OVERVIEW OF THE ECG

The tracing shows what appears to be a very fast (232bpm), wide QRS (±200ms), irregular rhythm with at least 2 QRS morphologies. No clinical information is given.

MORE DETAILED ANALYSIS OF THE ECG

When confronted with a bizarre, rapid ECG tracing which looks like ventricular tachycardia or fibrillation, particularly in the setting of a patient on a monitor, always consider the possibility of artefact. Careful inspection of an artefactual recording in all available leads will almost always reveal normal QRS complexes superimposed on the false ones (Figure 1). Their origin can be confirmed by their regularity and similarity to the R-R intervals before or after the event and the lack of a reactive sinus tachycardia following the abnormal tracing. The artefact is usually short-lived. The patient will not have any symptoms related to the event.

There is no evidence of normal QRS complexes on this ECG. In addition, it would be unusual for artefact to persist long enough to be recorded on a 12 lead ECG. Artefact can therefore be excluded.

The differential diagnosis is therefore that of an irregular wide QRS tachycardia, which includes all the other possibilities given. In contrast to a regular wide QRS tachycardia, atrial fibrillation is usually the underlying mechanism, not ventricular tachycardia (VT) (Figure 2).

All the QRS complexes are negative in the mid-chest leads and positive in V6. However, AF with left bundle branch block

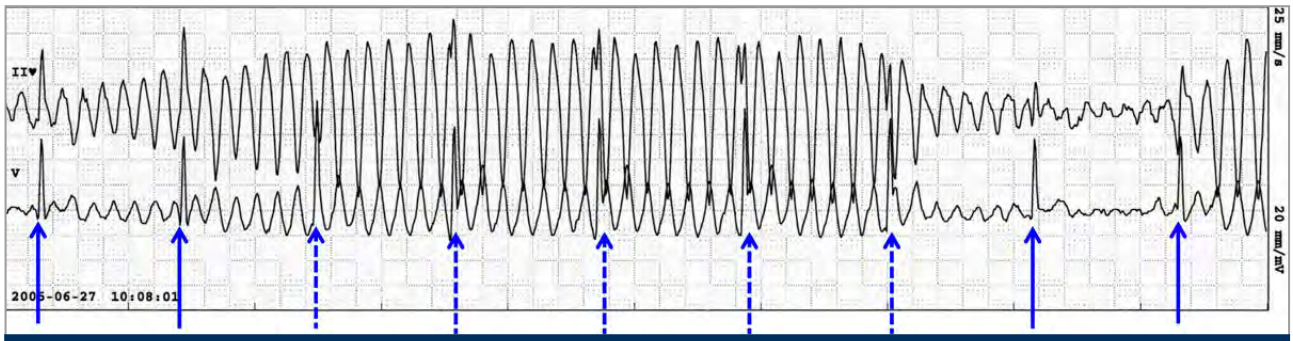

FIGURE 1: Monitor strip showing movement artefact. The arrows indicate the normal QRS complexes hidden by the artefact.

