

Chapter 29 | Can the ECG guide treatment of narrow QRS tachycardia?

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Case presentations

Case 1: A 36-year-old female presents to the emergency department (ED) with palpitations and dizziness. She has no past medical history. She is diaphoretic and pale and her vital signs show a blood pressure of 100/60, pulse of 160, respirations of 20/minute, and oxygen saturation of 98% on room air. Her examination otherwise shows clear lung fields, and a normal mental status. The electrocardiogram (ECG) shows a narrow complex, regular tachycardia (Figure 29.1).

Case 2: A 28-year-old male presents to the ED with dizziness on a Monday morning. He has no past medical history, but does admit to drinking significant amounts of alcohol over the weekend. He has no chest pain, but notes near syncope with sudden onset of the dizziness at 8:30 am this morning. Vital signs are temperature of 37 °C (98.6 °F), blood pressure of 110/70, pulse of 140, respirations 20/minute, with oxygen saturation of 99% on room air. Physical examination shows an irregularly irregular pulse, no cardiac murmur or gallop, clear lung fields, no thyromegaly, and normal peripheral pulses. His ECG (Figure 29.2) shows atrial fibrillation with a rapid ventricular response.

Case 3: A 78-year-old female presents to the ED with dizziness and respiratory distress. The patient began feeling poorly earlier today, and her breathing worsened while she was visiting with her family. She has no chest pain, and has a long history of chronic obstructive pulmonary disease (COPD). Vital signs show a normal temperature, blood pressure of 130/70, pulse of 120, respirations 28/minute and oxygen saturation of 87% on oxygen. Examination reveals an anxious patient who is somewhat pale. Lungs show diffuse wheezes, and peripheral pulses are present but weak. The patient has a history of similar episodes, which have

required intubation in the past. Her ECG (Figure 29.3) shows an irregular narrow QRS tachycardia.

The ECG in narrow QRS tachycardia: management considerations

Approximately 50,000 ED visits per year are attributed to supraventricular tachycardia (SVT) [1]. This is a fairly common tachydysrhythmia and represents only one variation of narrow QRS tachycardias. The 12-lead ECG is an important tool in managing the adult patient presenting to the ED with tachycardia. One of the critical steps in the management of the tachycardic patient is determining whether the QRS complex is wide or narrow. Once the QRS complex has been deemed to be narrow with a regular pattern, the differential diagnosis list is limited to a manageable number of possibilities, including sinus tachycardias, atrioventricular nodal re-entrant tachycardias (AVNRT), focal and non-paroxysmal junctional tachycardias, atrioventricular re-entrant tachycardias (AVRT) with extranodal accessory pathways, focal atrial tachycardias, and atrial flutter [2]. The irregular narrow QRS tachycardias include atrial fibrillation, atrial flutter with variable conduction, and multifocal atrial tachycardia (MAT).

Patients with acute tachydysrhythmias represent typical urgent and emergent presentations. Management of these patients usually involves time-critical decisions requiring simultaneous evaluation and management to ensure the best possible patient outcome. The following chapter will discuss some of these tachycardias and how the ECG can be used to guide management decisions.

Narrow QRS tachycardias – regular

Electrocardiogram diagnosis is the key to treating narrow QRS complex tachycardias. While the focus here is on management, some discussion of the ECG diagnosis process is

