Narrow Complex Tachycardia: Recognition and Management in the Emergency Room

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Abstract
Cardiac arrhythmias often present as urgent medical conditions requiring immediate care. Patient presenting with a tachyarrhythmia is a common finding in the emergency room. They also occur commonly in patients undergoing non-cardiovascular procedures including surgeries. It is thus pertinent that the physician handling such cases must be appropriately trained to diagnose and provide emergency management till the case is referred to a specialist. Most cases present as narrow or a wide complex tachycardia. The differential diagnosis is arrived at by deciding on the ECG morphology along with relevant history and physical examination where feasible. This article describes the bedside approach to diagnose and treat an arrhythmia presenting as a narrow complex.

INTRODUCTION
Cardiac tachyarrhythmias often present as urgent medical conditions requiring immediate care. With any type tachycardia, the first step is the assessment of the vital signs, to differentiate between tachycardias with or without hemodynamic instability. The arrhythmia may be the primary cause of hemodynamic instability, or a concomitant symptom of another serious condition. In this series of a two part article we shall take a panoramic view of recognizing and treating arrhythmias that present with either a narrow complex ECG or a wide complex tachycardias (WCT) in an emergency setting. This differentiation of WCT (QRS duration ≥120 msec) and narrow (QRS duration <120 msec) is practical and easily determined even in an emergency setting where time is an important constraint to make a diagnosis and apply appropriate therapy.

In the case of hemodynamic instability at presentation, or at any time during the treatment of cardiac tachyarrhythmias, electrical cardioversion should be immediately considered as a life-saving measure. Cardioversion is a safe and effective therapy for almost all tachyarrhythmias. History taking and a comprehensive physical examination is important but can be done later after the patient stabilizes. If the patient does not require immediate cardioversion, a thorough physical examination along with relevant history-taking is mandated along with a 12-lead ECG. The ECG interpretation should always be made in the light of history and physical examination. In the haste to treat the arrhythmia itself, concomitant treatment of the underlying cause and precipitating factors, as well as supportive care should not be delayed. Pharmacological antiarrhythmic therapy can be considered if the patient is hemodynamically stable, and/or electrical cardioversion is not feasible, desirable or successful. To determine the appropriate therapy, the first step however is to classify tachycardias as regular or irregular, narrow or wide QRS complex; the latter may be monomorphic or polymorphic.

NARROW QRS COMPLEX TACHYCARDIA (NCT)
Once a NCT is identified the first step is to determine whether the rhythm is regular or irregular. The differential diagnosis of a NCT is given in Table 1.

Irregular narrow QRS complex tachycardia
The commonest arrhythmia in clinical practice with this

Table 1: Differential diagnosis of narrow complex tachycardia

<table>
<thead>
<tr>
<th>Narrow complex tachycardia</th>
<th>Irregular</th>
<th>Regular</th>
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<td>Atrial fibrillation</td>
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<td>Atrial flutter with variable block</td>
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Atrial fibrillation is the most common sustained arrhythmia encountered in clinical practice. Approximately 0.4 percent of persons in the general population have permanent or intermittent atrial fibrillation, and the prevalence of the arrhythmia increases to 6 percent in persons older than 80 years. Atrial fibrillation can result in serious complications, including congestive heart failure, myocardial infarction, and thromboembolism. Considerable evidence has accumulated that atrial fibrillation occurring in a background of rheumatic heart disease, as is rampant in India, is decidedly associated with increased risk of stroke. In the Framingham Heart Study, patients with rheumatic heart disease and AF had a 17-fold increased risk of stroke compared with age matched controls, and the attributable risk was five times greater than in those with nonrheumatic AF. In recent years, management strategies for atrial fibrillation have expanded significantly, and new drugs for ventricular rate control and rhythm conversion have been introduced. Physicians, handling such cases in the emergency room thus have the challenge of keeping current with recommendations on heart rate control, antiarrhythmic drug therapy, cardioversion, and relevant antithrombotic therapy.