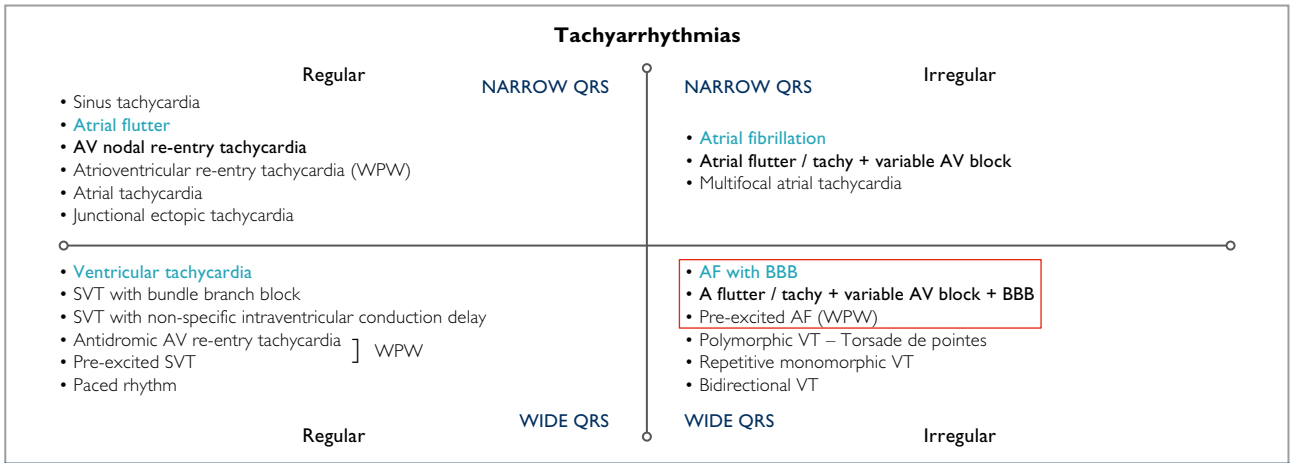

FIGURE 2: Tachyarrhythmias can be divided into 4 quadrants, depending on whether the rhythm is regular or irregular, and whether the QRS is narrow (= <100ms) or wide (= >120ms). Most irregular, wide QRS tachycardias are due to atrial fibrillation, with QRS morphology dependent on the presence of bundle branch block or pre-excitation.

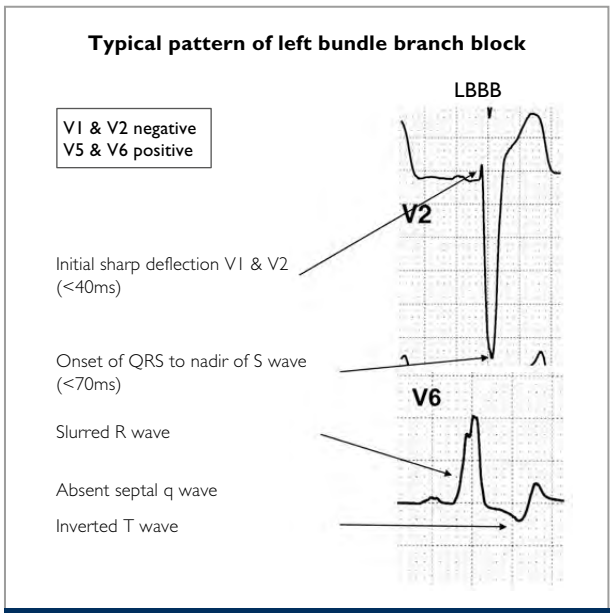

FIGURE 3: Left bundle branch block is best characterised by the pattern in V1 - V2, by the small, narrow r wave and relatively sharp descent of the S wave. V6 can be misleading as many ventricular tachycardias and pre-excited rhythms can mimic the "M" pattern, which is not always present in otherwise typical LBBB.

(LBBB) is excluded by the time from the onset of the QRS in V2 to the nadir of the S wave = 95ms (LBBB <70ms) (Figure 3).

While the varying QRS morphology superficially resembles torsade de pointes (TDP), there is no twisting from negative to positive in any of the leads. Torsade de pointes usually starts with a short-long-short sequence (R on T phenomenon). It is unusual to record TDP on a 12 lead ECG as it is usually transient, or else degenerates into ventricular fibrillation (Figure 4).

Bidirectional VT is characterised by a regular alternation of QRS axis, usually best seen in the limb leads (Figure 5). This is not bidirectional VT.

We are therefore left with polymorphic VT or pre-excited AF, conducted rapidly via an accessory pathway in a patient with WPW syndrome.

Polymorphic VT occurs most commonly in the setting of an acute coronary syndrome, particularly a STEMI. It tends to be non-sustained (last less than 30 seconds), but may rapidly degenerate into ventricular fibrillation (VF) (Figure 6). Like TDP, it is unusual to record it on all 12 leads of an ECG. The mor-

