	▪ ART ▪ NC SVT with bystander AVF ▪ VT

AFF 5 atriofascicular fiber; **ART** 5 antidromic reciprocating tachycardia; **AVF** 5 atrioventricular fiber; **AVN** 5 atrioventricular node; **dcAVF** 5 decrementally conducting atrioventricular fiber; **FVF** 5 fasciculoventricular fiber; **LAD** 5 left axis deviation; **LBBB** 5 left bundle branch block; **NC** 5 narrow complex; **NFF** 5 nodofascicular fiber; **NVF** 5 nodoventricular fiber; **SVT 5** supraventricular tachycardia; **VA** 5 ventriculo-atrial; **VT** 5 ventricular tachycardia.

I. **AFF Atriofascicular Fiber/Pathway:** An atriofascicular pathway (Mahaim fiber) is characterized by **decremental antegrade conduction properties in the absence of retrograde conduction**. During antidromic Atrioventricular Re-entry Tachycardia (AVRT), the surface ECG demonstrates left bundle branch block (LBBB) morphology with late precordial transition such as the first ECG. Atrioventricular Re-entry Tachycardia (AVRT) is a form of paroxysmal supraventricular tachycardia that occurs in patients with accessory pathways(APs), usually due to formation of a re-entry circuit between the AV node and AP. ECG features depend on the direction of conduction, which can be orthodromic or antidromic. In **orthodromic** AVRT, anterograde conduction is via the AV node, producing a regular narrow complex rhythm (in the absence of pre-existing bundle branch block. In **antidromic** AVRT, anterograde conduction is via the accessory pathway (AP), producing a regular wide complex rhythm. This can be difficult to distinguish from ventricular tachycardia (VT) Often triggered by premature atrial or premature ventricular contraction In both forms, the features of pre-excitation are lost *achyarrhythmias in pre-excitation can also be facilitated by direct conduction from the atria to the ventricles via the AP, bypassing the AV node. This is seen with atrial fibrillation or atrial flutter in conjunction with WPW,*



Orthodromic

Antidromic

Orthodromic AVRT: Anterograde conduction through AV node

Antidromic AVRT: Retrograde conduction through AV node

In **orthodromic** AVRT, anterograde conduction is via the AV node, producing a regular narrow complex rhythm (in the absence of pre-existing bundle branch block).

In **antidromic** AVRT, anterograde conduction is via the AP, producing a regular wide complex rhythm. This can be difficult to distinguish from VT.

Often triggered by premature atrial or premature ventricular contractions (PVCs). In both forms, the features of pre-excitation are lost.

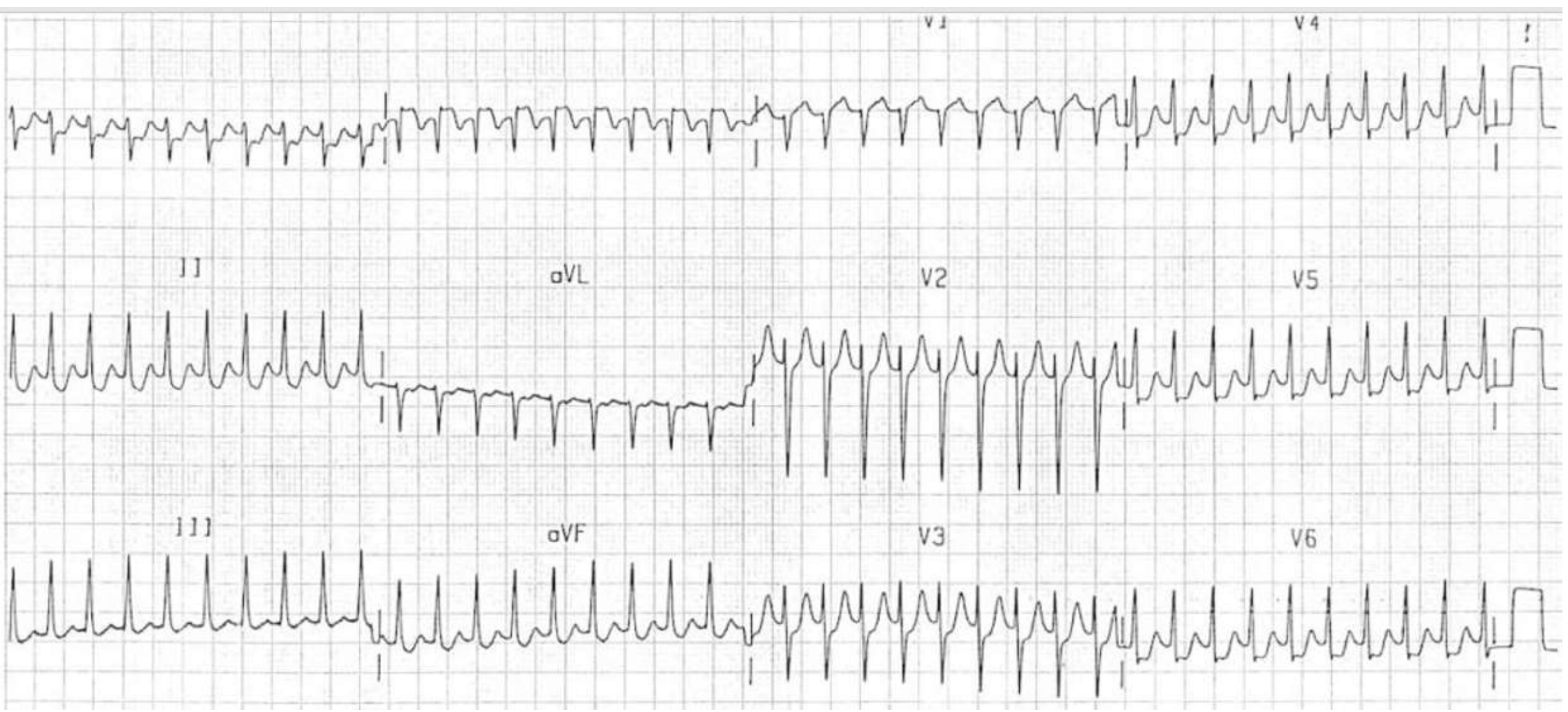
Tachyarrhythmias in pre-excitation can also be facilitated by direct conduction from the atria to the ventricles via the AP, bypassing the AV node. This is seen with atrial fibrillation (AF) or atrial flutter (Afl) in conjunction with WPW.

Orthodromic AVRT

In orthodromic AVRT, anterograde conduction occurs via the AV node, resulting in a normal direction of ventricular depolarization. This can occur in patients with a concealed AP that conducts retrograde only, not evident on sinus rhythm ECG.

ECG features of AVRT with orthodromic conduction

1. Rate usually 200-300 bpm
2. Retrograde P waves are usually visible, with a long RP interval
3. QRS < 120ms unless pre-existing bundle branch block, or rate-related aberrant conduction
4. QRS alternans: phasic variation in QRS amplitude associated with AVNT and AVRT, distinguished from electrical alternans by a normal QRS amplitude
5. Rate-related ischemia is common



Orthodromic AVRT: Regular, narrow complex tachycardia

Orthodromic AVRT, or just AVNRT?