Diagnosis

The diagnosis of atrial fibrillation should be considered in patients who present with complaints of shortness of breath, dizziness, or palpitations. The arrhythmia should also be suspected in patients with acute fatigue or exacerbation of congestive heart failure. In some patients, atrial fibrillation may be identified on the basis of an irregularly irregular pulse or an electrocardiogram (ECG) obtained for the evaluation of another condition. Cardiac conditions commonly associated with the development of atrial fibrillation include rheumatic mitral valve disease, coronary artery disease, congestive heart failure, and hypertension. Noncardiac conditions that can predispose patients to develop atrial fibrillation include hyperthyroidism, hypoxia, alcohol intoxication, and surgery.

The ECG is the mainstay for diagnosis of atrial fibrillation (Fig. 1). An irregularly irregular rhythm, inconsistent R-R interval, and absence of P waves are usually noted on the cardiac monitor or ECG. Atrial fibrillation waves (f waves), which are small, irregular waves seen as a rapid-cycle baseline fluctuation, indicate rapid atrial activity (usually between 450 and 600 beats per minute) and are the hallmark of the arrhythmia, although they may not always be visible on a surface ECG. Atrial fibrillation should also be distinguished from atrial tachycardia with variable atrioventricular block, which usually presents with an atrial rate of 120-200 beats per minute. In this condition, the atrial rate is regular (unlike the irregular disorganized f waves of atrial fibrillation), but conduction to the ventricles is irregular. The resultant irregularly irregular ventricular rhythm may be difficult to differentiate from atrial fibrillation. However for emergency practice it is useful to consider any irregularly irregular tachycardia with no clear p waves seen as atrial fibrillation unless proven otherwise.

Treatment

Ventricular rate control

Ventricular rate control may be the only therapy needed if electrical or pharmacological cardioversion is not planned. However, in patients with recent onset atrial fibrillation, intravenous rate control with a beta-blocker or a calcium antagonist, followed by pharmacological cardioversion is a reasonable strategy. This protocol has two predominant advantages; it will remove the risk of 1:1 conducted atrial flutter induced by class IC drugs (such as flecainide) if used. Second, it will result in immediate symptom relief until cardioversion takes place.

Intravenous beta-blocker (metoprolol, propranolol, esmolol or other agents), or calcium-blocker (verapamil, diltiazem) therapies have both been shown in randomized controlled trials to be effective in ventricular rate control in patients with preserved left ventricular function, with a rapid onset of action. However, beta-blockers and calcium antagonists should be used with caution in patients with CHF. A small study suggested that intravenous diltiazem is safe in patients with moderate to severe CHF. Verapamil should not be used in patients with left ventricular dysfunction given its negative inotropic effect. Table 2 summarizes the drugs used for rate control.

Digoxin has been used for many years for the treatment of recent onset atrial fibrillation. However, neither oral, nor intravenous digoxin is more effective than placebo for the restoration of sinus rhythm and it may even prolong the duration of atrial fibrillation. Although digoxin may reduce the resting ventricular rate, its efficacy is reduced in conditions with high sympathetic tone, and its onset of therapeutic effect only begins at 60 minutes, peaking at 6 hours. Digoxin can be a useful addition to beta blockade or calcium channel blockade for the treatment of atrial fibrillation with rapid ventricular response, especially in the setting of atrial fibrillation associated with congestive heart failure.

Intravenous amiodarone is also effective in ventricular rate control; however, it may lead to cardioversion and therefore the risk of thromboembolism should be considered before its application. A recent randomized trial compared iv. amiodarone with diltiazem in critically ill patients. Amiodarone and diltiazem were both effective in achieving rate control at 4 hours; diltiazem caused hypotension more frequently. Thus, amiodarone may be especially useful in patients with left
ventricular dysfunction.

**Pharmacological cardioversion**

Patients with atrial fibrillation or flutter that is clearly of no more than 48 hours duration may be cardioverted electrically or pharmacologically without anticoagulation with minimal risk of stroke. If the duration of arrhythmia is more than 48 hours, or unknown, cardioversion should only be attempted after three weeks of therapeutic anticoagulation, or after performing a transesophageal echocardiogram (TEE), to exclude the presence of atrial thrombi, and with concomitant heparin therapy. Whether pre-procedural anticoagulation or TEE is utilized, therapeutic anticoagulation should be continued for at least 4 weeks post-cardioversion for patients in atrial fibrillation for >48 hours post-cardioversion.

