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Selected Topics: Cardiology Commentary

ELECTROCARDIOGRAPHIC MANIFESTATIONS: NARROW QRS COMPLEX TACHYCARDIAS

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□ **Abstract—Narrow QRS complex tachycardia is a common dysrhythmia in Emergency Medicine practice. Diagnosis and mechanism often can be made by 12-lead electrocardiographic (EKG) analysis but may subsequently require electrophysiologic testing. The clinical manifestations are varied and dependent upon heart rate, prior cardiac disease, and general physiologic status. Patient management is directed towards the etiology and mechanism of the dysrhythmia and includes vagal maneuvers, pharmacologic therapy, and cardioversion. Hemodynamically compromised patients must be promptly treated. Patients are often admitted to the hospital but selected patients can be safely discharged from the Emergency Department for outpatient evaluation and management. Pediatric and pregnant patients are, in general, treated the same as adults. Several case examples and EKGs are presented.** **© 2003 Elsevier Science Inc.**

□ **Keywords—supraventricular tachycardia (SVT); narrow complex tachycardia; electrocardiogram; EKG; dysrhythmia**

INTRODUCTION

Narrow QRS complex tachycardia (NCT) is one of the most common abnormal cardiac rhythms seen in the Emergency Department (ED) (1). It is defined as a tachycardia with a QRS less than 0.12 s duration where conduction begins proximal to the bundle of His (2). This rhythm, by definition, is a supraventricular tachycardia (SVT), although confusion can occur when ventricular tachycardia presents with a narrow QRS (3). Alternatively, some SVTs present with a wide QRS complex because of aberrant conduction (4). This article will focus on the narrow complex rhythms. The term paroxysmal atrial tachycardia, or PAT, is obsolete and should not be used. The approach to the ED patient with NCT depends on hemodynamic stability, the electrophysiologic mechanism, and site of origin of the dysrhythmia (5). Other factors such as medications and pre-existing cardiac disease may influence diagnosis and treatment (6). The hemodynamically unstable patient with a tachydysrhythmia should be immediately cardioverted. All other patients should undergo appropriate evaluation, including a 12-lead electrocardiogram (EKG). A rhythm strip alone can be misleading, especially when evaluating QRS duration, P-wave morphology, and ST-T-wave abnormalities.

NCTs can produce a variety of signs and symptoms, ranging from asymptomatic to cardiovascular collapse. Frequently, patients will complain of a rapid or irregular heart beat. Dizziness or syncope is correlated to the heart rate and pre-existing cardiac function. The following cases are examples of NCT.

CASE PRESENTATIONS

Case 1

A 22-year-old man presented to the ED with the sensation of rapid heartbeat that began suddenly 1 h prior to

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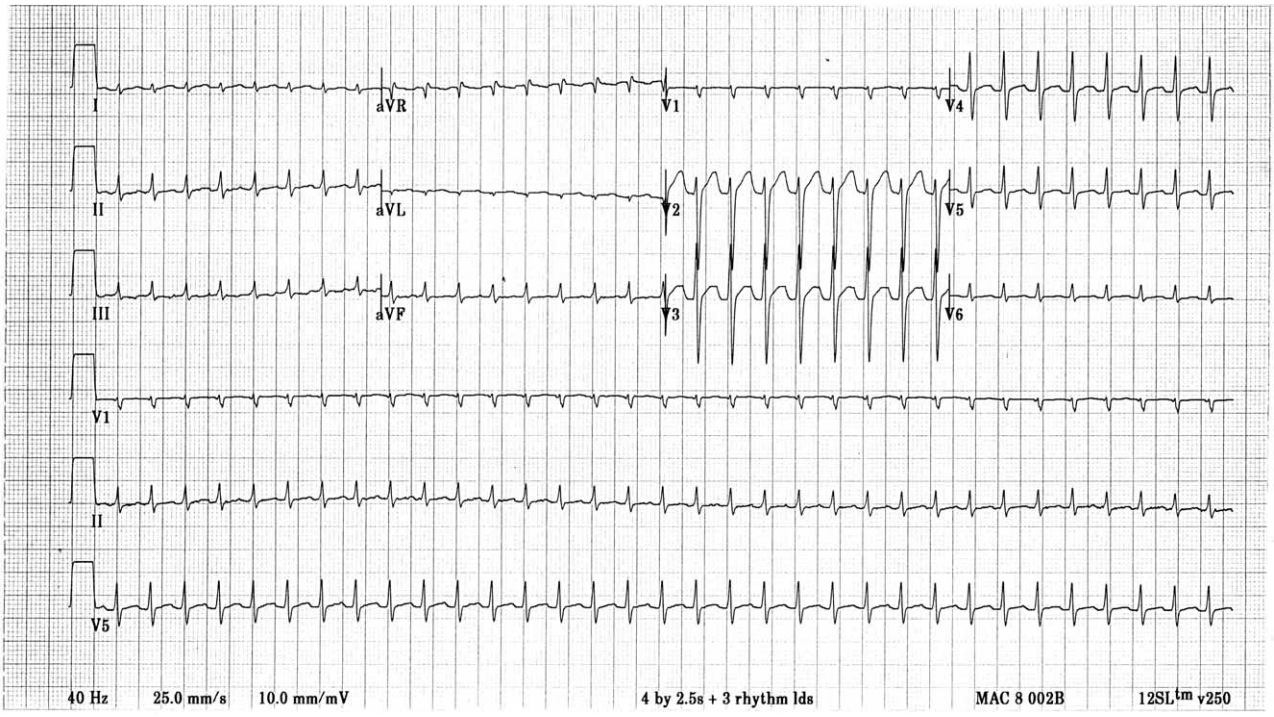