This rhythm can appear very similar to AVNRT, but the **RP interval** can assist us to differentiate:

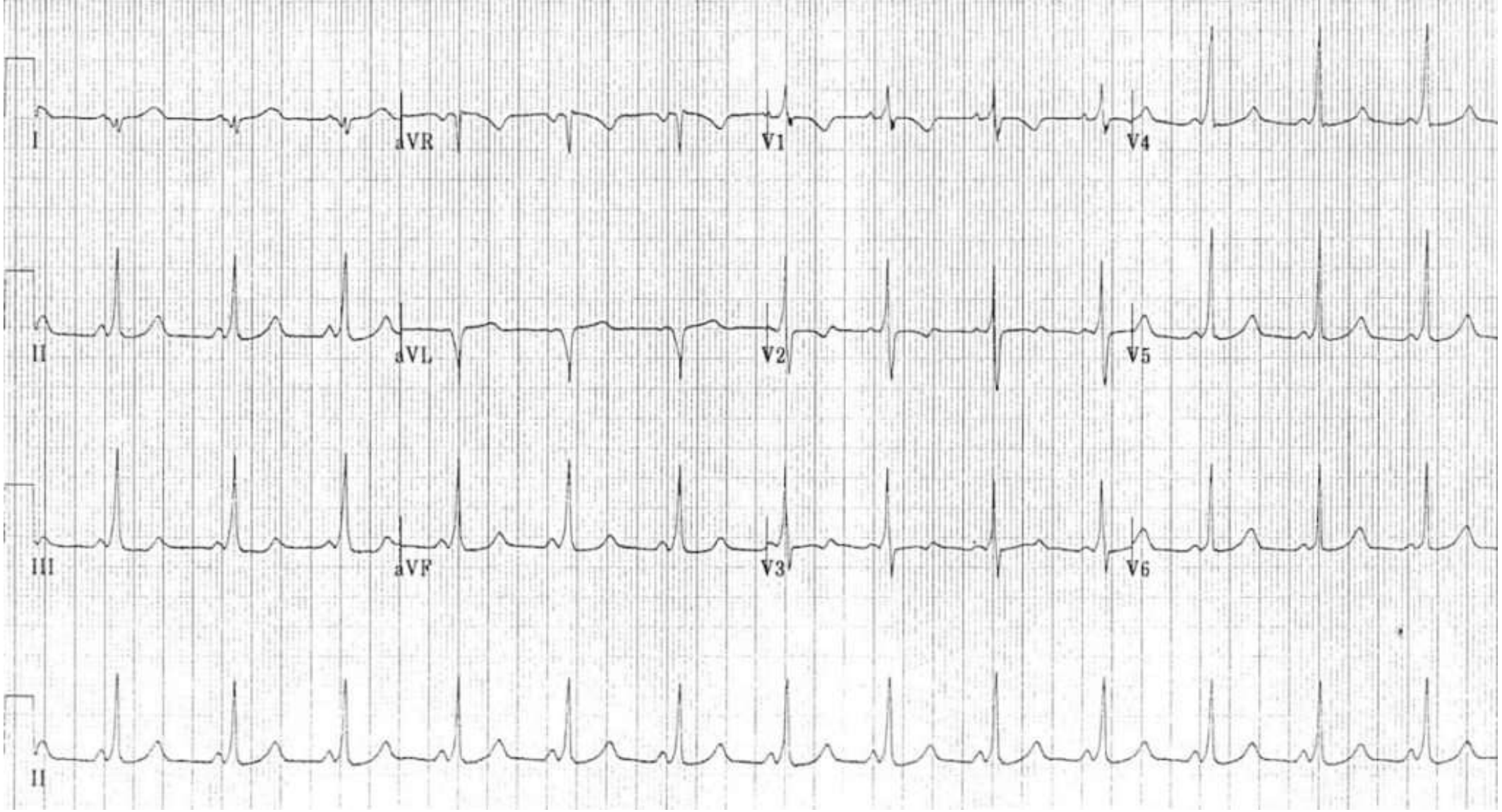
In typical **AVNRT,** retrograde P waves occur early, so we either don't see them (buried in QRS) or partially see them (pseudo R' wave at terminal portion of QRS complex)

In **AVRT,** retrograde P waves occur later, with a *long* RP interval > 70 msec

In the above example, look closely at V1 — P waves are evident as a small notch at the beginning of the T wave, with a long RP interval, indicating this is likely orthodromic AVRT.

Fortunately, treatment is fairly similar for both.

Treatment of orthodromic AVRT As always, patients that are unstable due to this rhythm require urgent DC cardioversion The anterograde portion of conduction is typically the "weak link" of the re-entry circuit. Management options in the stable patient therefore target slowing conduction through the AV node A stepwise approach similar to AVNRT can be employed, beginning with vagal manoeuvres followed by adenosine and/or verapamil. *Note that with administration of any AV nodal blocking drug, there is a very small but significant risk of inducing AF.* If verapamil is used, patients should be observed for at least 4 hours to ensure AF does not develop as a consequence of AV nodal blockade.



The patient reverts to sinus rhythm after treatment with adenosine. Wolff-Parkinson-White (WPW) pattern is now evident on the baseline ECG; this confirms that the initial rhythm was orthodromic AVRT. Tall R waves in right precordial leads (V1-3) indicate a left-sided AP