The success of pharmacological cardioversion is strongly dependent on the duration of atrial fibrillation, with higher success for shorter duration episodes and very low success rates in chronic atrial fibrillation. Importantly, in recent onset (<24 h) atrial fibrillation, the spontaneous cardioversion rates are also very high, approaching 50-70% at 24 hours. A description of the different drugs that can be used for medical cardioversion is provided below (Table 3).

**Propafenone**

Propafenone is a Vaughan-Williams classification 1C agent (pure Na channel blocker), available intravenously in some countries. Intravenous propafenone was effective in the cardioversion of recent onset (<48 h) atrial fibrillation in randomized, placebo controlled trials, with a conversion rate of 76.1% at 24 hours. A recent meta-analysis of a single dose of oral propafenone for conversion of recent onset atrial fibrillation also showed a high conversion rate, 72-78% within 8 hours. In this meta-analysis, the incidence of 1:1 conduction atrial flutter as an adverse effect was only 0.3%. These data further support the efficacy of oral and intravenous propafenone in the treatment of recent onset (<48 h) atrial fibrillation without structural heart disease.

Propafenone should be avoided in patients with CAD or left ventricular systolic dysfunction. It has limited efficacy for cardioversion in persistent (longer than 48 hours duration) atrial fibrillation and in atrial flutter.

**Flecainide**

Flecainide is also a drug with class IC mechanism of action. A recent randomized study compared the efficacy of intravenous and oral flecainide in recent onset (48 hours) atrial fibrillation. Intravenous flecainide restored sinus rhythm more rapidly than oral; however, at 8 hours 75% of the oral and 72% of the intravenous flecainide group had converted. A recent metaanalysis showed that oral single dose flecainide...
converted recent onset atrial fibrillation in 75-91% of the patients at 8 hours, and it was significantly more efficacious than placebo, and at least as efficacious as oral propafenone.\textsuperscript{11}

Similar to propafenone, flecainide also can cause proarrhythmia in the form of atrial flutter with 1:1 conduction, or bradyarrhythmia after conversion to sinus rhythm. It should also be avoided in patients with structural heart disease, and it has limited efficacy in patients with atrial flutter.\textsuperscript{1}

**Amiodarone**

Amiodarone has been utilized both for recent onset and persistent atrial fibrillation/flutter. It can be administered in the form of single intravenous bolus, bolus followed by infusion, iv bolus followed by oral doses and single or divided high oral doses for the purpose of rate control or pharmacological cardioversion. The different dosing regimens and clinical settings make comparison of these studies difficult.

A recent randomized study compared intravenous flecainide, propafenone, and amiodarone (5mg/kg, followed by 50 mg/h) in recent onset (<48 hours) atrial fibrillation. The conversion rates at 12 hours were 90%, 72%, 64% in the flecainide, propafenone and amiodarone group, respectively. The conversion rates in the propafenone and amiodarone groups did not differ significantly (p=0.39), but flecainide was significantly more effective than propafenone (p=0.022), or amiodarone (p=0.002). However the conversion rates at 24 hours of study drug administration was not reported, and there was no placebo group in this study.\textsuperscript{12}

A recent meta-analysis showed that the conversion rates of recent onset atrial fibrillation with bolus amiodarone vary from 34-69%. Conversion rates are increased to 55-95% with the bolus followed by infusion administration.\textsuperscript{13} Another meta-analysis showed an average 76% conversion rate for iv, amiodarone, 72% for other anti-arrhythmics and 60% for placebo, respectively.\textsuperscript{14} Another recent meta-analysis of studies of amiodarone versus placebo and IC agents was reported.\textsuperscript{15} The 24 hour conversion rates were 82% and 56%, for amiodarone and placebo, respectively and 66%, and 71% for amiodarone and IC agents, respectively.

Similar to other drugs, the conversion rate with amiodarone in patients with atrial fibrillation of longer than 7 days duration is low,\textsuperscript{1} and its efficacy in conversion of atrial flutter is not well established. Amiodarone can be safely used in patients with left ventricular dysfunction and coronary artery disease.\textsuperscript{1}

A reasonable strategy is to use amiodarone if rapid conversion is not desired or necessary, especially in patients with underlying heart disease or heart failure who require hospital admission. In general, amiodarone is somewhat less effective than the other agents described here at conversion of atrial fibrillation to sinus rhythm, but is among the most effective agents for maintenance of sinus rhythm after cardioversion. Long term use of amiodarone is often limited by toxicity to the thyroid, lungs and other tissues.