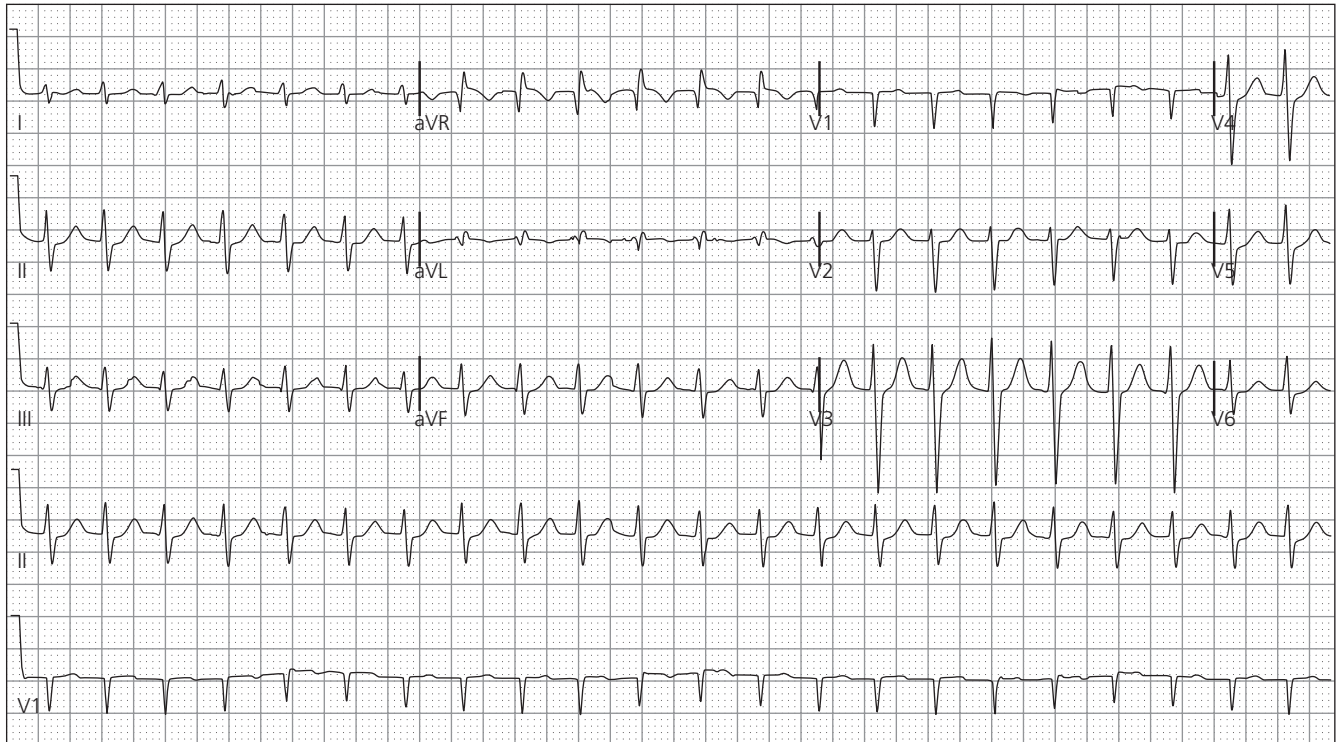


Figure 29.1 AVNRT. The rhythm has a typical regular, narrow complex pattern.