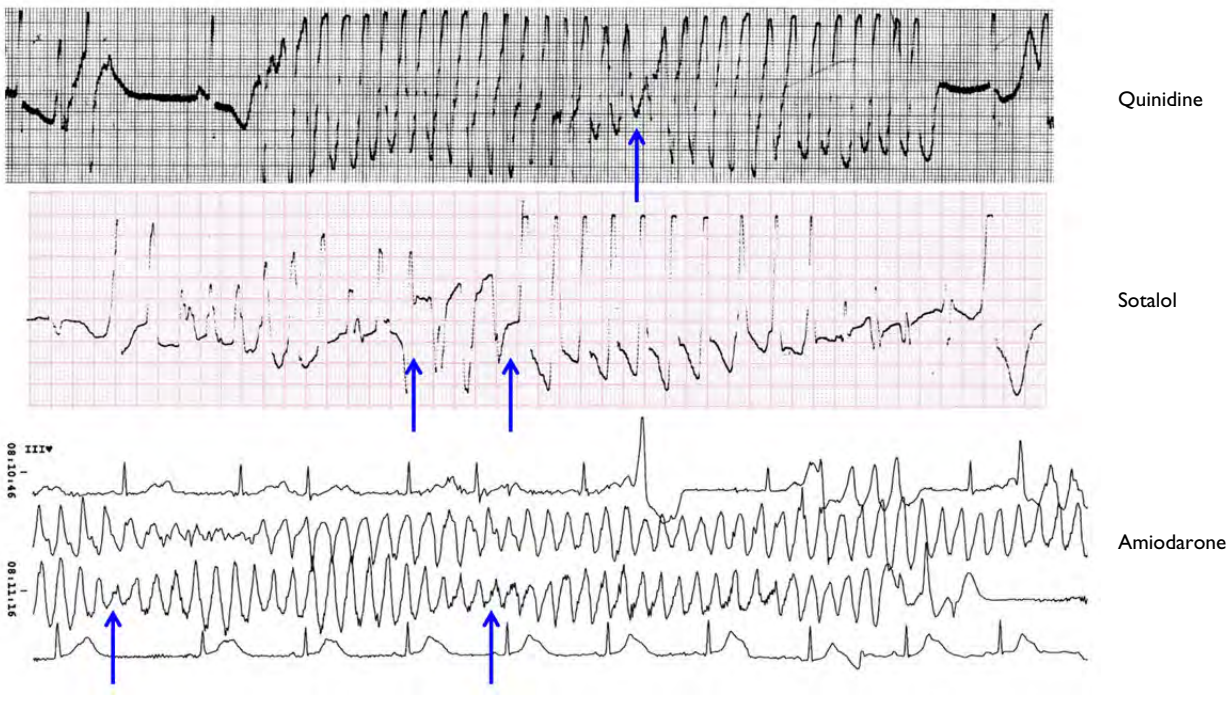

FIGURE 4: Torsade de pointes was first described by Dessertenne in 1966, based on the twisting pattern of the QRS complexes. The term is now reserved for rhythms with this pattern associated with QT prolongation. TDP is most commonly induced by the wide variety of drugs which prolong repolarisation, usually by blocking the I_{Kr} channel. It is usually initiated by a pause following a premature beat. TDP also occurs in the congenital long QT syndromes.

The arrows in these 3 examples, induced by antiarrhythmic drugs, indicate the points of reversal of the QRS axis.

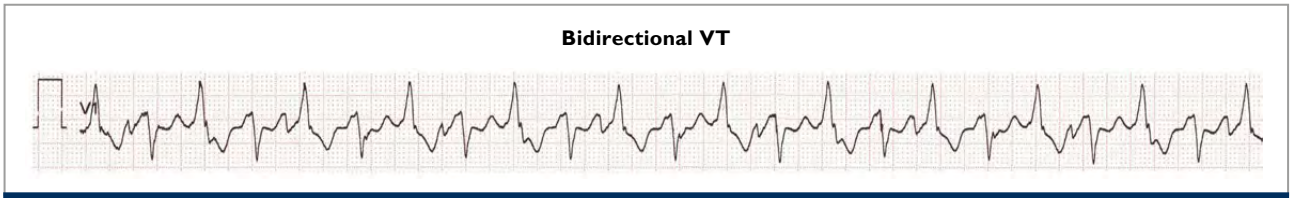

FIGURE 5: Bidirectional ventricular tachycardia in a patient with catecholaminergic polymorphic VT.