Figure 1. Regular, supraventricular tachycardia at 200 beats/min. No discernable P waves. T wave flattening in leads II, III, aVf.

arrival. He had these symptoms numerous times before this visit, often requiring ED visits for treatment. He denied using any medications, caffeine use, illicit drug use, or use of herbal preparations. The examination was remarkable for a regular pulse of about 200 beats/min, blood pressure of 125/85 mm Hg, no murmur, rubs or other abnormalities. The 12-lead EKG (Figure 1) shows a regular supraventricular tachycardia at around 200 beats/min, normal axis, no discernable P waves, and some T-wave flattening in the inferior leads. The rhythm

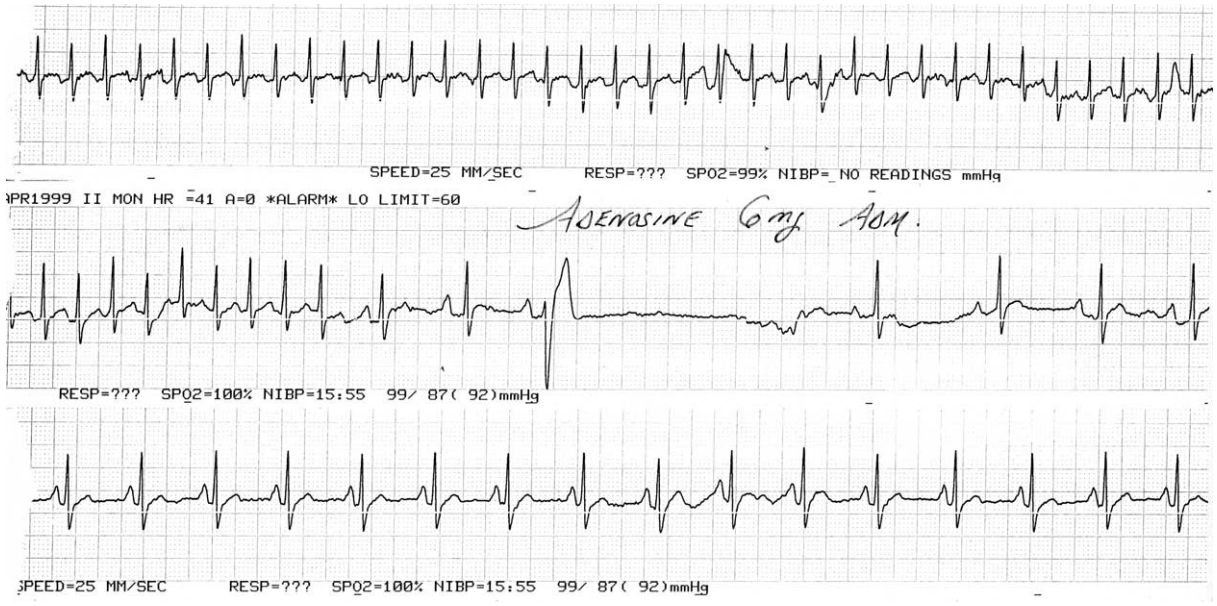

Figure 2. Rhythm strip. Normal sinus rhythm. Note the several second pause after the aberrantly conducted beat in the center panel.