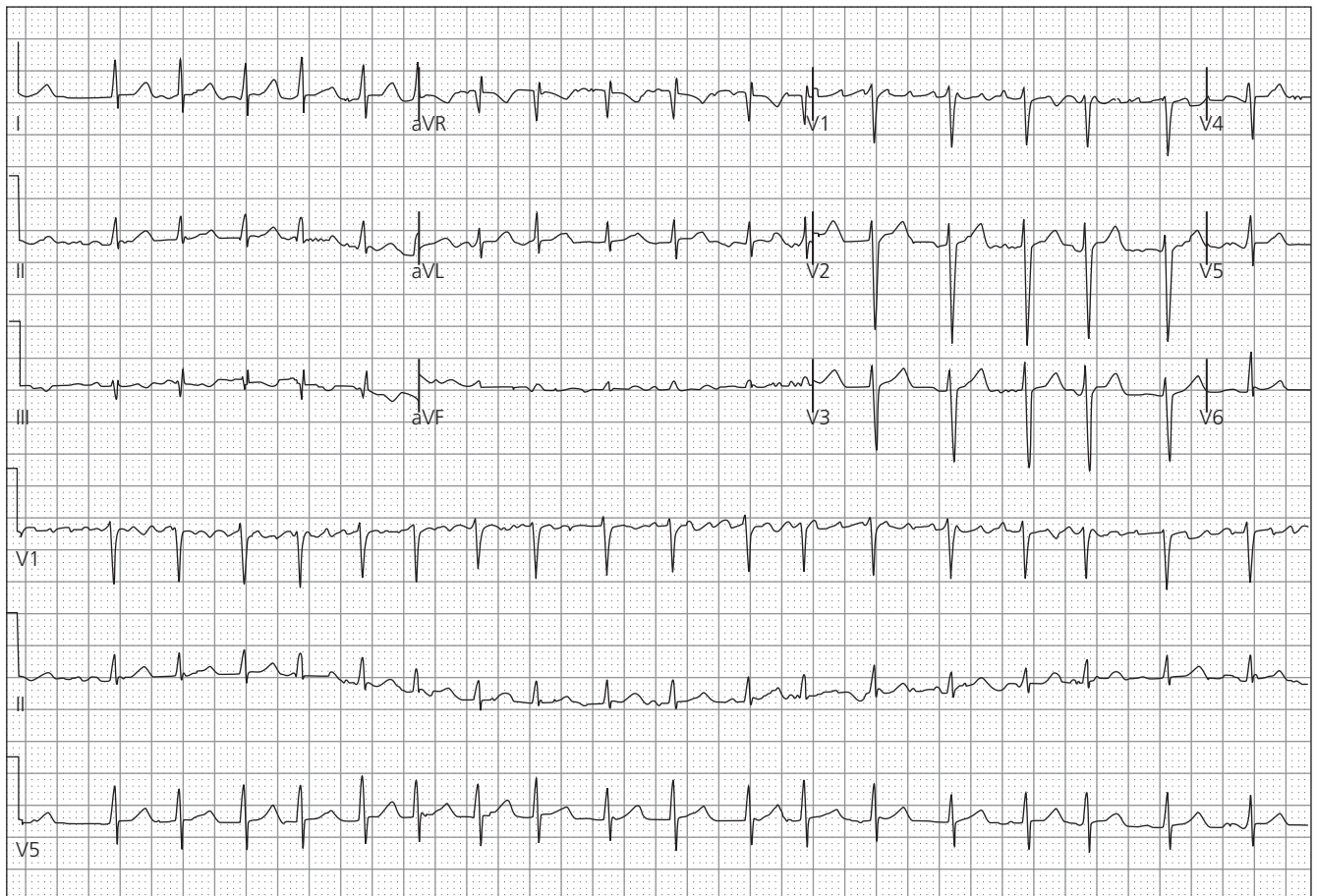


Figure 29.2 Atrial fibrillation. The rhythm is irregularly irregular with disorganized atrial activity.

