

SYSTEMATIC ANALYSIS OF THE ECG

1. **Rate and regularity:** irregularly irregular with a ventricular rate of 144bpm.
2. **QRS complexes** are narrow, duration 80ms, with an axis of 60°. Although some minor variability in the QRS morphology is noted, there is normal R-wave progression, no pathological Q-waves and also no slurring suggestive of delta waves of antegrade accessory pathways.
3. **Atrial activity:** Normal sinus activity or normally formed P-waves cannot be identified. However, rapid atrial activity is obvious especially in the VI rhythm strip. Measurement of PR intervals is inappropriate and impossible because of the variable and inconsistent relationship between atria and ventricles.
4. **Other:** minor insignificant infero-lateral ST and T-waves abnormalities are noted. QT is unremarkable.

IN SUMMARY

The ECG shows a rapid supraventricular tachycardia (SVT) that is irregular.

FURTHER DISCUSSION

The differential diagnosis of an irregular SVT includes atrial fibrillation, atrial flutter with variable AV conduction and multifocal

atrial tachycardia. The latter can be excluded as it is associated with P-waves of various morphologies but which are discrete and "well-formed". In this ECG, the atrial activity appears to be almost continuous and very rapid with a time interval between 2 successive "peaks" in VI as short as 140ms (3½ small blocks) giving an instantaneous rate of over 425bpm. Whilst the appearance in segments of the ECG may be compatible with the "saw-tooth" pattern of atrial flutter, the rate here is faster than typical atrial flutter (usually 250-350bpm) and, more importantly, the atrial activity, if one follows it from beginning to end of the rhythm strip, is clearly variable and irregular (see Figure 1a).

Atrial flutter is probably the most stable pathological arrhythmia that can occur and can last for minutes to years. It is characterised by stability and a continuous stable recurring atrial pattern (see Figure 1b). Thus, this is not atrial flutter and, by exclusion and by default, must be atrial fibrillation. (Answer 1b)

The coarseness of the AF, sometimes described as being more organised, is likely to be due to the chance similar orientation of the multiple re-entry circuits that may be found in AF.

The clinical relevance in this patient of the AF and its management will be related to 1) the heart rate and 2) the associated risk of systemic thromboembolism.



FIGURE 1: Comparison of atrial electrical activity in atrial fibrillation and atrial flutter

1A: Atrial Fibrillation (ECG#25): In the rhythm strip, VI, note the variable morphology of the atrial activity. Often atrial fibrillation is more chaotic. This more "organised" AF may be mistaken for atrial flutter because of apparent regular activity. However, analysis over a longer period shows that the transient apparent regularity and morphology degenerates and is typical of AF.

1B: Atrial Flutter: This VI rhythm strip recorded in a different patient shows the very regular stable atrial activity of typical atrial flutter that does not vary from beginning to end of the ECG.

Ventricular Rate

A crucial observation in any patient with AF is the ventricular response rate. What determines this? The colloquial term of “rapid atrial fibrillation” is incorrect as variation in the electrical activity in the atria, which is always around 300-500 activities per minute irrespective of how the ventricles respond, does not determine the ventricular rate. The AF is always fast in the atria. So why is there, using the correct terminology, “AF with a fast ventricular response”? The ventricular rate is determined by the AV node, which is very sensitive to the autonomic nervous system. Parasympathetic innervations, via the vagus, and sympathetic stimulation, via nerves and especially circulating catecholamines, influence the filtering action of the AV node by decreasing and increasing respectively the number of impulses passing through the AV node (see Figure 2). If the AV node were a simple connection, like a wire, between the atria and ventricles and if it were not for its special processing and filtering function, AF, by conduction to the ventricles and causing ventricular fibrillation (VF), would be a uniformly fatal arrhythmia. We know that it is not. The exception is where the electrical connection between the atria and ventricle is not only the AV node but also an accessory pathway (Wolff-Parkinson-White) which does act like a short circuit or simple wire. In this situation, with pre-excited AF there is risk of VF and sudden death. (See ECG Quiz #5, SA Heart J: 2006 issue 4)

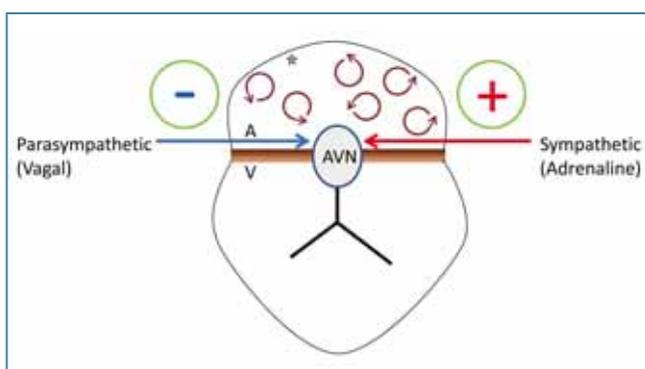


FIGURE 2: The AV node in atrial fibrillation

In atrial fibrillation, conduction of the rapid chaotic atrial electrical activity, which is related to multiple re-entry circuits, to the ventricles is dependent on the AV node. The autonomic nervous system determines the activity of the AV node with parasympathetic activity (e.g. vagal tone, digoxin) reducing conduction of impulses and sympathetic activity (mediated by catecholamines) promoting conduction and increasing the ventricular rate.