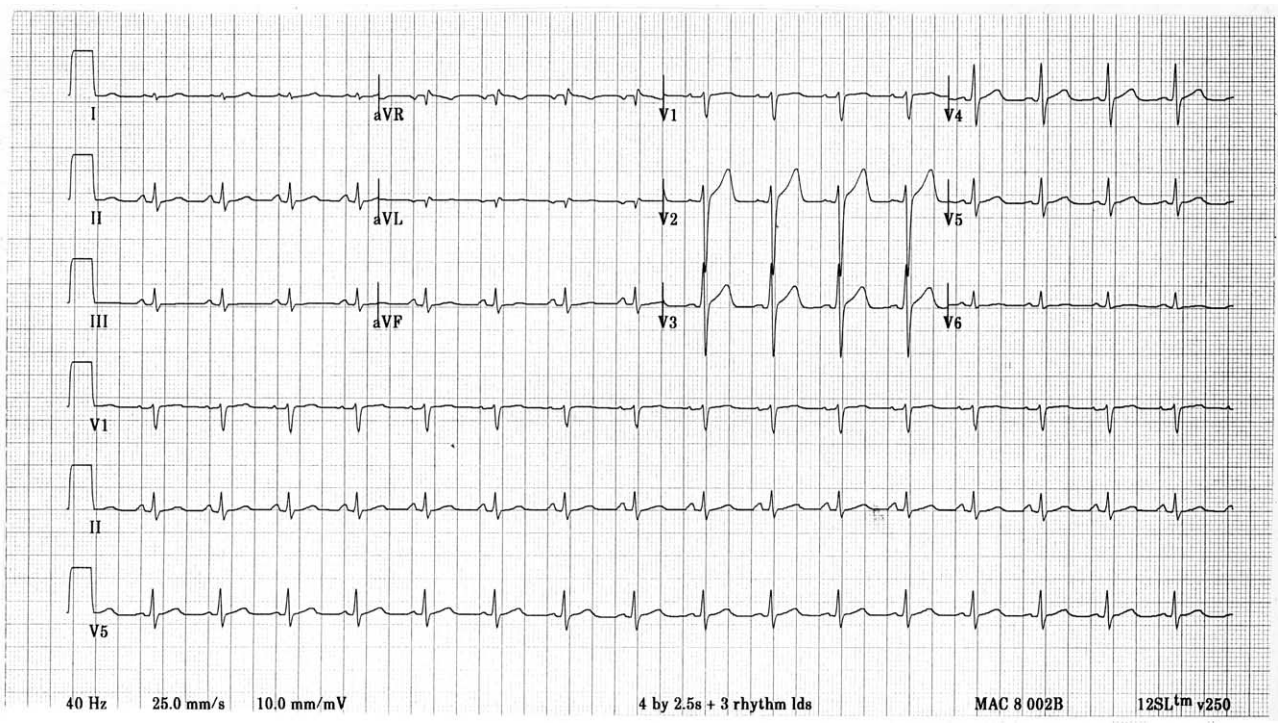

Figure 3. Normal sinus rhythm. No delta wave noted.

is most likely an AV nodal re-entry tachycardia. The patient was given 6 mg of adenosine i.v. with conversion to sinus rhythm (Figure 2). Repeat EKG (Figure 3) shows sinus rhythm without evidence of an accessory pathway. The patient was referred to an electrophysiologist but refused an electrophysiolic study (EPS) and possible ablation.

Case 2

An 85-year-old woman presented to the ED after a fall and shortness of breath. The patient denied chest pain, palpitations, or loss of consciousness. The patient had a history of severe chronic obstructive pulmonary disease (COPD) and was taking theophylline, digoxin, furosemide, albuterol and ipratropium inhalers and alprazolam. The patient denied use of over-the-counter medications or herbal preparations and drank one cup of coffee per day. There was no tobacco use for 10 years but over 100 pack-years of use. Examination revealed a thin elderly woman in moderate respiratory distress. Vital signs were normothermia, the pulse was irregularly irregular with a rate of about 110 beats/min, blood pressure (BP) was 174/92 mm Hg, and the respiratory rate was 30 breaths/min. Pulse oximeter revealed an 85% oxygen saturation on room air. There were no murmurs or gallops. The lungs had decreased air movement and a prolonged expiratory period but no wheezes. The 12-lead EKG (Figure 4) shows multifocal atrial tachycardia (MAT) with three different P-wave morphologies and a rate of about 106 beats/min, left axis deviation, and minimal ST segment depression in leads V5, V6 and AVL. Theophylline level was 7.1 ug/mL, digoxin was 1.0 ng/mL and potassium was 3.5 mmol/L. The patient was treated for her respiratory compromise with bronchodilatators, steroids and oxygen to maintain saturations at around 90%. No specific treatment for the MAT was required.

Case 3

A 64-year-old man came to the ED because of irregular pulse. He had noted this abnormality for about 3 days. He denied chest pain, shortness of breath or dizziness. He had a past history of hypertension. His medications were Aspirin, Lisinopril, Metoprolol and Citalopram. Physical examination revealed normothermia. The pulse was irregular and between 105 and 150 beats/min, blood pressure was 170/89 mm Hg and respirations were 18 breaths/min. The heart had an irregularly irregular rate with grade I/VI systolic murmur at the lower left sternal border. The lungs were clear. There was no peripheral edema. The EKG (Figure 5) shows atrial fibrillation with a ventricular rate of 110 to 150 beats/min and areas that