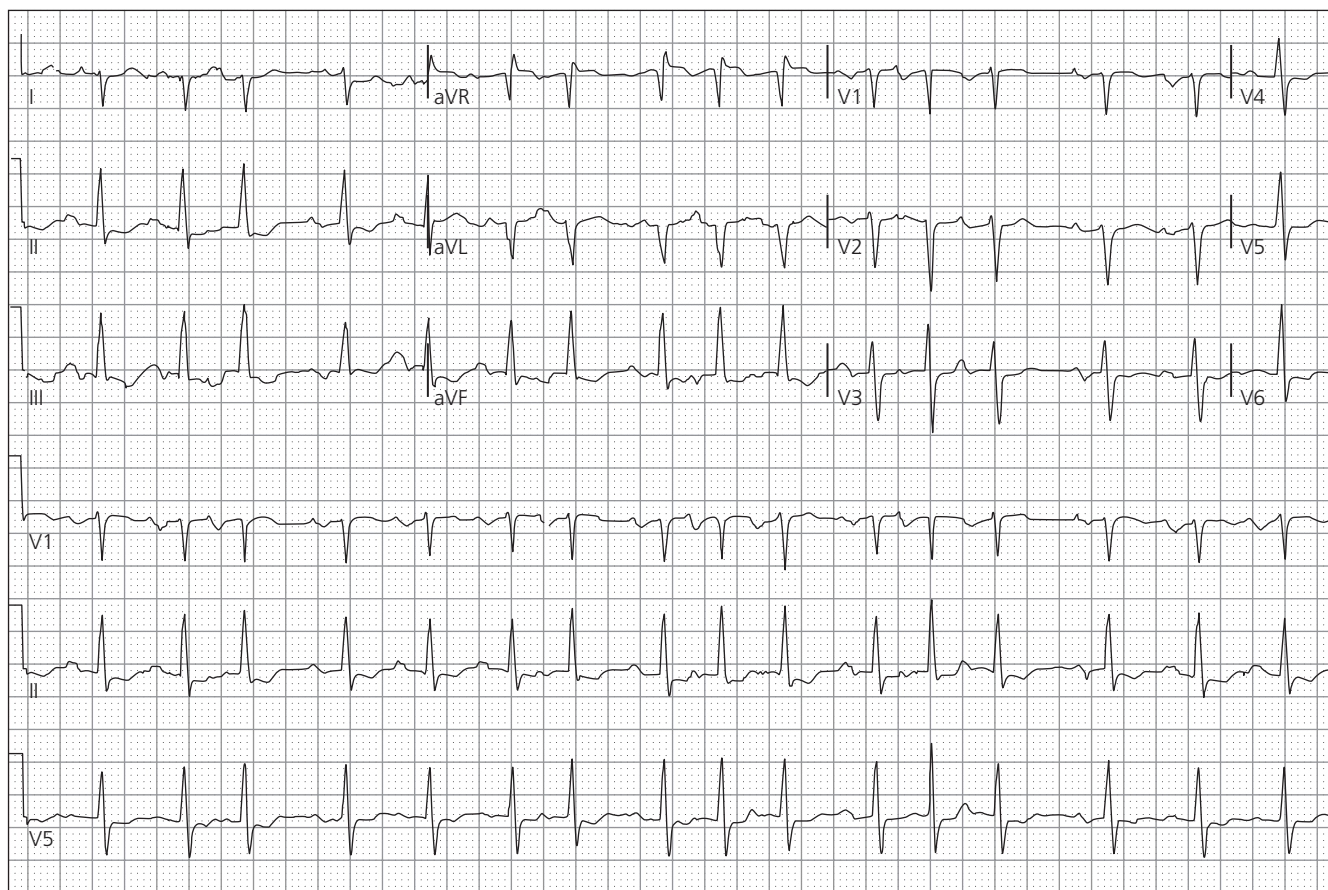


Figure 29.3 MAT. The atrial and ventricular complex occur irregularly, and the atrial complexes demonstrate \geq three morphologies consistent with origin from various sites within the atria.

needed. Several studies have looked at the use of the surface ECG in differentiating the mechanisms of narrow QRS complex tachycardias. In one study, multiple criteria were developed for various tachycardia types. Presence of a P wave separate from the QRS complex was seen frequently with atrioventricular (AV) reciprocating tachycardia, as was QRS alternans. In lead V1, a pseudo-R' pattern and pseudo-S wave in the inferior leads were seen commonly with AVNRT. Presence of P waves separate from the QRS, as well as RP/PR interval greater or equal to 1, were also associated with atrial tachycardias [3]. Similar findings regarding visible P waves with narrow QRS tachycardia were seen in another, more recent study, where an R' pattern in V1 or S wave in lead II were associated with AVNRT. This study also suggests the need for consideration of AVRT in elderly and female patients with narrow QRS tachycardia and visible P waves, when pre-excitation is not noticed [4]. Several studies also focus on the more specialized, invasive diagnosis and management of narrow QRS complex tachycardia in the electrophysiology lab. [5,6]. Simultaneous diagnostic testing and management can be accomplished via use of the Valsalva maneuver or carotid sinus massage in appropriate patients [7,8]. Another technique that may aid in visualizing P waves

is to increase the speed of the ECG recording (see Figure 29.4) [9]. Despite the criteria above, the diagnosis and treatment of narrow QRS complex tachycardias remains difficult.

Initial medical management of AVNRT (Figure 29.1) usually involves vagal maneuvers, followed by intravenous (IV) adenosine if vagal maneuvers are unsuccessful. Adenosine is rapid-acting, has a very short duration of action, and a good safety profile, including use in prehospital care [10,11]. Treatment with adenosine has also been compared with verapamil and similar efficacy rates have been noted [12,13]. Additional treatment options include other calcium channel-blockers and beta-blockers, and “pill in the pocket” regimens have been proposed. Treatment involves prescribing an oral medication to be taken on an as-needed basis. The combination of oral diltiazem and propranolol seems to be effective for this purpose [14].