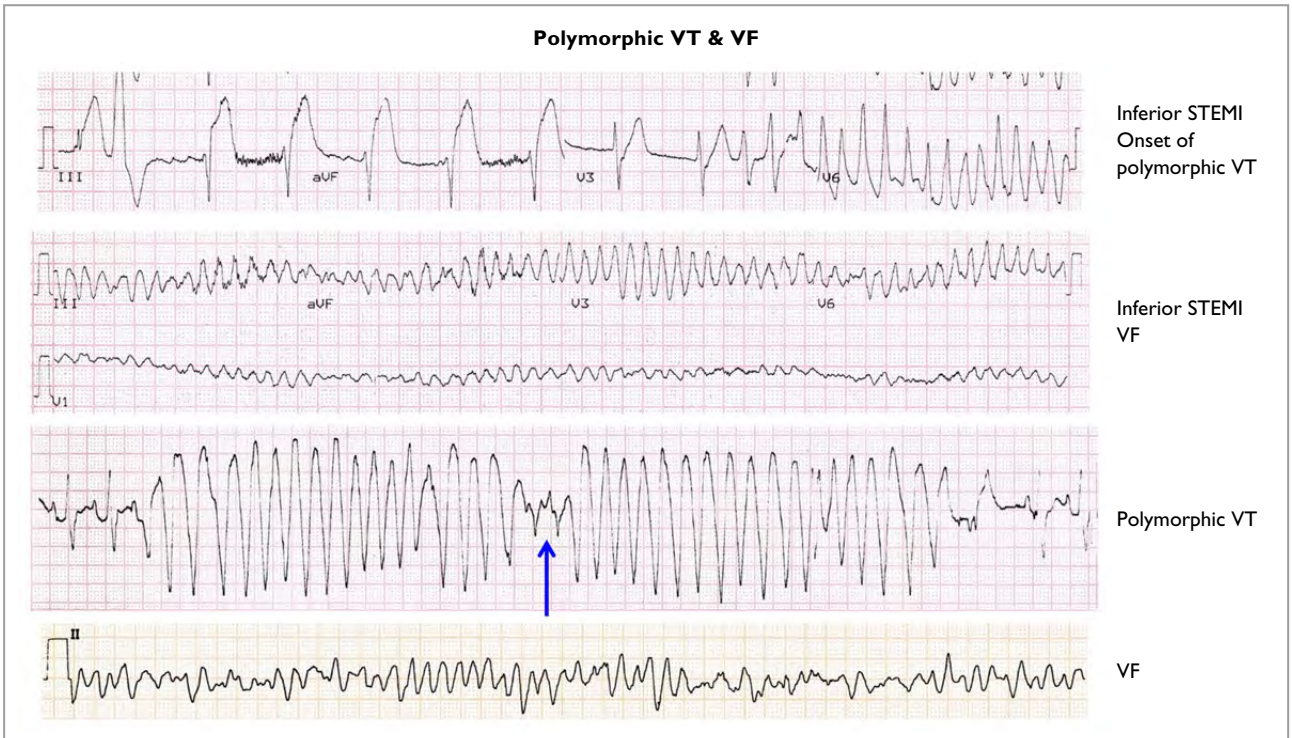

FIGURE 6: Ischaemia-induced polymorphic VT which may be self-limiting or degenerate into ventricular fibrillation (VF). While it may resemble TDP, the axis does not necessarily rotate (arrow). VF is an extremely rapid, chaotic rhythm, not commonly recorded on a 12 lead ECG.

phology of the complexes also tends to vary more than in the case of this ECG.

The most likely diagnosis is therefore (c): Pre-excited atrial fibrillation.

The patient was a 30-year-old woman, working in the Winelands. She was previously well apart from intermittent palpitations. She developed rapid palpitations, dizziness and near-syncope and was admitted to Paarl Hospital where the ECG was recorded before cardioversion. After cardioversion, her sinus rhythm ECG was diagnostic for WPW syndrome (Figure 7). The PR interval is extremely short (60ms); the end of the P wave merges with the beginning of the QRS. The QRS is very wide, at least 200ms. The pattern resembles LBBB, particularly in V6. However, the initial r wave in V1-2 is 70ms (LBBB = <40ms) and the time from the onset to the nadir of the QRS is 115ms (LBBB = <70ms). The slurred onset of the QRS is due to delta waves with a high degree of pre-excitation. The pseudo LBBB pattern with a late QRS transition suggests a right free wall AP.

Subtle changes in QRS morphology are often present in patients with pre-excited AF due to varying degrees of fusion between AP and AV nodal conduction. In this ECG, there are 2 distinct QRS morphologies; they are both wide and pre-excited with similar axes in all the leads. This suggests a strong possibility that there are 2 distinct APs, both on the right side, in close proximity to one another. Their refractory periods are similar, in that the shortest R-R interval of the larger complexes is 230ms and the smaller 210ms during atrial fibrillation. This indicates a risk of degeneration into VF[1] (Figure 8). Ablation of the pathways was therefore mandatory.

Two right free wall APs were successfully ablated (Figure 9).