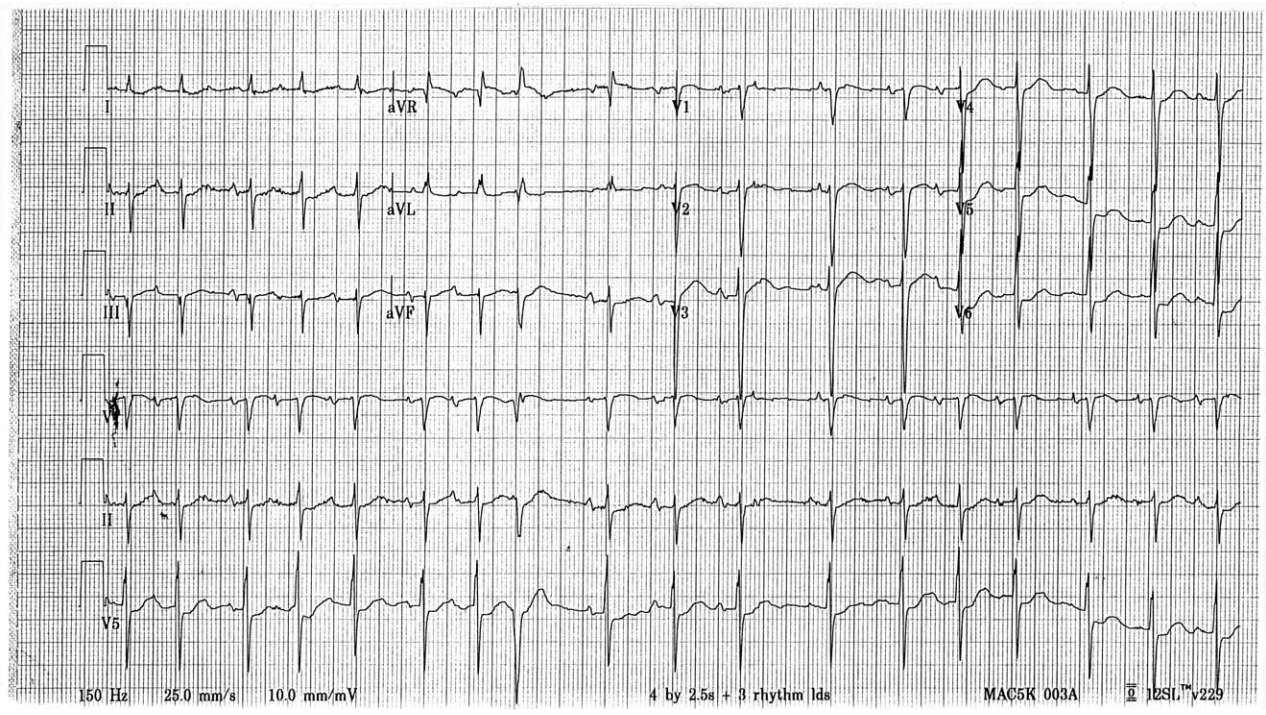

Figure 4. Multifocal atrial tachycardia. Three different P wave morphologies are present. ST segment depression in leads V5 and V6. Left axis deviation.

are regular at 150 beats/min compatible with atrial flutter, left anterior fasicular block, and normal complexes. The patient was anticoagulated, had a transesophageal echocardiogram that revealed no intracardiac blood clots, and was cardioconverted to sinus rhythm with 75 Joules. A repeat EKG (Figure 6) revealed normal sinus rhythm at 90 beats/min, normal complexes, left anterior fasicular block, and left atrial abnormality.

Case 4

A 43-year-old man came to the ED with a complaint of rapid heartbeat for 3 h duration. He denied chest pain, shortness of breath, dizziness, or other symptoms. The patient did not take medications, ingest caffeine products or use illicit drugs. On examination the pulse was regular at 200 beats/min and the blood pressure was 146/88 mm Hg. The cardiac examination revealed normal heart sounds, no murmurs or rubs. The 12-lead EKG (Figure 7) shows a regular, narrow complex tachycardia at around 200 beats/min, normal axis, no P waves, and ST-T-wave abnormalities diffusely. The patient was given 6 mg of adenosine i.v. This initially slowed the rate and revealed a P-wave rate of approximately 200/min (Figure 8) but then returned to the rapid ventricular rate. The patient then received 20 mg of diltiazem i.v. and changed to an atrial ectopic rhythm with 2:1 conduction and a ventricular rate of about 100 beats/min (Figure 9). The non-conducted P waves were readily observed in leads II and III in between the QRS and T wave. The P-wave morphology was similar to the P waves in Figure 8. After conscious sedation with Midazolam and Fentanyl, the patient was cardioverted with 100 Joules to a normal sinus rhythm (Figure 10). The patient had electrophysiologic mapping that revealed 2 atrial foci in the right atrium that were ablated.

DISCUSSION

The initial approach to the patient with NCT is to determine hemodynamic stability. The actual heart rate is not the determining factor of instability, but the ability of the heart to perfuse to the critical tissues. This might manifest as cardiac ischemia, heart failure, mental confusion, or hypotension. The unstable patient should be cardioverted with 100 or 200 Joules, synchronized. Vagal maneuvers can be attempted in the stable patient. Carotid sinus massage and Valsalva maneuver are successful in 20–80% of patients (7,8). However, there have been case reports of stroke following carotid sinus massage in the elderly (9).

A clinically useful classification system for NCT is based upon the anatomic origin of the dysrhythmia that allows formulation of a rational treatment strategy. The causes of NCT are varied and are listed in Table 1. One