Atrioventricular reciprocating tachycardias involve the presence of an accessory or bypass tract around the AV node. Wolff-Parkinson-White (WPW) syndrome most commonly provides the substrate for this dysrhythmia. In sinus rhythm, widening of the QRS complex is seen along with shortened PR interval; additionally, the QRS complex is initially slurred, known as the delta wave. Wolff-Parkinson-White syndrome,

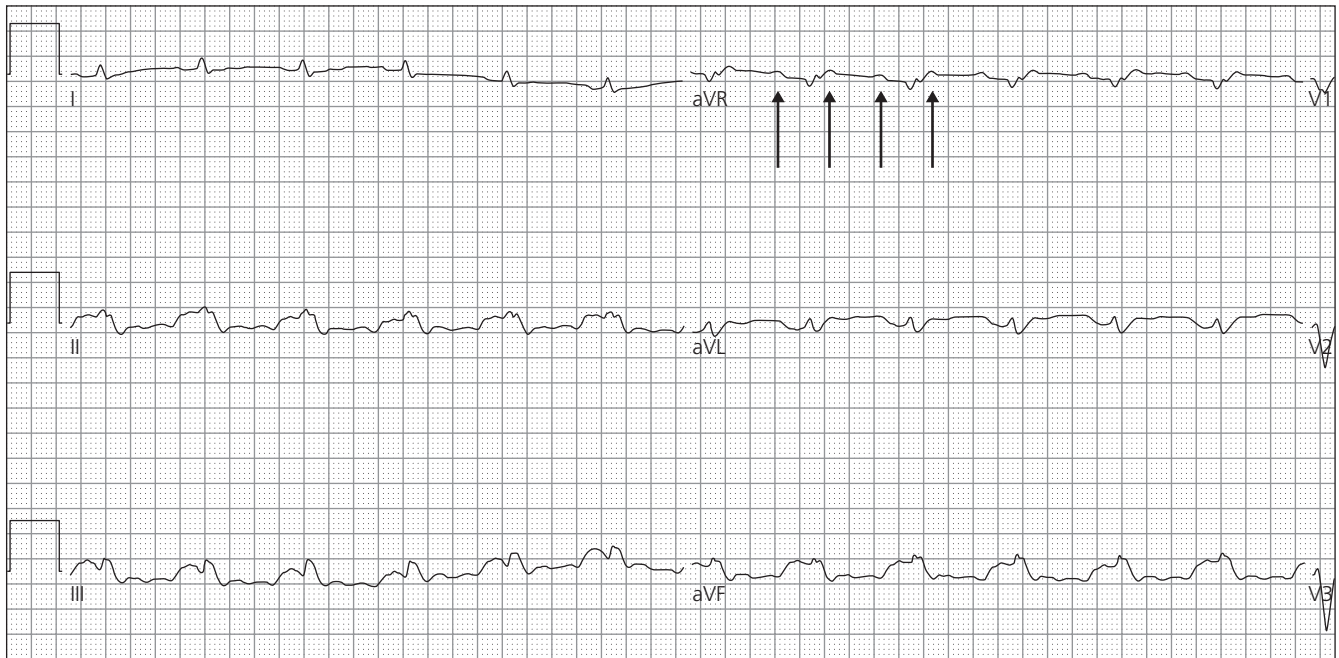


Figure 29.4 ECG recorded as 50 mm/s. Normally ECGs are recorded at 25 mm/s. In this case, the ECG computer setting was changed to “stretch out” the tracing. This allows easier distinction of the P waves (arrows). Note that because the tracing occurs at half-speed, the atrial and ventricular rates are halved as well. (ECG courtesy of Amal Mattu, MD.)

as well as other forms of AVRT, can also involve a “concealed” accessory pathway, with orthodromic conduction leading to a narrow QRS tachycardia. Because the tachycardia may manifest with a narrow QRS complex, it is important to evaluate the baseline ECG, after return to sinus rhythm for evidence of pre-excitation. ST segment elevation in lead aVR, seen with AVRT, may also help differentiate this mechanism from other types of SVT [15]. Patients with wide QRS complex AVRT due to WPW syndrome typically respond to medical treatment with procainamide or synchronized cardioversion. Long-term treatment most often involves ablation of the accessory pathway.

Electrocardiogram diagnosis of sinus tachycardia is based on verification of a sinus mechanism. P wave axis is helpful in this regard. Positive (upright) P waves in the inferior leads, with negative P waves in aVR define the ECG criteria for sinus rhythm [2]. Integrating the clinical findings is also key for sinus tachycardia. The tachycardia typically begins and ends gradually (non-paroxysmal), as opposed to the sudden onset associated with many AVRT or AVRNT. There is also some evidence that the amplitude of the QRS complex in leads V2–V5 may increase with SVT, but not with sinus tachycardia. This may be useful in differentiating these rhythms at faster rates [16]. Thus, using the ECG for patients with sinus tachycardia can guide therapy toward the underlying condition (e.g. pain, fever, hypovolemia, anxiety, etc.) [2]. Specific medications for sinus tachycardia are sometimes indicated – e.g. beta-blockers as part of the treatment regimen for thyrotoxicosis – but treatment, in general, should be focused on correcting the underlying cause.