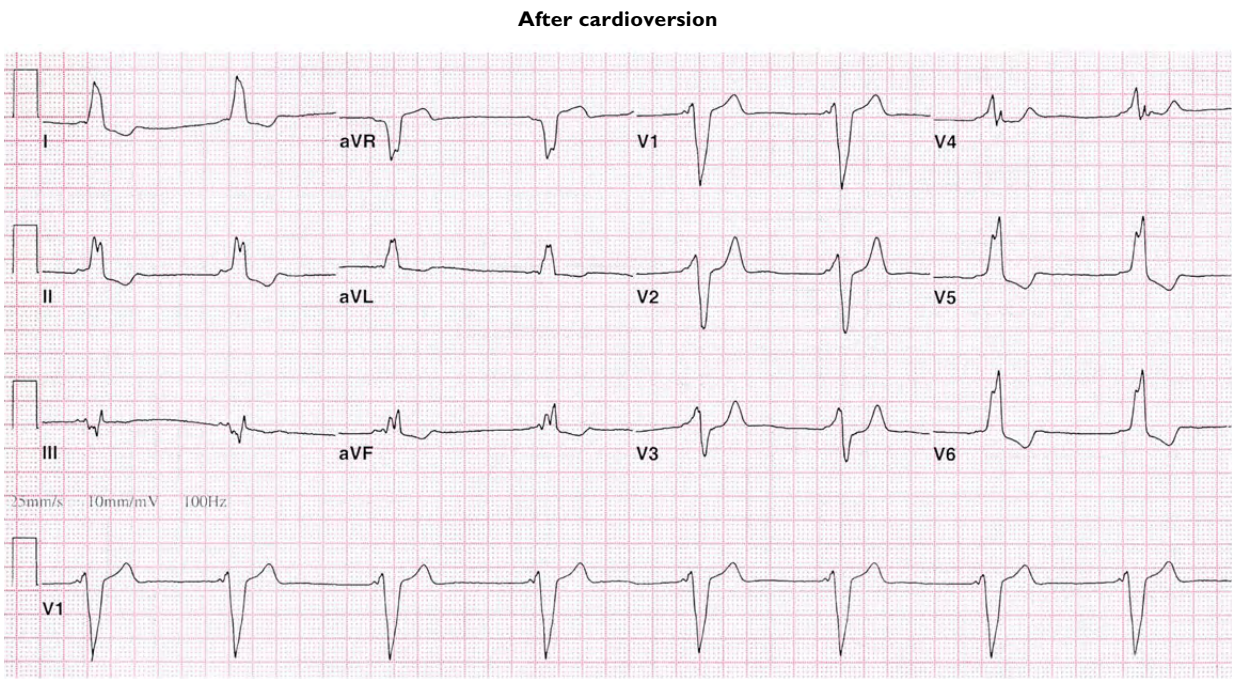

FIGURE 7: The ECG in sinus rhythm after cardioversion shows a clear-cut Wolff-Parkinson-White pattern (see text).