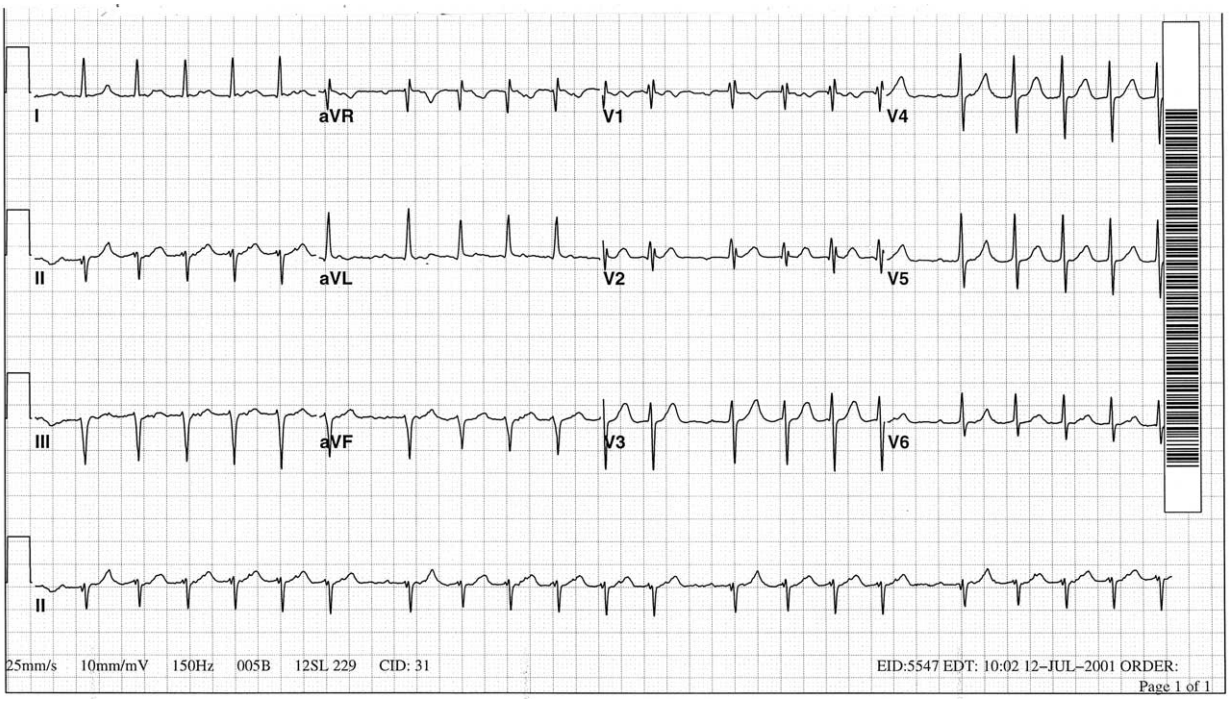

Figure 5. Atrial fibrillation with a ventricular rate of 110-150 beats/min. Left anterior fasicular block. Note the pseudo R' deflection in lead V1.

group of NCTs is independent of the atrioventricular (AV) node and is more a function of abnormal automaticity or triggered activity within the atria (10). Included in this group are sinus tachycardia and the atrial tachycardias. There are many causes of these dysrhythmias and treatment must be directed toward the etiology. The

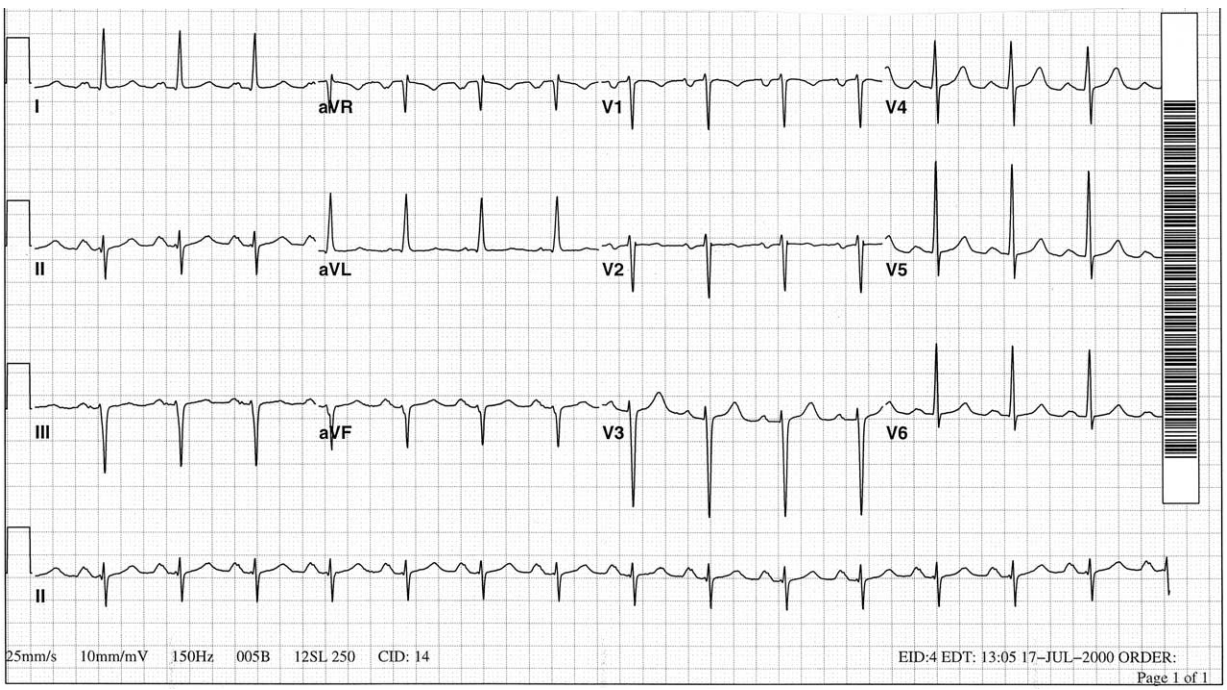

Figure 6. Normal sinus rhythm. Left anterior fasicular block. Left atrial abnormality.