Focal atrial tachycardias can usually be differentiated from sinus tachycardias by evaluating the P wave axis. Inverted P waves in the inferior leads (II, III, aVF) indicate an abnormal site of origin of the rhythm. A regular, narrow QRS complex tachycardia with an abnormal P wave axis should lead to consideration of an atrial tachycardia. Management of automatic atrial tachycardias is difficult as medical therapies and electrical cardioversion are often ineffective. Attempts to control ventricular rate usually involve either beta-blockers or calcium channel-blockers, often with limited success. Direct suppression of the atrial focus can be attempted by using class Ia or Ic agents, or with class III agents such as sotalol or amiodarone [2].

The ECG criteria for atrial flutter (see Figure 29.5) include organized regular atrial activity and atrial impulses at a rate of 250–350. Typically, atrial flutter is due to one of a variety of re-entry loops and manifests with 2 : 1 ventricular conduction. The organized atrial activity (saw-tooth waves) seen on ECG affects treatment planning. Class Ic agents may slow the flutter waves, but do not typically lead to conversion to sinus rhythm. Class III agents such as amiodarone tend to be more effective in terminating atrial flutter. Electrical cardioversion, often with relatively low energy levels, is very effective for atrial flutter as well [2].

Focal junctional tachycardias have ventricular rates of 100–250 and frequently show ECG evidence of AV dissociation. These rhythms are sometimes known as automatic or paroxysmal junctional tachycardias and there is a paucity of information in the medical literature regarding their management. Stress or exercise can be precipitants, and potential