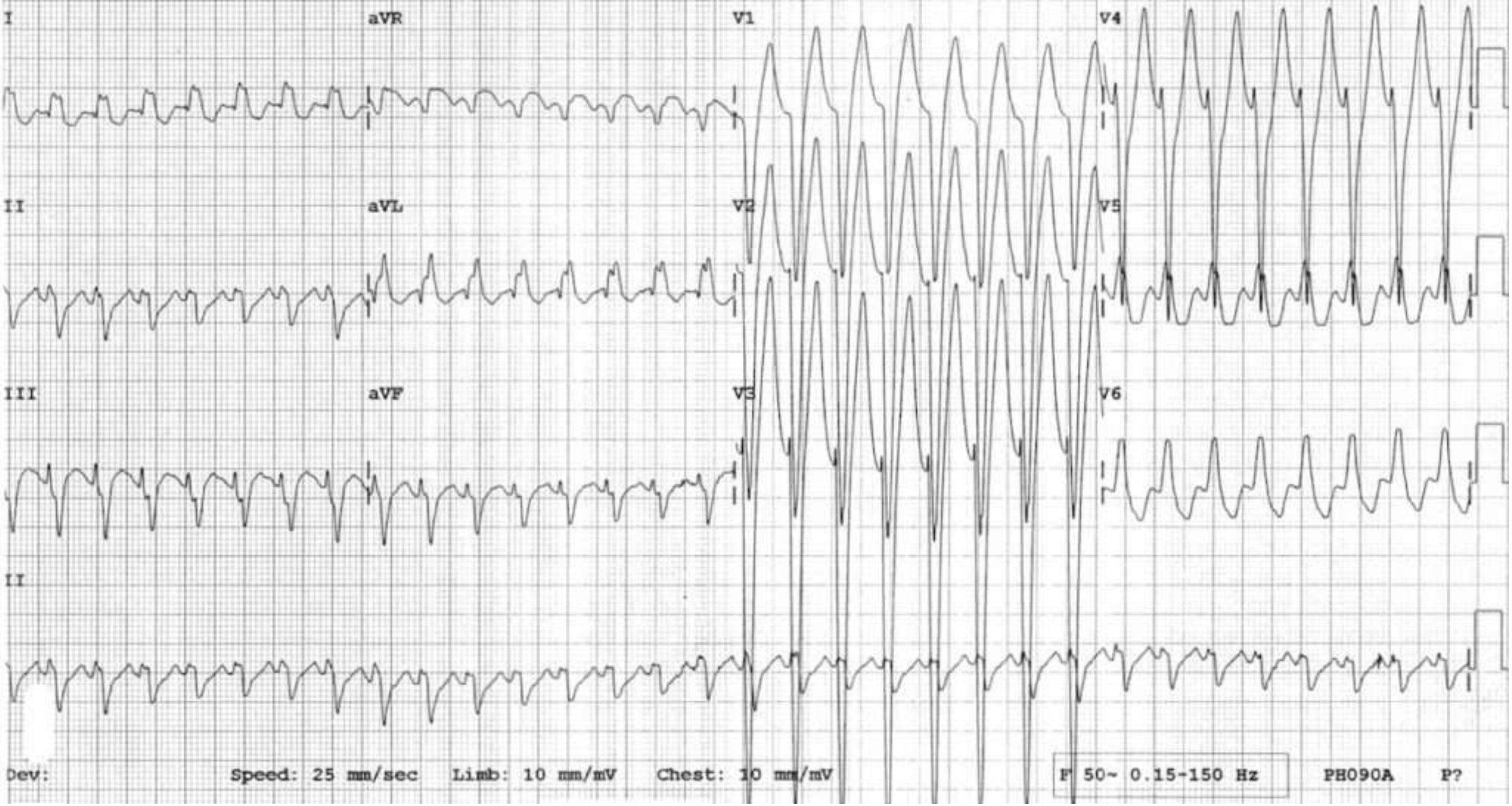
Antidromic AVRT

Antidromic AVRT is rare, and makes up only 5% of tachyarrhythmias in patients with WPW. As the name suggests, it involves anterograde conduction via the AP. Retrograde conduction is usually via the AV node, but can also be via another AP. The abnormal direction of ventricular depolarisation results in a broad complex tachycardia, which can be easily mistaken for VT.

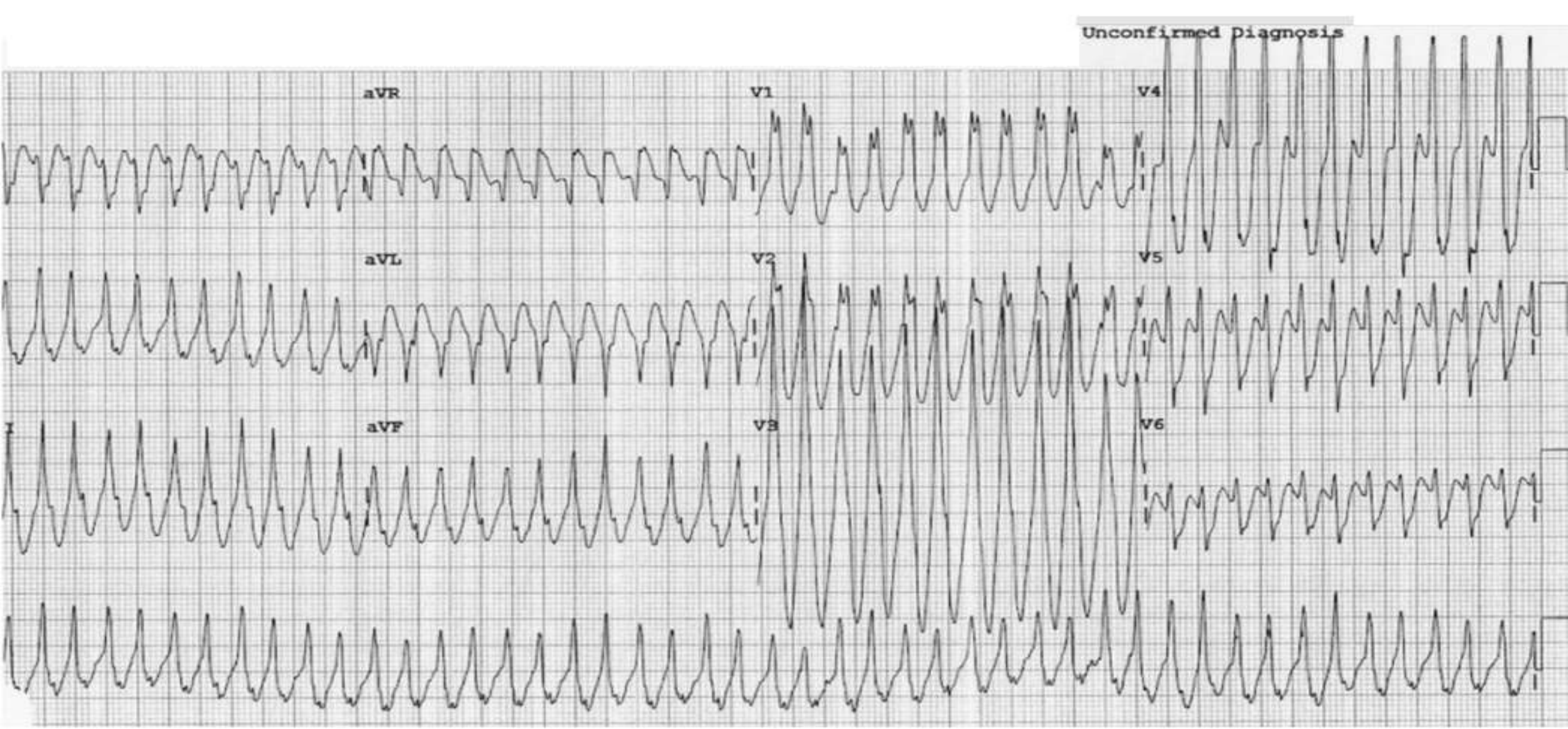
ECG features of AVRT with antidromic conduction:

Rate usually 200-300 bpm

Wide QRS complexes due to abnormal ventricular depolarisation via AP



Antidromic AVRT: Regular broad complex tachycardia



Antidromic AVRT in a 5-year old boy with WPW Regular, broad complex tachycardia at ~280 bpm; this would be very difficult to distinguish from VT However, given the child's age, VT is very unlikely: > 95% of broad complex tachycardias in children are actually some form of SVT with aberrancy. Treatment vagal manoeuvres.

Treatment of antidromic AVRT

This rhythm can be difficult to distinguish from VT, and if there is any doubt, we should presume a diagnosis of VT and treat accordingly

In stable patients, drug therapy should be targeted at the AP

Procainamide (class I) would be our first line antiarrhythmic. Ibutilide (class III) and amiodarone are second-line options, but their effectiveness is less established