Therefore, rather than jumping to treating the AF, the clinical question should be: why is the ventricular rate so fast? What is driving the AV node? What is turning on the sympathetic system? This requires conscious exclusion of a number of clinical scenarios amongst which are: fever, infection, anaemia, thyrotoxicosis, pain or heart failure. This patient's presentation is that of heart failure with dyspnoea, raised JVP, chest crackles. Certainly, a rapid tachycardia can push one into heart failure. What is more likely is that heart failure has pushed this patient into a rapid ventricular response to AF or even contributed to the triggering of AF. His management in the first instance is of the driving factors of the fast ventricular response. Hence, a diuretic (answer 2h) is the most appropriate “antiarrhythmic”.

Management of AF

Direct attempts to deal with the AF or the rate by using cardioversion, amiodarone, verapamil, adenosine, atenolol, are not only inappropriate at this stage but may be dangerous. There is no indication, unless haemodynamically unstable, for immediate cardioversion of AF to sinus rhythm whether electrically or pharmacologically with amiodarone. The heart failure must be treated first and will almost certainly slow the ventricular rate. Unless the catecholamine stimulation is removed by treating the heart failure with diuretics, cardioversion is likely to fail with early recurrence of atrial fibrillation. Dealing with the sympathetic stimulation directly with atenolol in a patient with congested lungs and “wet” heart failure is contra-indicated.

Later on, if after the acute congestive heart failure is treated the heart rate is still inappropriately fast, a beta-blocker may be very useful. In addition, verapamil with its negative inotropic properties would be ill advised in a patient with heart failure. Digoxin whose action is neurocardiogenic via the parasympathetic nervous system is unlikely to have any acute beneficial benefits because what is necessary is not only parasympathetic stimulation but also a sympathetic withdrawal which digoxin does not provide. Therefore, it is no longer used in this scenario.

Stroke risk and prevention

A more important reason for not cardioverting the patient with AF acutely is the risk of systemic thromboembolism, particularly stroke. The risk applies to both electrical and pharmacological cardioversion. It is generally considered safe to do this if the duration of

the episode of atrial fibrillation is known to be less than 24 hours. This patient had symptoms for four days.

The next consideration is whether anticoagulation is required. The first question in this regard that one needs to ask in a patient presenting with AF: is there structural heart/valvular disease? There may be clues: a history of rheumatic heart disease, or abnormal clinical findings such as of mitral stenosis. Structural heart disease is best assessed with an echocardiogram. If the answer is yes, then anticoagulation is indicated (obviously taking into account and counterbalanced by risk of bleeding.)

In AF associated with non-valvular heart disease, the next question is: is the risk of stroke or thromboembolism high enough to justify use of an anticoagulant? Probably, a better question would be: is the risk sufficiently low to make anticoagulation unnecessary? Various risk scoring systems have been devised. The CHADS₂ and updated CHA₂DS₂-VASc scoring system has been validated and is favoured by the European Society of Cardiology in their AF management

TABLE 1 AND 2: The CHADS₂ and updated CHA₂DS₂-VASc scoring systems in non-valvular atrial fibrillation

Note that the subscript 2 signifies that that factor's point is doubled. A total score of 2 or more recommends chronic oral anticoagulation (OAC) (warfarin or dabigatran). A score of 1 was previously taken as an indicator of aspirin; however, the new guidelines, state that OAC is preferable to aspirin. A score of 0 requires no antithrombotic treatment; this is considered preferable to aspirin. The patient must be rescored regularly as age and other co-morbidities may alter the total and the recommendation for antithrombotic treatment. (Ref: www.escardio.org/guidelines)

CHADS ₂ score	
Cardiac Failure	Score: ■ 0 = nil (preferred) or aspirin ■ 1 = oral anticoagulant (preferred) or aspirin ■ 2 = warfarin
Hypertension	
Age	
Diabetes	
Stroke x 2	

CHA ₂ DS ₂ -VASc score
Cardiac Failure
Hypertension
Age ≥75yrs (x 2)
Diabetes
Stroke x 2
Vascular disease
Age 65-74yrs
Sex category / female

guidelines (see Tables 1 and 2) (www.escardio.org/guidelines). This patient would score: 1 for congestive heart failure, 1 for hypertension, 1 for diabetes, 1 for age >65yrs = 4. A score of 1 or higher warrants anticoagulation with warfarin or one of the new non-Vitamin K anticoagulants, such as dabigatran. (Answer 2m) Aspirin is inadequate. If this patient had been in atrial flutter, risk of thromboembolism is similar to AF and the scoring system should also be applied.

Longer term management

All of the ablation procedures mentioned in the question would be inappropriate in the acute setting or immediately after the first presentation. In the longer term, if symptoms of palpitations persist or if the ventricular rate is uncontrolled, ablation may be considered. If the patient had been in atrial flutter, ablation with creation of a line of block along the isthmus between the inferior vena cava and tricuspid annulus, the CTI, could be curative.

Answers: 1b; 2h; 2m.

CONCLUSIONS / LESSONS

- In an irregular SVT, atrial fibrillation (AF) is the default diagnosis.
- Atrial flutter, which may be associated with a regular or irregular ventricular rhythm, is characterised by very stable atrial activity, usually 250-350bpm. If not obscured by QRS complexes, irregular atrial electrical activity on ECG excludes atrial flutter.
- Fast ventricular response in AF behoves one to search for causes of sympathetic stimulation.
- The risk of stroke and systemic thromboembolism in AF that is not associated with valvular or structural heart disease is best assessed using the CHADS₂ score (or its update).