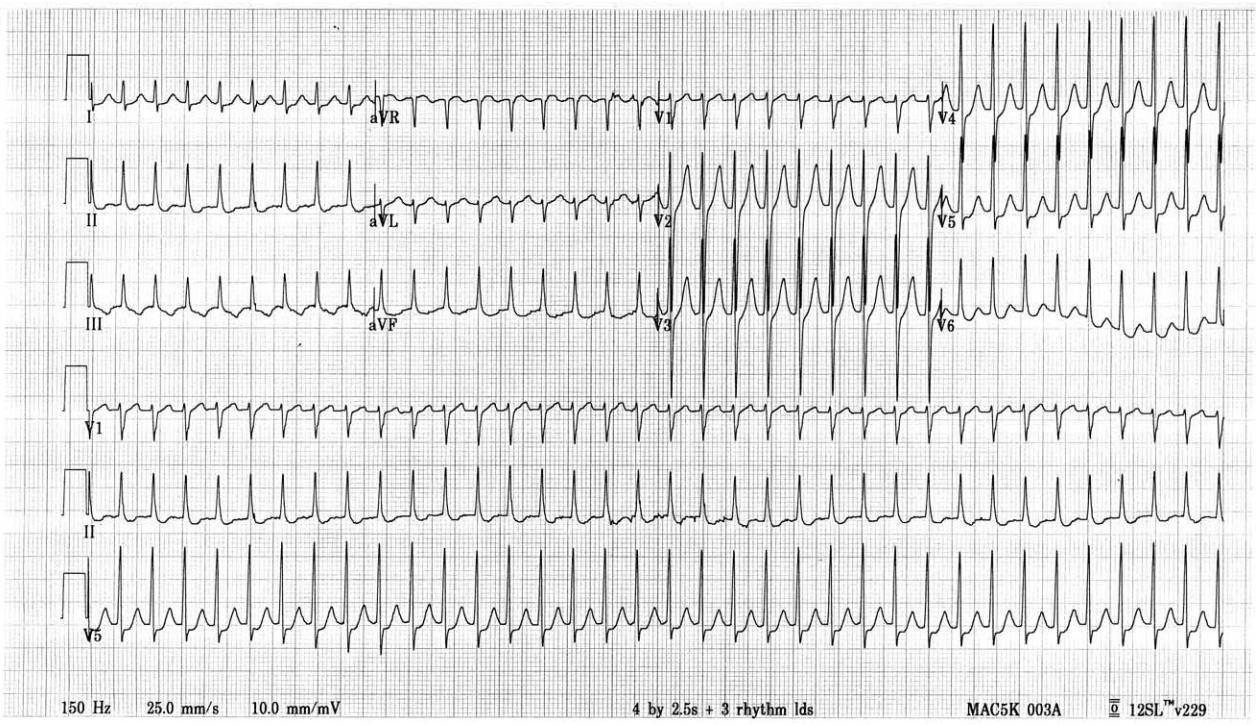

Figure 7. Regular narrow complex QRS tachycardia at 200 beats/min. Normal axis. No discernable P waves. Diffuse ST segment depression.

other large group of NCTs is classified as AV node-dependent. These include AV node reentry, AV reentrant and junctional tachycardias. The AV reentrant NCT may be associated with an accessory pathway. The AV nodal reentry, in which the reentrant circuit is confined to the AV node or the region around the AV node, is the most common type of NCT in adults (after excluding sinus tachycardia) (11).

The mechanism of an NCT often can be determined utilizing clinical and EKG clues, but definitive diagnosis and treatment often requires electrophysiologic testing. Case 4 illustrates how the mechanism can be determined

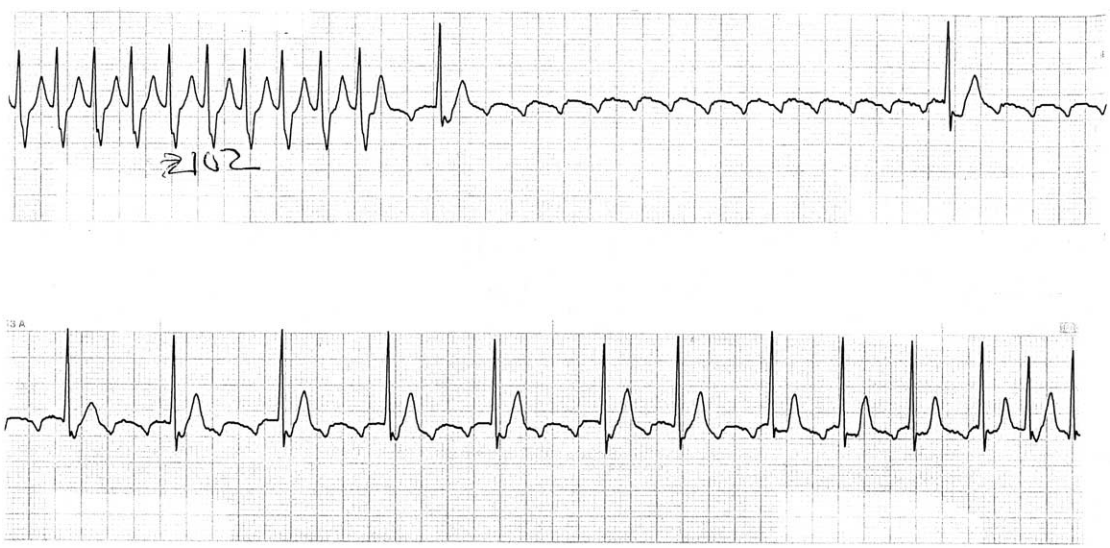

Figure 8. Rhythm strip. Atrial rate of approximately 200 beats/min.