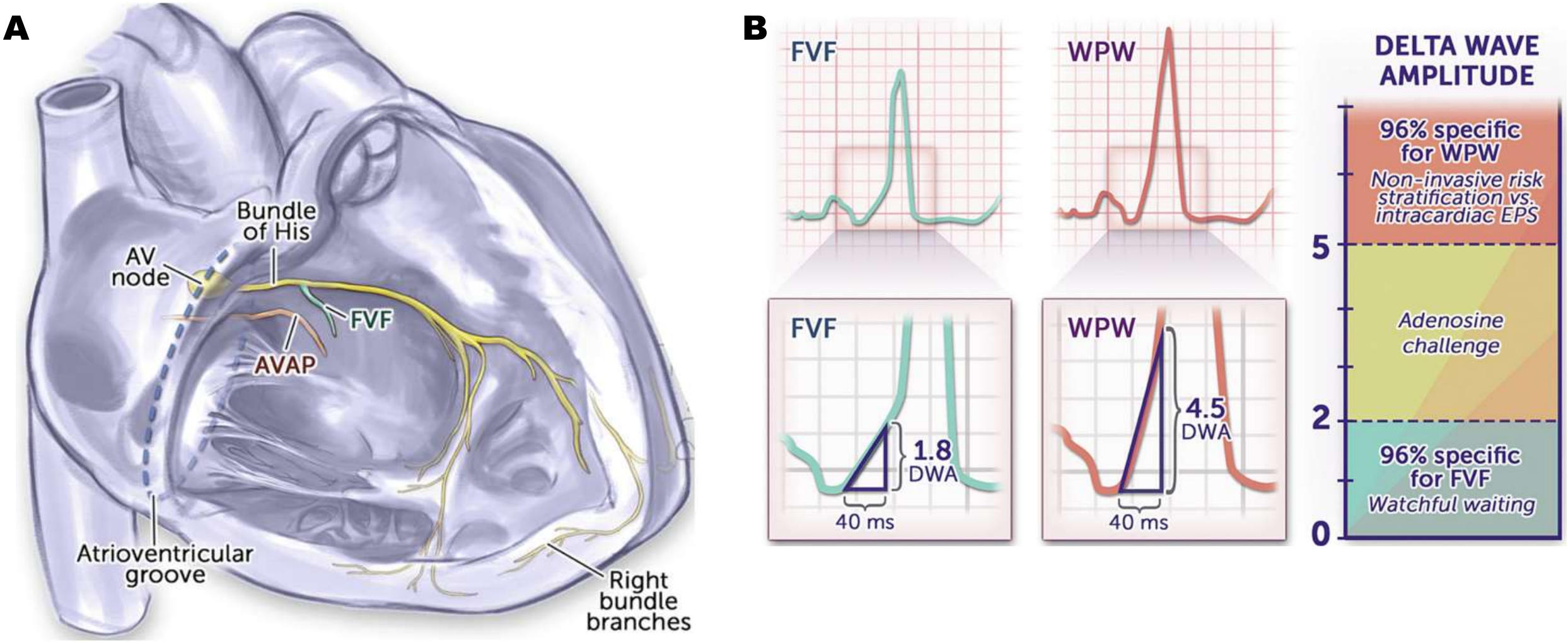
DC cardioversion may still be required if drug therapy fails

The re-entry circuit involves the AV node — why can’t we use AV nodal blocking agents?

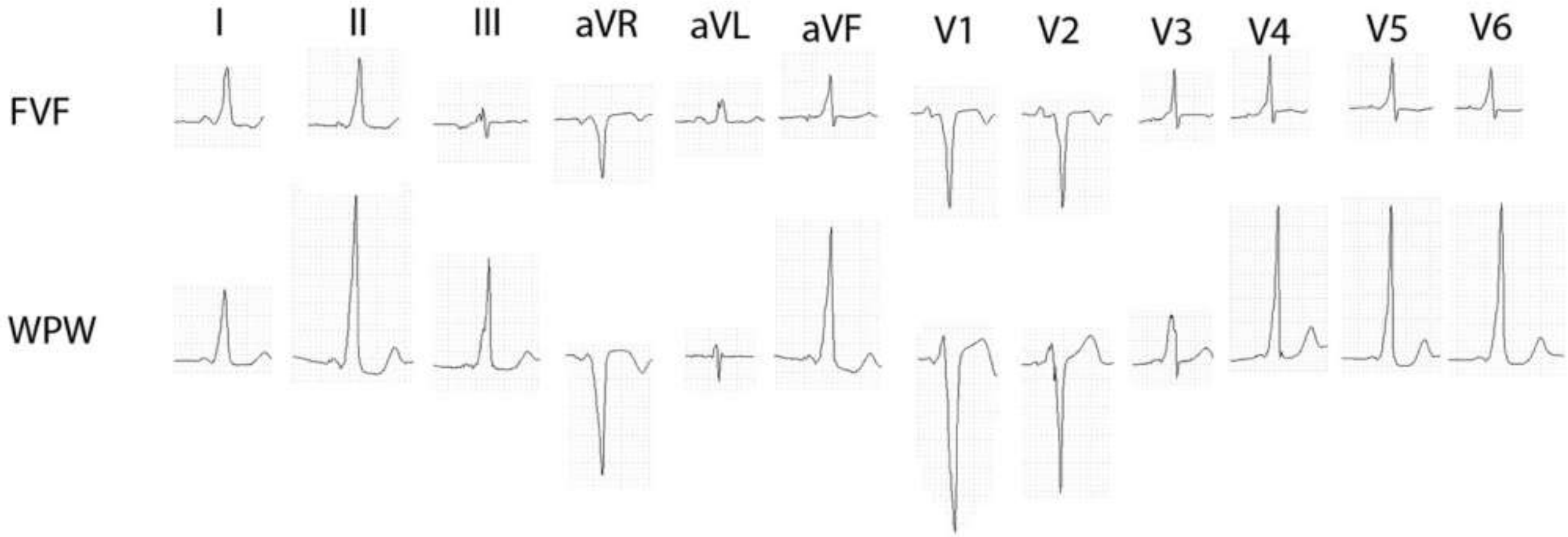
II. **ART Antidromic Reciprocating Tachycardia** patients with ART have a critical anatomic requirement (> 4 cm) between an antegrade bypass tract limb and a retrograde AV nodal limb. In ART utilizing a paraseptal AP a critical degree of slow conduction within the circuit provides unusual electrophysiologic substrate to overcome the expected anatomical constraints. A “latent Mahaim fiber” not cause preexcitation but can support ART. This is a rare form of wide complex tachycardia in children with WPW. The incidence and electrophysiologic characteristics of ART in children with WPW have not been well described. ART is a rare finding in children undergoing EP study. Over half of the patients with ART were found to be high risk and multiple AP were uncommon. Unlike the adult population, ART occurred commonly with septal APs.(**Scott R Ceresnak 1, Ronn E Tanel, Robert H Pass, Leonardo Liberman, Kathryn K Collins, George F Van Hare, Gregory J Gates, Anne M Dubin. Clinical and electrophysiologic characteristics of antidromic tachycardia in children with Wolff-Parkinson-White syndrome. Pacing Clin Electrophysiol. 2012 Apr;35(4):480-8. doi: 10.1111/j.1540-8159.2011.03317.x**.)

III. **FVF Fasciculoventricular Fiber** The etiology of preexcitation in patients with an anteroseptal preexcitation pattern, whether because of a benign FVF or because of potentially serious WPW syndrome, can be noninvasively deduced using the surface ECG. **A higher delta wave amplitude is an independent risk factor for the presence of WPW syndrome** and can accurately distinguish WPW syndrome from a FVF with good test accuracy characteristics. (**Edward T O'Leary 1, Elizabeth S Dewitt 1, Douglas Y Mah 1, Kimberlee Gauvreau 1, Edward P Walsh 1, Vassilios J Bezzerides 2 Differentiation of fasciculoventricular fibers from anteroseptal accessory pathways using the surface electrocardiogram. Heart Rhythm. 2019 Jul;16(7):1072-1079. doi: 10.1016/j.hrthm.2019.02.011**.)

Delta Wave Amplitude (DWA) in fasciculoventricular fiber(FVF) vs. WPW



A: Similar anatomic locations of a fasciculoventricular fiber (FVF) and an atrioventricular (AV) accessory pathway (AVAP) in the right anteroseptal region of the AV groove. **B**: Left: Example of delta wave amplitude (DWA) measurements in an FVF vs AVAP (Wolff-Parkinson-White [WPW] syndrome). Right: Proposed management algorithm. EPS 5 electrophysiology study



Examples of surface electrocardiograms. QRS morphologies on standard 12-lead ECGs of a patient with an isolated fasciculoventricular fiber (**FVF**; top row) and a patient with a right anteroseptal atrioventricular accessory pathway (Wolff-Parkinson-White [WPW] syndrome; bottom row)..