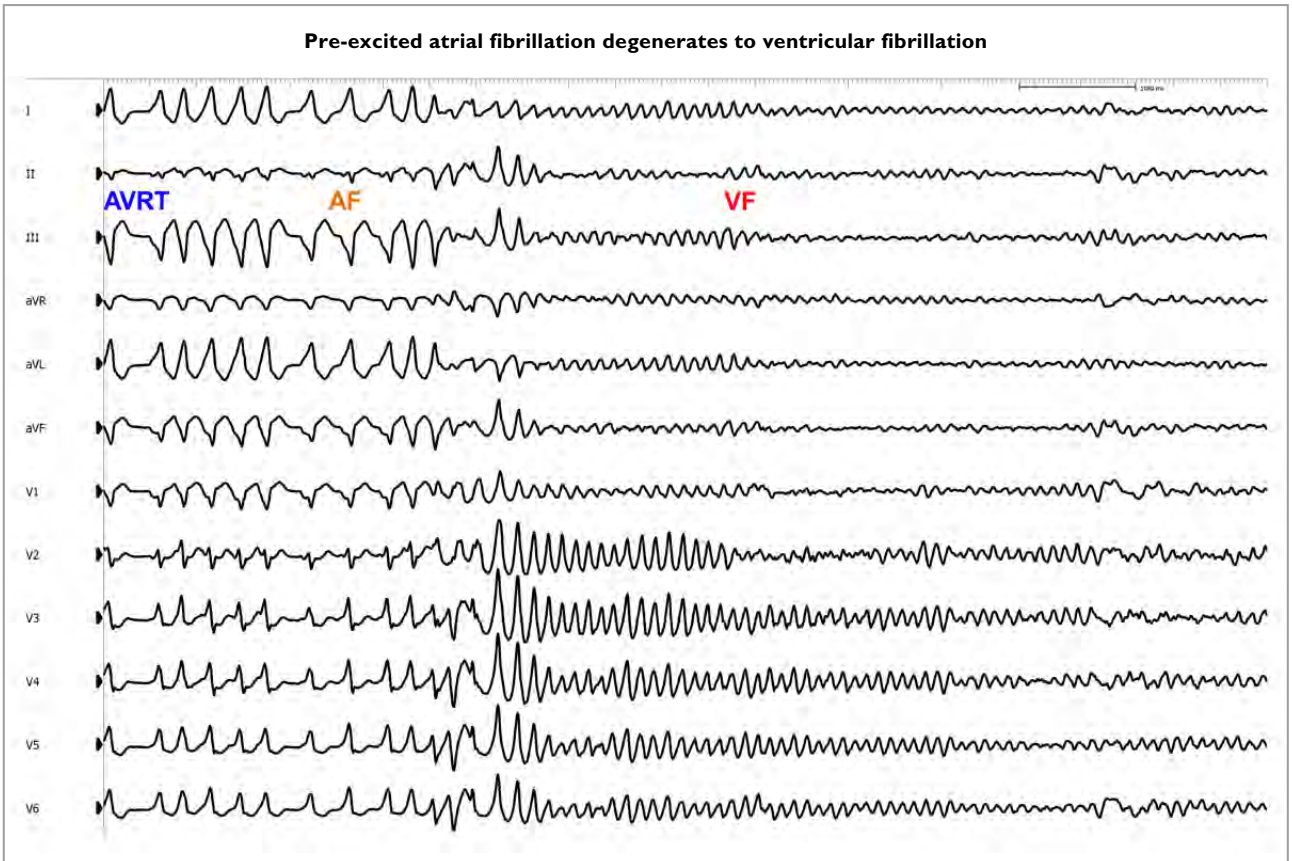

FIGURE 8: Twelve lead ECG of a patient with WPW recorded in the EP lab. Atrioventricular re-entry tachycardia (AVRT) was induced, which triggered pre-excited atrial fibrillation. The AF rapidly degenerated into ventricular fibrillation.

APPROACH TO THE DIAGNOSIS OF IRREGULAR, WIDE QRS TACHYCARDIAS (Table 1)

Unlike regular, wide QRS tachycardias, in which the default diagnosis is ventricular tachycardia, the most common mechanism for an irregular, wide QRS rhythm is AF. The QRS morphology is determined by the characteristics of the conduction pathway(s) from the atria to the ventricles. Left or right bundle branch block, usually pre-existing, can be diagnosed from the QRS morphology. If one or more accessory pathways is functional, the QRS will differ from typical LBBB or RBBB. Depending on the balance of refractory periods between the accessory pathway (AP) and the AV node, the QRS may exhibit varying degrees of fusion or an occasional narrow QRS (Figure 10).

Pre-excited AF may occur in up to 20% of patients with WPW syndrome, in the absence of structural heart disease, even in children as young as 10 years. It is often triggered by rapid atrioventricular re-entry tachycardia and does not usually recur after ablation of the accessory pathway. Multiple APs are not uncommon and can occur in 10% of WPW patients. Pre-excited AF accounts for the approximately 0.25% annual risk of sudden cardiac death in symptomatic patients with WPW and all patients should be referred for an ablation.[2,3] The risk of sudden death in asymptomatic WPW patients is lower and routine invasive management in most asymptomatic patients with the Wolff-Parkinson-White ECG pattern is not required.[3]

TABLE I: Irregular wide QRS tachycardias.