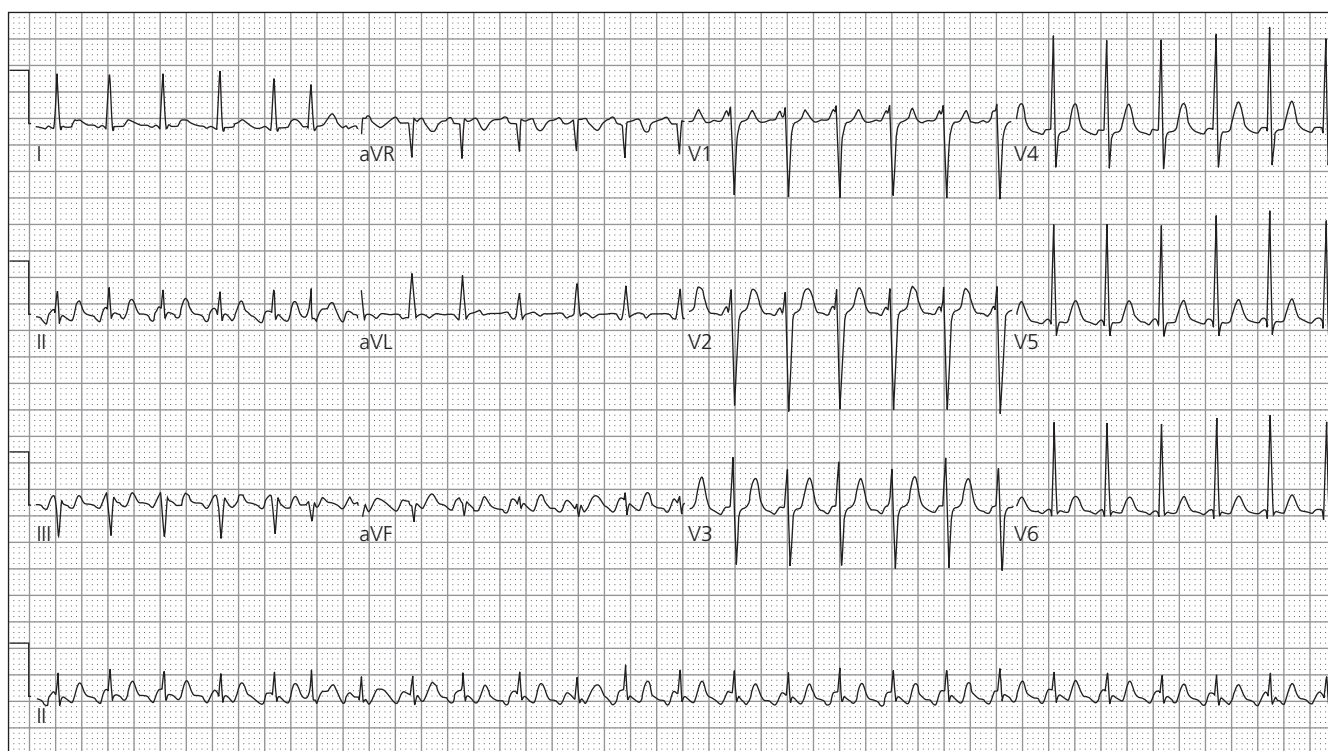


Figure 29.5 Atrial flutter with 2 : 1 AV conduction. The rhythm is regular with a ventricular rate of approximately 150 bpm, typical of this rhythm. Atrial activity manifests as the classic “saw-tooth” pattern with inverted flutter waves (“F waves”) in the inferior leads with a rate of 300 bpm. (ECG courtesy of Amal Mattu, MD.)

treatments include beta-blockers and IV flecainide. Ablation via catheter can result in a cure but is associated with a risk of AV block [2].

Non-paroxysmal junctional rhythms typically manifest with a slower ventricular rate (70–120 bpm) than the focal junctional tachycardias, and usually exhibit one-to-one AV association. A “warm up” and “cool down” pattern is seen at the beginning and end of the period of dysrhythmia, and this rhythm can sometimes be a marker for serious underlying problems such as digoxin toxicity, cardiac ischemia, or hypokalemia. Treatment, similar to MAT, centers on correcting the underlying medical problems, especially digoxin toxicity. Digitalis-binding antibodies may be required in serious cases [2]. For refractory cases, calcium channel-blockers or beta-blockers may be used [17].

Narrow QRS tachycardias – irregular

The differential diagnosis of the irregular narrow QRS complex tachycardia is somewhat simpler. Atrial fibrillation comprises the vast majority of irregular tachycardias (Figure 29.2). Atrial fibrillation is usually fairly evident from the surface ECG, although rapid ventricular rates can make this diagnosis somewhat more difficult. The presence of an

irregularly irregular rhythm with no evidence of organized atrial activity confirms the diagnosis of atrial fibrillation. Once this ECG diagnosis is made, treatment decisions are based on the desired end-result. Short-term management often includes ventricular rate control, accomplished by using calcium channel-blockers such as diltiazem or beta-blockers, such as esmolol or metoprolol [18,19]. Additional considerations include attempts at electrical cardioversion, pharmacologic cardioversion using amiodarone, flecainide, quinidine, ibutilide, or propafenone, as well as initiation of anticoagulation in cases of certain atrial fibrillation [18].

Multifocal atrial tachycardia is often secondary to cardiorespiratory issues, electrolyte abnormalities, or medication toxicity [2]. Electrocardiogram diagnosis hinges on identification of three or more distinct P wave morphologies, in conjunction with the appropriate clinical situation (Figure 29.3). Differentiating MAT from the more commonly seen atrial fibrillation is dependent on identification of distinct P waves and correlating this with the clinical scenario. Treatment should be aimed at the underlying condition, with specific medical management occasionally focusing on rate control with calcium channel-blockers, as cardioversion and other anti-arrhythmic medication therapy are not indicated [2]. Atrial flutter with variable conduction (see Figure 29.6) is a third, and relatively common, cause of irregular QRS tachycardia. In this rhythm, the AV conduction usually varies