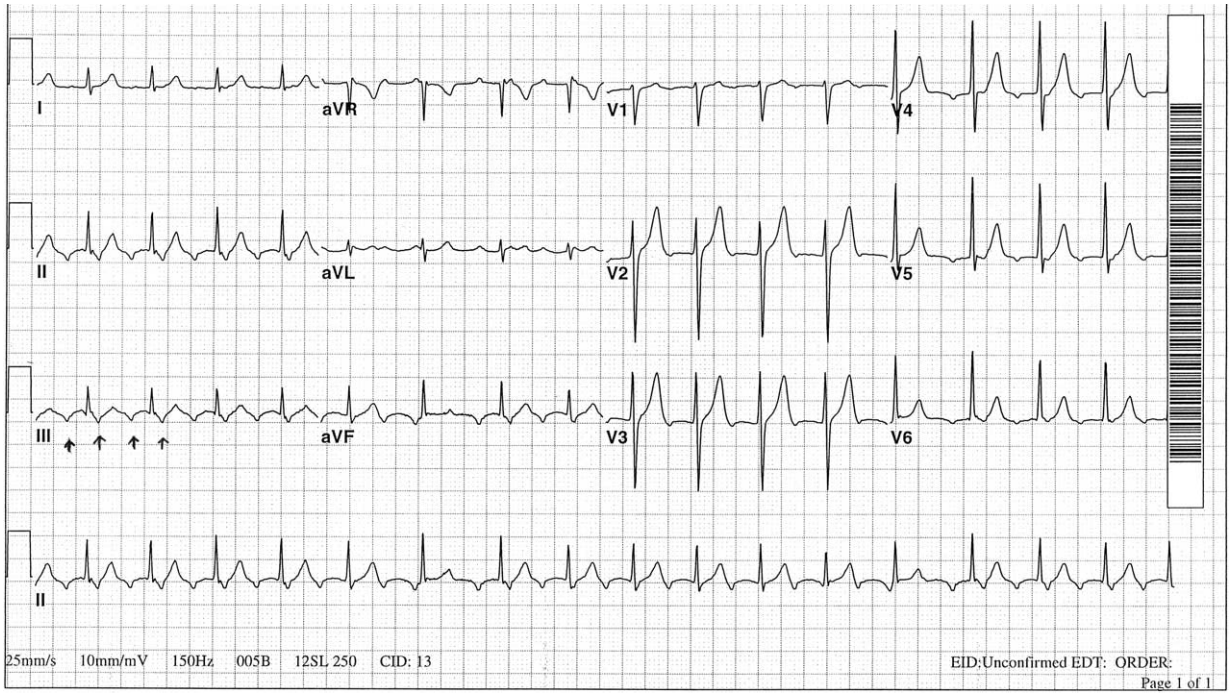

Figure 9. Ectopic atrial rhythm with 2:1 conduction and a ventricular rate of 100 beats/min. ST segment depression is no longer present (compared to Figure 7). Note non-conducted P waves (arrows).

in the ED utilizing EKG analysis after various pharmacologic interventions. Diagnosis of an NCT can be improved by increasing the EKG chart speed to 50mm/s (12). The AV node-independent group of NCTs are usually atrial tachycardias and often arise from a single localized atrial focus. Because the AV node is not in-

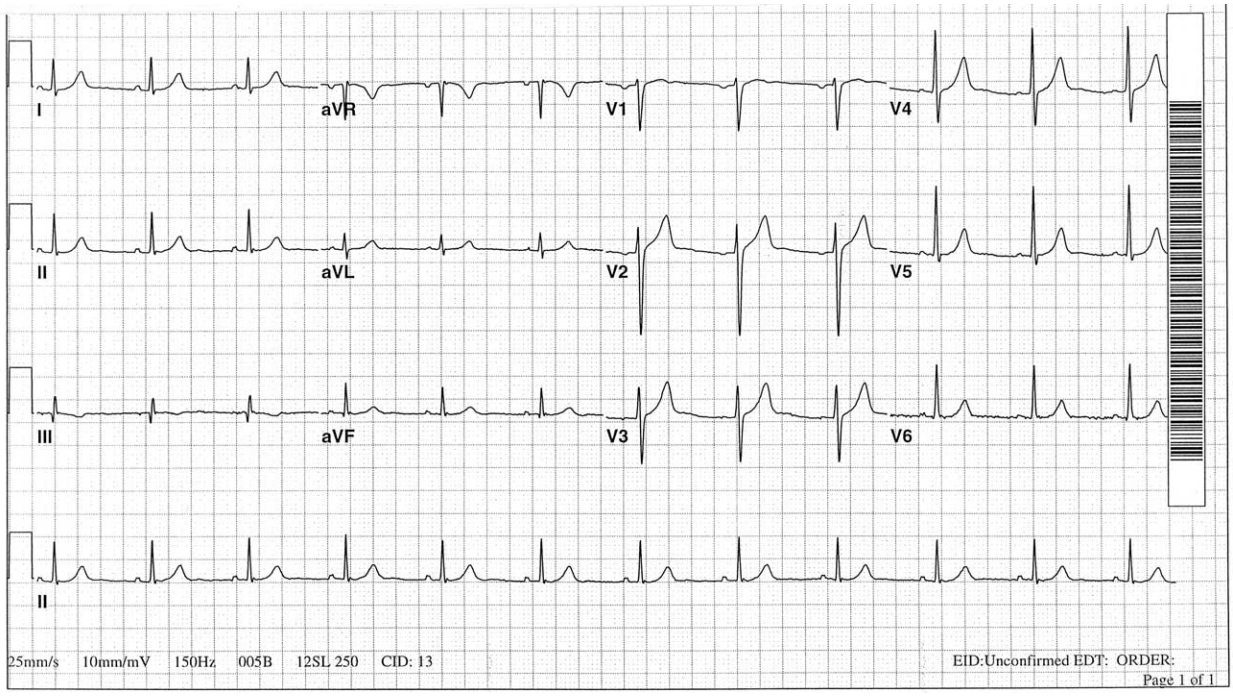

Figure 10. Normal sinus rhythm. Normal EKG.