The etiology of preexcitation in patients with an anteroseptal AVAP pattern, whether because of a benign FVF or because of potentially serious WPW syndrome, can be noninvasively deduced using the surface ECG. A higher DWA is an independent risk factor for the presence of WPW syndrome and can accurately diagnose WPW syndrome with good test accuracy characteristics. Prospective validation is needed, these findings along with additional noninvasive testing may spare patients with benign FVFs from undergoing unnecessary intracardiac evaluations and their associated risks.

IV. NFF Nodofascicular Fiber: The surface ECG performed during tachycardia does provide clues to the presence of a nodofascicular fiber although it is not diagnostic. The ECG during nodofascicular fiber tachycardia is sufficiently distinctive to serve as a guide in the differential diagnosis of left bundle branch block configuration tachycardia. In this regard, being aware of a possible nodofascicular fiber before electrophysiologic study might facilitate the evaluation

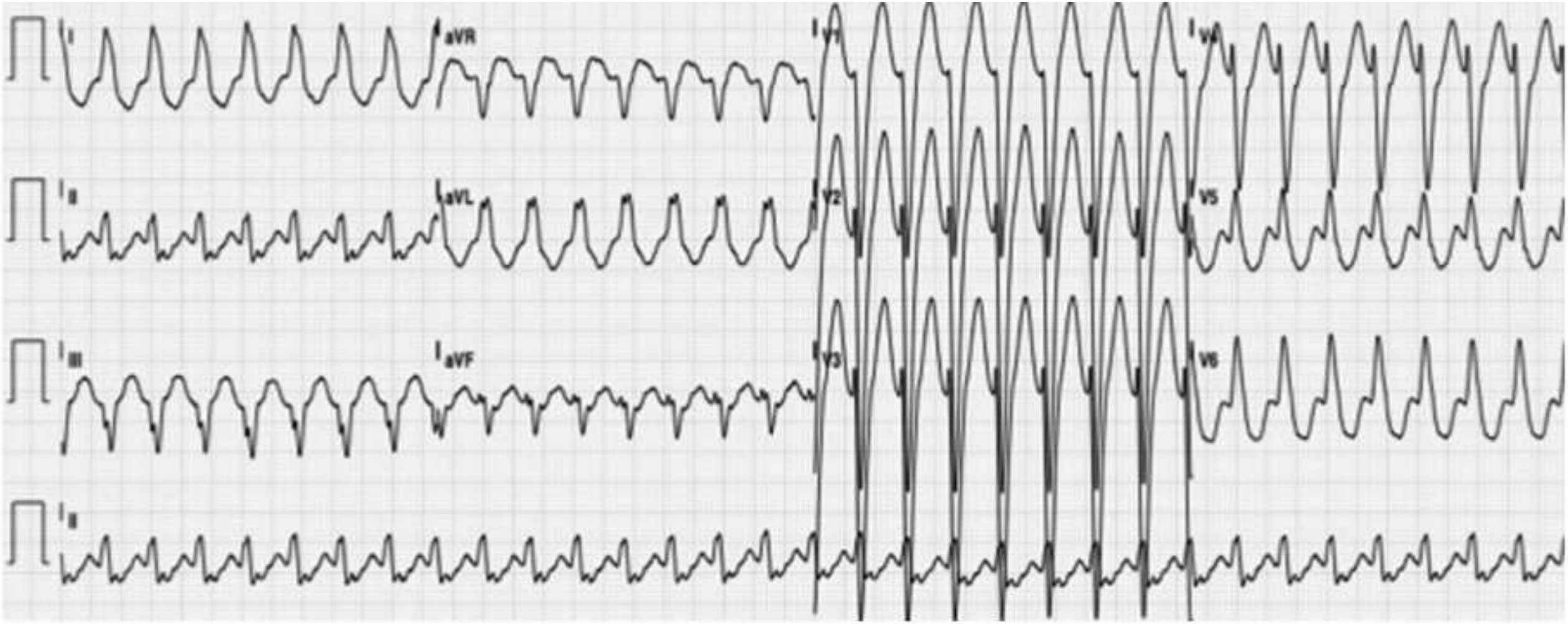
Electrocardiographic Characteristics in NFF Left Bundle Branch Block

1. Cycle length 220 to 450 ms in 100% of cases
2. QRS duration ≤ 150ms in 100% of cases
3. QRS Axis between 0° to - 75° in 100% of cases
4. R in lead I > 90% of cases
5. rS in lead VI in 100% of cases
6. Precordial transition after lead V4in 100% of cases **1**

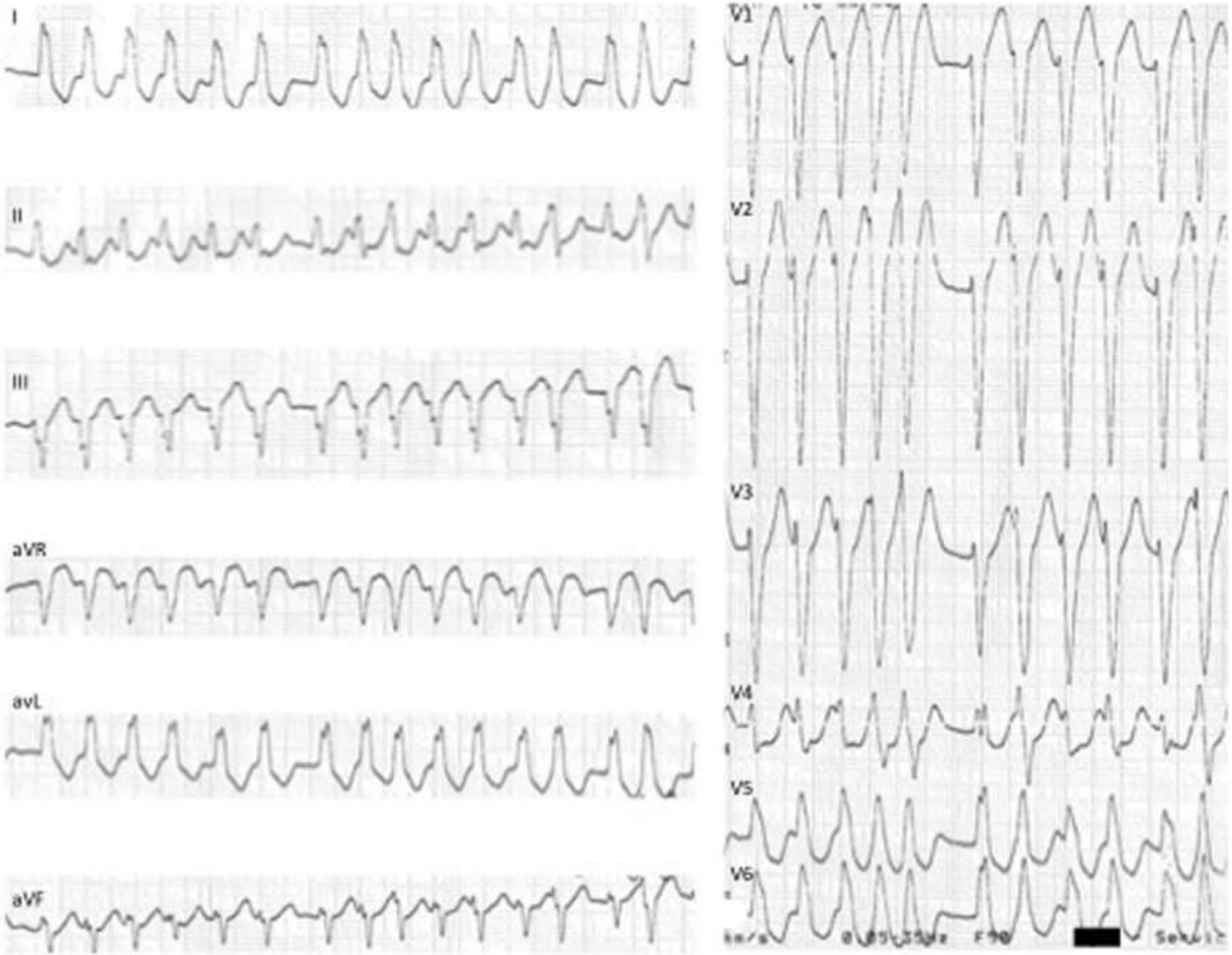
1. G H Bardy, J M Fedor, L D German, D L Packer, J J Gallagher, Surface electrocardiographic clues suggesting presence of a nodofascicular Mahaim fiberJ Am Coll Cardiol. 1984 May;3(5):1161-8. doi: 10.1016/s0735-1097(84)80173-4.