ECG features
Sustained tachycardia, typical bundle branch block – favours AF + BBB
Sustained tachycardia, not typical bundle branch block – favours pre-excited AF
Occasional narrower beats and slight variation in QRS favour pre-excited AF
Sustained tachycardia over 180bpm – favours pre-excited AF
Polymorphic VT usually non-sustained (unless > VF)
Torsade de pointes – twisting pattern and non-sustained (unless > VF)
Alternating axis – > bidirectional VT (catecholaminergic, digoxin)
Clinical context
History suggesting current or past MI – > polymorphic VT
Age – Youth favours pre-excited AF, but VT can occur from infancy to senility
Prior cardiac symptoms
Episodes of rapid palpitations, regular or irregular, favour AF
SOB, chest pain – > polymorphic VT
QT prolonging drug – > torsade de pointes
Signs of structural heart disease or heart failure – polymorphic VT
ECG in sinus rhythm
Delta waves confirm WPW
Pathological Q waves of old MI suggest VT, but beware pseudo-infarct patterns in WPW
Other: E.g. ARVC patterns, conduction problems (sarcoid), HOCM
Other tachycardia ECGs
Orthodromic or antidromic AVRT – WPW
AV dissociation
Different QRS pattern favours VT

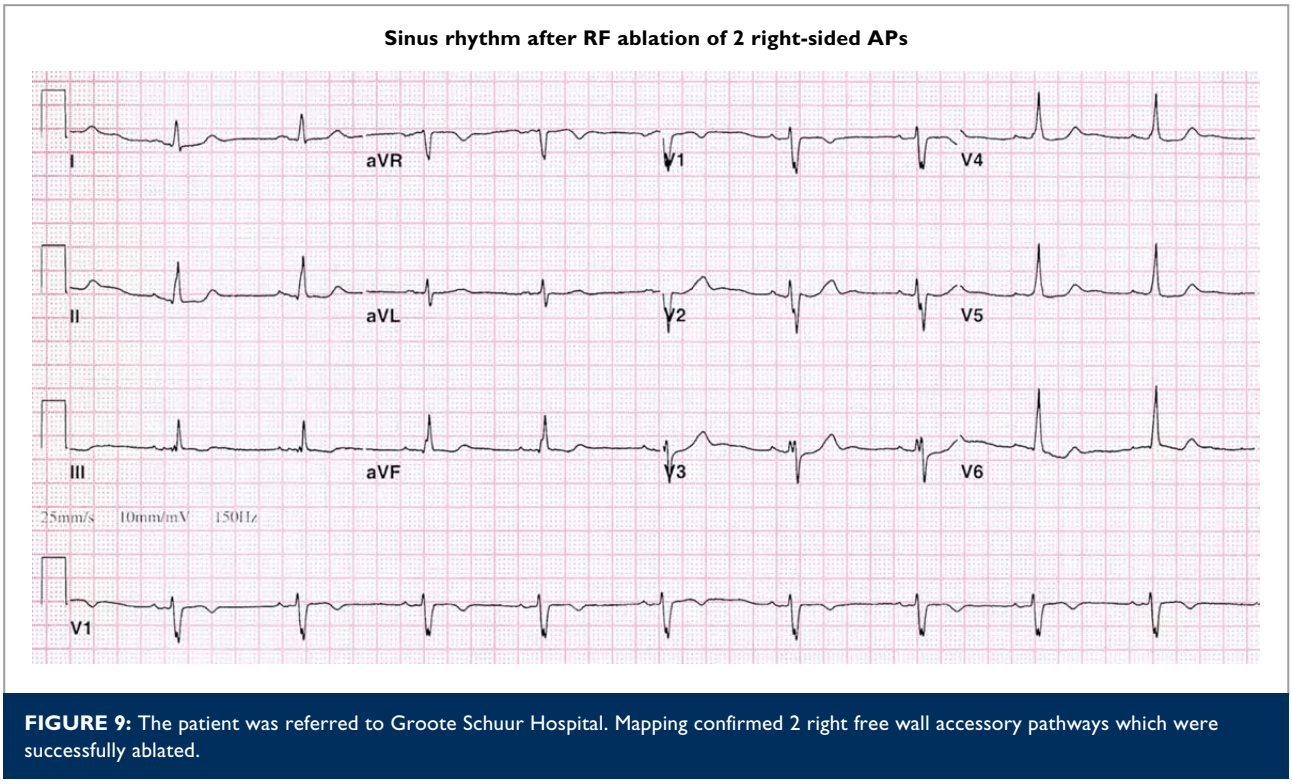

FIGURE 9: The patient was referred to Groote Schuur Hospital. Mapping confirmed 2 right free wall accessory pathways which were successfully ablated.