Table 1. Common Causes of Narrow Complex Tachycardia

Thyrotoxicosis
Sympathomimetics and catecholamines
Cocaine
Shock
Pulmonary embolism
Myocardial infarction/ischemia/cardiomyopathy/congestive heart failure
Digoxin toxicity
Theophylline toxicity
Electrolyte abnormality
Alcohol
Cor pulmonale

volved in the initiation or continuation of this tachycardia, there may or may not be an associated AV block. The P-wave morphology is variable and depends on the location of the focus. The location of the atrial focus often can be determined by analysis of leads aVL and V1 on the 12-lead EKG. A positive P-wave deflection in V1 has a sensitivity of 93% and a specificity of 88% in predicting a left atrial focus and a positive P wave in aVL, and has a sensitivity of 88% and specificity of 79% for predicting a right atrial focus (13). The P wave in Figure 9 is positive in lead aVL, suggesting a right atrial origin. Subsequent EPS in this patient in fact determined a right atrial origin to the atrial tachycardia.

Several studies have attempted to develop EKG criteria to distinguish the different types of NCT (13–16). Although none of the features studied is highly sensitive or specific, it is worth mentioning the criteria. Kalbfleisch et al. report that a pseudo R' in lead V1 and a pseudo S wave in the inferior leads are more common in AV node reentrant tachycardia. They define pseudo R' as an apparent R' deflection in lead V1 during tachycardia that was not present during sinus rhythm. The pseudo S wave was an apparent S wave present during tachycardia in the inferior leads that was not present during sinus rhythm. Approximately 20% of the tachycardias reviewed were incorrectly classified (14). P waves are not usually well seen and are buried in the QRS complex. This may cause a distortion of the QRS complex. This distortion of the terminal portion of the QRS is often called a pseudo S wave in the inferior leads or a pseudo R' in lead V1. This type of NCT is usually self-limiting and readily terminated by vagal maneuvers (17).

An AV nodal reentry tachycardia is usually initiated by an atrial premature beat. The anterograde conduction is through a slowly conducting pathway and the retrograde conduction is through a more rapidly conducting pathway (18).

MAT (Case 2) is an uncommon tachydysrhythmia that is usually observed in the setting of COPD. Diagnostic criteria include the presence of at least 3 different P-wave morphologies. The etiology is not clearly understood but is likely due to abnormal automaticity (19). The echocardiographic findings in MAT are not different than those found in atrial fibrillation, and therefore the genesis of MAT is less likely to be due to anatomic factors (20).

Atrial fibrillation is a very common atrial tachycardia that is readily diagnosed by the irregularly irregular rhythm and lack of P waves and is seen in paroxysmal and persistent forms. The disorganized atrial activity results in a large number of impulses reaching the AV node, but few being conducted to the ventricle because of refractoriness.

The actual ventricular rate is dependent on numerous factors such as pre-existing heart disease, age, medications, and metabolic abnormalities. Atrial fibrillation often can be diagnosed on physical examination by the irregularly irregular heart sounds, the loss of *A* waves in the jugular venous pulse, and the varied intensity of the first heart sound. In atrial fibrillation, the left atrium is frequently enlarged by echocardiography. In patients with paroxysmal atrial fibrillation, the sinus rhythm EKG may demonstrate a wide and biphasic P wave, especially in lead V1, suggestive of an enlarged atrium. The "fibrillation" baseline, or *f* waves, may be gross and distinct or barely perceptible. There is no correlation of the *f* wave size with atrial size or type of underlying heart disease (17).

There are toxic, metabolic and cardiovascular etiologies of NCT (Table 1). The first responsibility is to identify the hemodynamically unstable patient. Treatment of NCT should be directed at establishing hemodynamic stability by either conversion to normal sinus rhythm or slowing the ventricular response. The goal of treatment depends on the nature of the dysrhythmia, the underlying cause, and previous hemodynamic status of the patient. Adenosine is the first line of treatment for NCT (21). A 6-mg bolus is effective in converting 90% of AV node-dependent tachycardias (22). Occasionally, 12 or 18 mg will be required, especially if methylxanthines, including caffeine, are used by the patient. If conversion does not occur, the transient AV block that often occurs helps in diagnosis of the dysrhythmia, as illustrated in Case 4, Figure 8 (23). Excellent rate control is achieved with i.v. bolus and continuous infusion of diltiazem or verapamil. Hypotension is the main adverse effect of calcium channel blockers and is found in 10–15% of treated patients (24). Other treatment options include beta blockers, amiodarone, or cardioversion (25). One recent study demonstrates a 94% conversion rate of SVT to sinus rhythm within 2 h after a single oral dose of 120 mg diltiazem and 80 mg propranolol (26). ED discharge is safe in selected patients after conversion to sinus rhythm (27,28). In addition, intravenous magne-

sium has been utilized with varying (33-58%) success in converting NCT to sinus rhythm (29,30).

Treatment of children with NCT is similar to adults, using vagal maneuvers and adenosine as first line management (31). Catheter ablation has a high success rate (32). Use of adenosine in pregnancy is probably safe, although large series have not been collected (33,34).

CONCLUSION

Patients with NCT demonstrate a wide range of clinical manifestations. Careful examination of the 12-lead EKG can usually identify the mechanism of this common tachydysrhythmia. Hemodynamically compromised patients should be immediately cardioverted. Selected patients often can be discharged from the ED but many will require hospital admission. Ablation therapy is becoming more common and many patients will be referred for this therapy.

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