V. **NVF Nodoventricular Fiber or Nodoventricular Mahaim Fiber: The differentiation between a NVF and an AP with long conduction times and decremental properties could be very difficult even at detained electrophysiologic study. The finding that the S-Delta interval (this is the interval between the stimulus artifact and the onset of the delta wave during atrial pacing from two atrial sites) could become shorter than the A2HB-Delta interval (the retrograde atrial electrogram on the His channel and the onset of the delta wave during ventricular pacing) provides strong evidence that this AP was not connected to the AV node but arose directly from the atrial tissue of the posteroseptal region (W J Schoen, O Fujimura. Variant preexcitation syndrome: a true nodoventricular mahaim fiber or an accessory atrioventricular pathway with decremental properties? J Cardiovasc Electrophysiol. 1995 Dec;6(12):1117-23. doi: 10.1111/j.1540-8167.1995.tb00390.x.)**

VI. AVF Atrioventricular Fiber



12-lead ECG: regular tachycardia, 190 bpm, wide QRS (134ms), typical left bundle branch block pattern, -30◦ axis, monophasic R in DI, rS in V1 and RS transition in V5, suggestive of tachycardia of atrioventricular reentry mediated by VA-M. VA-M: Mahaim-type accessory pathway.



VA-M-mediated pre-excited atrial fibrillation: irregular tachycardia, 230 bpm, with wide QRS and different morphologies (particularly in V4 and V5). VA-M: Mahaim-type accessory pathway.

VA-M exhibit NAV-like properties and are associated with two types of tachycardias: SVT in which VA-M is part of the tachycardia circuit and SVT in which VA-M is not part of the circuit but contributes to ventricular activation. In general, the VA-M are located in the lateral tricuspid annulus.

The mapping of VA-M can be achieved with multiple techniques, mapping and ablation of the site with M potential in the tricuspid ring are the ones with the highest rate of success. The occurrence of BAD is considered a predictor of success. The post-ablation ECG is important in confirming the procedure success.

On the basis of the above, in the interest of accuracy and fairness, the eponyms such as Mahaim fiber should be abandoned and that all pathways should be identified by their anatomical-physiological properties. The specificity of the term used require s detailed intracardiac electrophysiological testing. However, a general term is needed until proof of mechanism is available. This is where the label “Mahaim fiber” has been convenient, in part because of its brevity and unfortunate tradition. The term **“atypical preexcitation pattern”** when there is evidence of preexcitation in sinus rhythm or tachycardia that is not characteristic of the usual WPW pattern. In summary, a qualitative description of the preexcitation pattern while in sinus rhythm should not be complicated: absent, typical, and atypical should suffice for the nonelectrophysiologist. The electrophysiologist understands that no preexcitation may exist in sinus rhythm in patients with AFF or nodofascicular fiber pathways; typical preexcitation refers to the WPW pattern; and atypical preexcitation refers to that created by decrementally conducting AFF, NVF, or FVF pathways. The specific term will depend on additional information from ECGs and electrophysiological testing (see Table 1). The above schema will help practitioners at all levels communicate more effectively when describing preexcitation patterns and their associated regular, wide complex tachycardias.

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Understanding paper speeds

Paper output speed is the rate at which the ECG machine produces a trace

Standard output is 25mm per second

If a different paper speed is used, standard rate calculations will have to be modified appropriately

The standard paper speed is 25mm/sec:

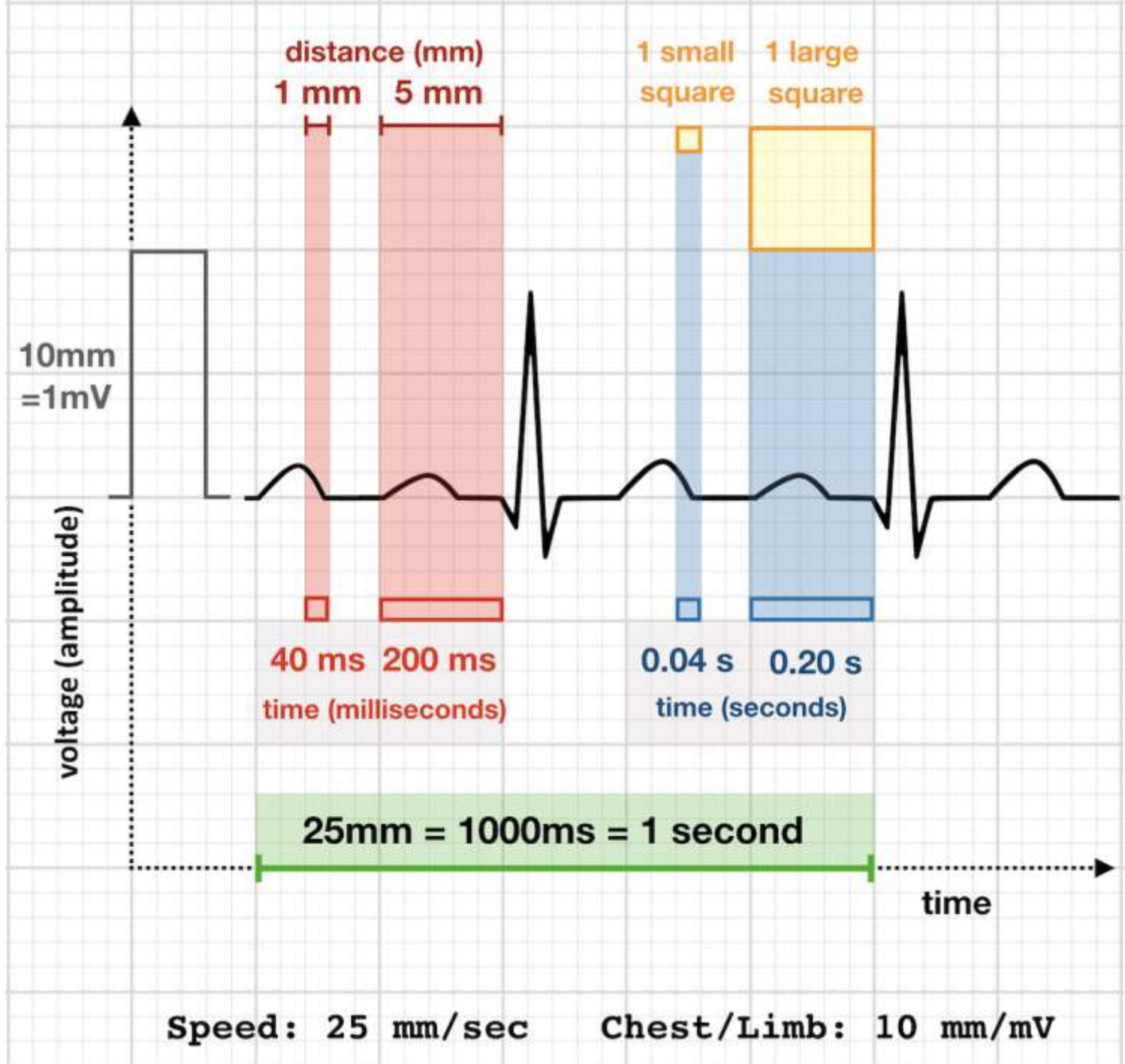
- 1 SMALL square (1mm) = 0.04 sec (40ms)
- 5 SMALL squares (5mm) = 1 LARGE square = 0.2 sec (200ms)
- 5 LARGE squares = 1 second