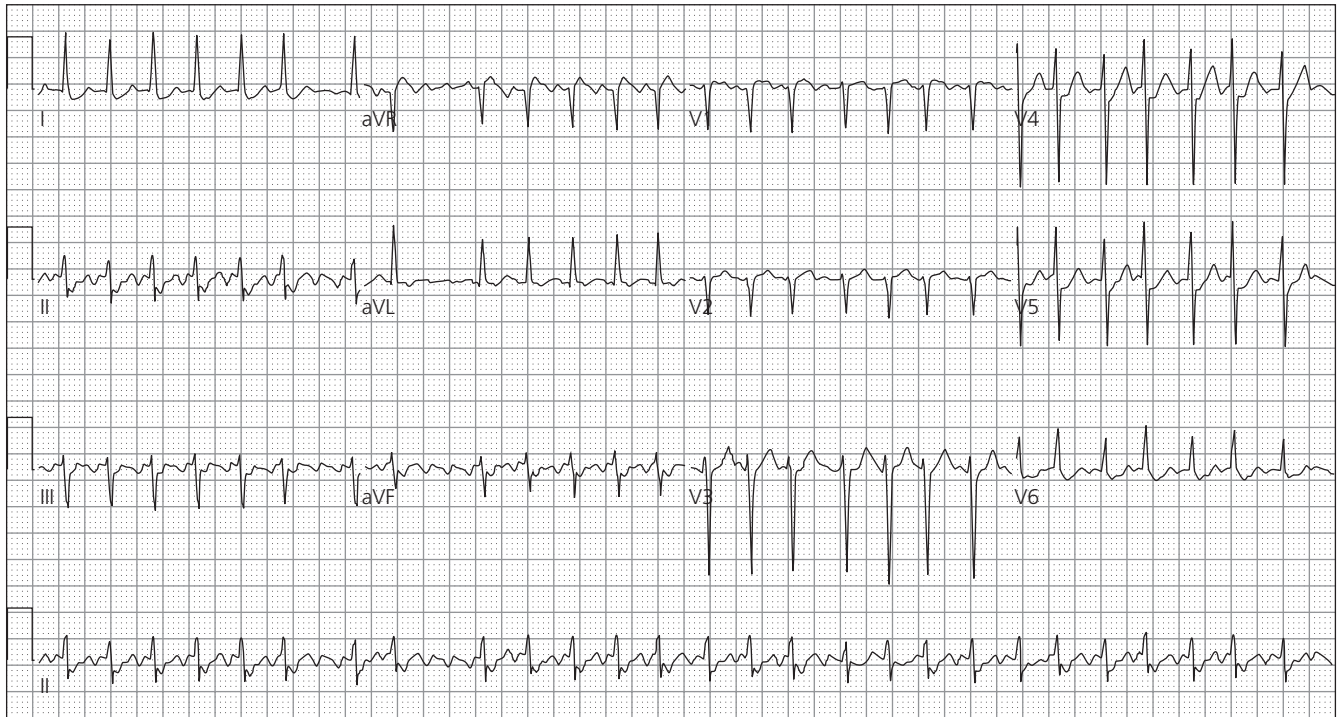


Figure 29.6 Atrial flutter with variable AV conduction. F waves are noted in the inferior leads at a rate of approximately 300 bpm, diagnostic of atrial flutter. The ventricular response is irregular because the AV conduction varies between 2 : 1, 3 : 1, and even 4 : 1. Note that in some portions of the rhythm when the conduction ratio is consecutively 2 : 1, the ventricular response is *regular*. Atrial fibrillation, on the other hand, would remain irregular throughout. (ECG courtesy of Amal Mattu, MD.)

between 2 : 1, 3 : 1, and sometimes 4 : 1 conduction producing an overall irregular rhythm. The irregularity often causes a misdiagnosis of atrial fibrillation. Treatment of this irregular atrial flutter is similar to treatment for regular atrial flutter.

Case conclusions

In Case 1, the ECG (Figure 29.1) demonstrates a regular, narrow QRS complex tachycardia; the rhythm diagnosis is AVNRT. The rhythm was compromising, yet the patient was considered hemodynamically stable. She received intravenous adenosine with prompt return to sinus rhythm. A repeat ECG was within normal limits. In Case 2, the ECG (Figure 29.2) demonstrates atrial fibrillation with an irregularly irregular rhythm and disorganized atrial activity without discernible P waves. The patient initially was managed with volume expansion with normal saline followed by rate control with intravenous diltiazem. His rate markedly slowed, yet he remained in the dysrhythmia. He was admitted to the hospital where he ultimately converted to sinus rhythm; subsequent evaluation was unremarkable. Lastly, Case 3 (Figure 29.3) illustrates MAT; the patient was experiencing an exacerbation of COPD coupled with volume depletion and hypokalemia. The underlying conditions were managed with ultimate conversion to sinus tachycardia.

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