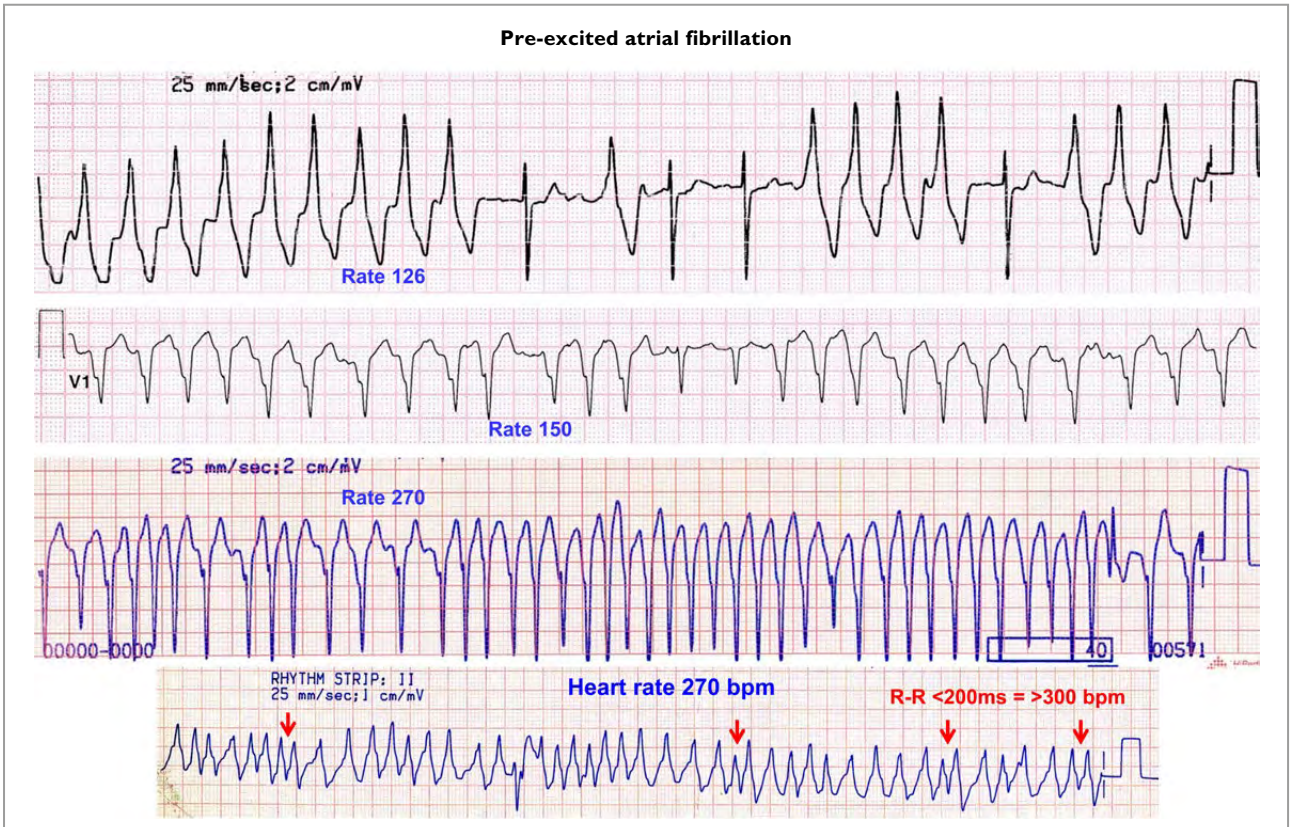

FIGURE 10: Pre-excited AF is not always rapidly conducted. The top panel shows pre-excited AF with a minimum R-R interval of 380ms, indicating a relatively safe accessory pathway refractory period. The pre-excited beats are interspersed with others conducted via the normal conducting system.

The second panel is from another patient with a relatively long AP refractory period, whereas the lower 2 are from patients with short refractory periods (<240ms), very rapid ventricular responses and a risk of degeneration into VF.

All the traces show the characteristic QRS variability due to variable degrees of fusion between conduction via the His-Purkinje system versus the accessory pathway(s).

CONCLUSIONS

- AF is the most common mechanism for sustained irregular wide QRS tachycardias, but other atrial arrhythmias, such as atrial flutter may be responsible.
- The QRS morphology is determined by pre-existing or rate-related bundle branch block, or accessory pathway(s).
- Polymorphic VT and torsade de pointes are usually non-sustained (<30 seconds), unless they degenerate into VF.
- Bidirectional VT is uncommon. It may be due to catecholaminergic VT[4] or digoxin toxicity.

Conflict of interest: none declared.

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ECG and **QUESTION** on page 124