At standard paper speed of 25mm/sec, the rhythm strip comprises of:

250 SMALL squares = 50 LARGE squares = 10 seconds

Before calculating rate in beats per minute (bpm), we should understand that a rhythm strip recorded for 1 minute will therefore compromise:

1500 SMALL squares = 300 LARGE squares = 1 minute


distance (mm)
1 mm
5 mm
1 small square
1 large square
10mm =1mV
voltage (amplitude)
40 ms
200 ms
time (milliseconds)
0.04 s
0.20 s
time (seconds)
25mm = 1000ms = 1 second
time
Speed: 25 mm/sec
Chest/Limb: 10 mm/mV

Calculating rate

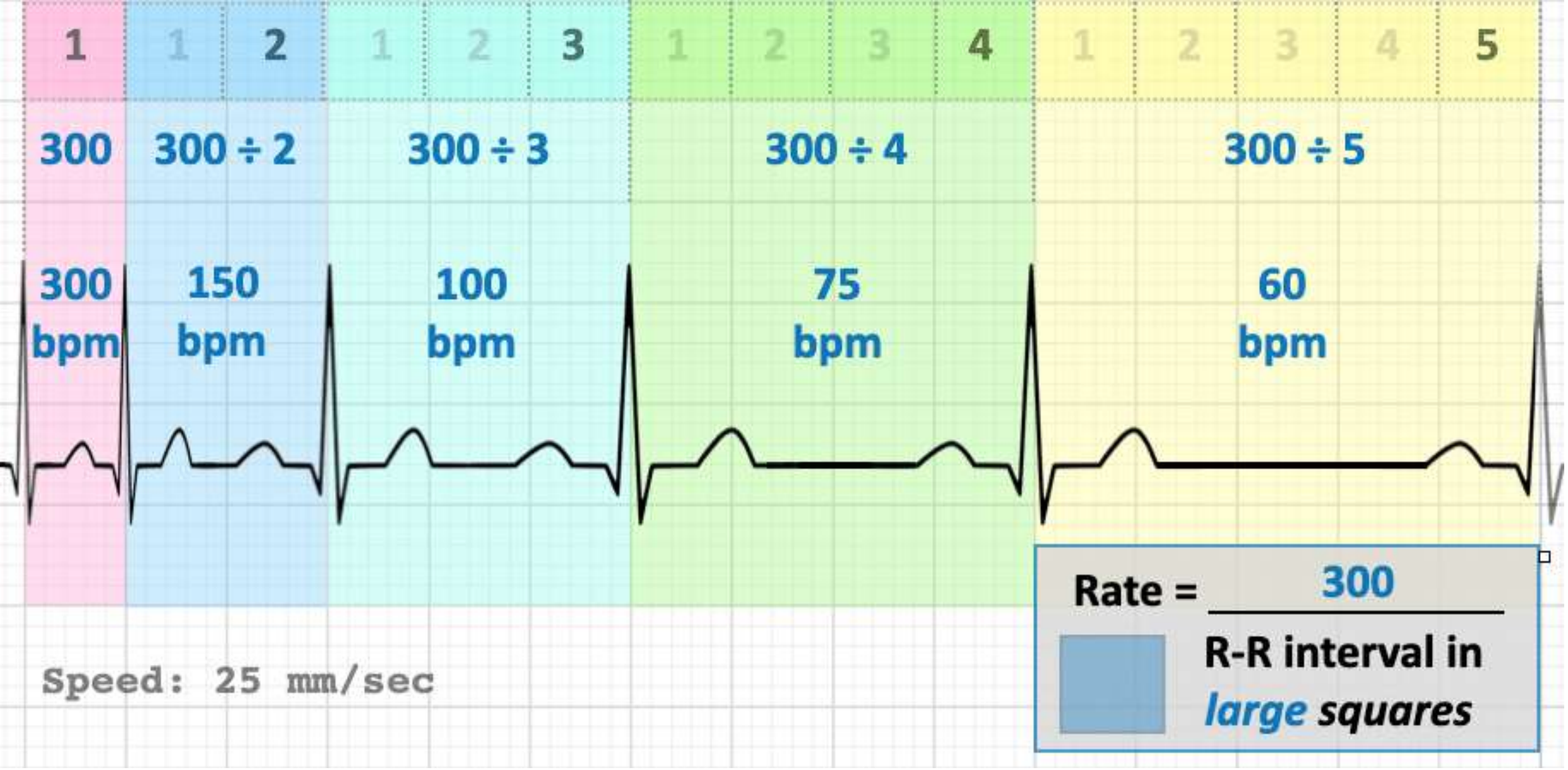
There are **three** main methods of calculating ECG rate. There is no specific *best* method, and preference varies between clinicians. However, certain methods may be better suited for rhythms such Brady arrhythmias or tachyarrhythmia's.

1) Large square method

Recall above that 300 large squares is equal to 1 minute at a paper speed of 25mm/sec

We can thus calculate bpm by dividing 300 by the number of LARGE squares between each R-R interval (space between two consecutive R waves = one beat)

For example, two large squares between each R-R interval implies a rate of 150 bpm, three implies a rate of 100 bpm and so forth:

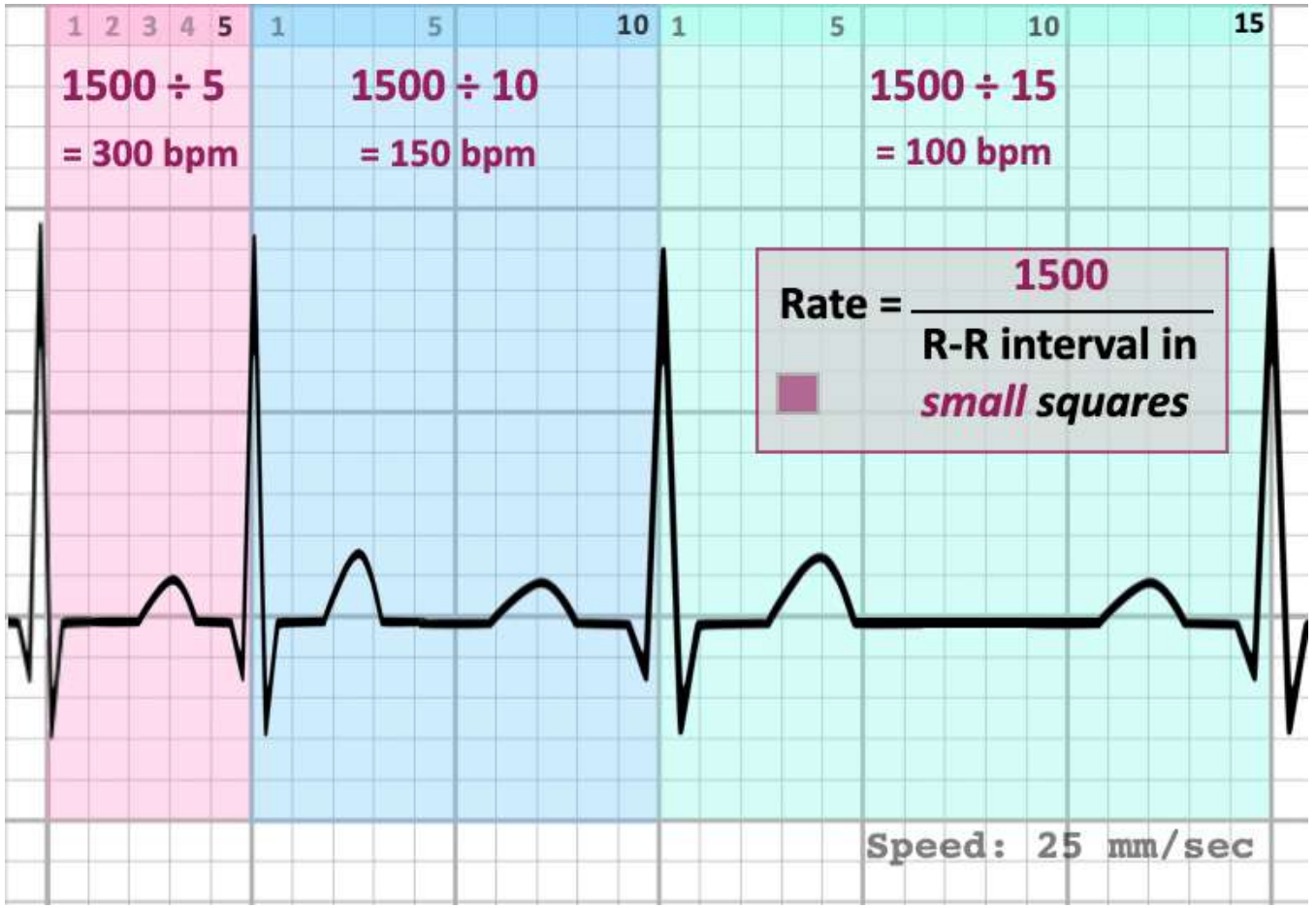


Large square method: Divide 300 by the number of large squares between R-R interval. Useful for regular rhythms Useful as quick calculation for regular rhythms at regular rate

2) Small square method

Similar to above, except 1500 is divided by the number of SMALL squares between consecutive R waves

For example, 10 small squares between R-R interval implies a rate of 150 bpm, 15 implies a rate of 100 bpm, and so forth



Small square method: Divide 1500 by number of small squares between R-R interval.

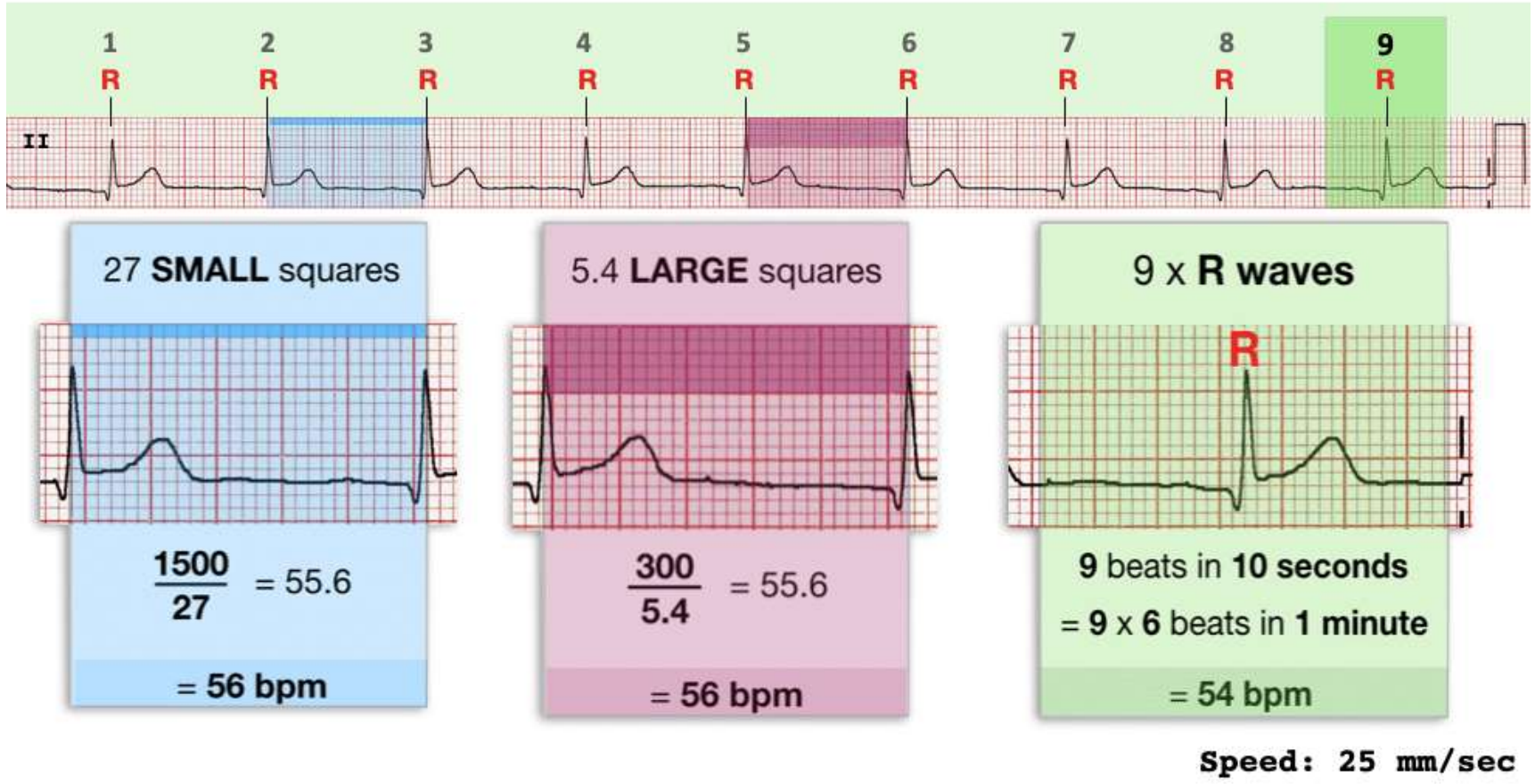
Useful for very fast regular rhythms, as likely to provide more accurate rate than large square method

3) R wave method

Rate = **Number of R waves (rhythm strip) X 6**

The number of complexes (count R waves) on the rhythm strip gives the *average* rate over a ten-second period. This is multiplied by 6 (10 seconds x 6 = 1 minute) to give the **average** beats per minute (bpm)

Useful for slow and/or irregular rhythms



Comparison of three methods: The R wave method is often easiest as a quick calculation