**Ibutilide**

Ibutilide is a class III antiarrhythmic agent, only available intravenously. It blocks the rapid component of the delayed rectifier potassium channel (I\textsubscript{Kr}), and augments plateau sodium current,\textsuperscript{16} thereby markedly increasing the QT interval. In randomized trials it was effective in conversion of atrial fibrillation and flutter of more than 7 days (3h-90 days) duration, with conversion rates of 34.9% to 47%.\textsuperscript{17-19} The conversion rates for atrial flutter were much higher than for atrial fibrillation. The highest conversion rates were achieved with short arrhythmia duration (<30 days). The incidence of proarrhythmia due to torsades de pointes VT was 4.3%, including 1.7% being sustained.\textsuperscript{19} In another recent trial, ibutilide pretreatment also increased the success rate of electrical cardioversion in persistent (mean 117 days) atrial fibrillation.\textsuperscript{20} Sustained polymorphic tachycardia occurred in 3% of the patients, in the setting of EF<20%.

In a recent observational study, intravenous ibutilide was administered to patients treated with long term oral amiodarone therapy and persistent atrial fibrillation or flutter; 39% of the patients with atrial fibrillation and 54% of those with flutter converted; the incidence of torsade was 1.4%.\textsuperscript{21} It should be noted that due to the pro-arrhythmic risk of ibutilide, patient monitoring with QT analysis must be performed for at least 6 hours post-administration of the agent.

**Dofetilide**

Dofetilide is a pure class III antiarrhythmic drug\textsuperscript{22} with QT effects similar to ibutilide. Randomized clinical trials of intravenous dofetilide in patients with atrial fibrillation/flutter of up to 6 months duration, showed conversion rates of 30-38%.\textsuperscript{23-26} The conversion rates were significantly higher with patients with atrial flutter (54-70%), and atrial fibrillation with less than 24 hours duration (67%). The incidence of torsade was 3-8%. The intravenous form of dofetilide is currently only investigational.

Oral dofetilide was tested in two similar, randomized, controlled multicenter trials, the EMERALD and SAFIRE-D study.\textsuperscript{27,28} In these studies, patients with atrial fibrillation/flutter from 1 week to 2 years duration were involved. The conversion rates at 3 days were highest in the dofetilide 500 mcg bid treatment arms, 29% and 29.9%. The incidence of torsades VT was 0.8%, and one sudden cardiac death occurred (0.4%).

Dofetilide is thus effective in the conversion of atrial flutter, and moderately effective in the conversion of atrial fibrillation of longer duration, with a moderate risk of proarrhythmia; it has a delayed onset of action, and requires in-hospital initiation of therapy with QT monitoring.

**Other Irregular Arrhythmias**

Multifocal atrial tachycardia (MAT) and atrial flutter with variable AV block can produce irregularly irregular rhythm which may be mistaken for atrial fibrillation.

MAT is a form of atrial tachycardia with at least three p wave morphologies and often occurs in the setting of severe metabolic disease. It can be treated by rate control with beta blockers or calcium channel blockers to slow AV nodal
conduction. It is not typically responsive to electrical cardioversion, but does respond to treatment of the underlying illness.

Atrial flutter may present either with a consistent A:V relationship, producing a regular ventricular rhythm, or with a variable A:V relationship, producing an irregular ventricular rhythm. The diagnosis and treatment of atrial flutter is discussed below.

**Atrial Flutter**

Classical atrial flutter is caused by a re-entrant circuit confined to the right atrium in which the impulse travels up the atrial septum, with epicardial breakthrough superiorly in the right atrium where the impulse then travels inferiorly down the right atrial free-wall to re-enter the atrial septum. When the circulating wave-front re-enters the atrial septum, it travels through an isthmus bounded by the inferior vena cava, Eustachian ridge, the coronary sinus os on one side and the tricuspid valve annulus on the other side (the cavo-tricuspid isthmus).

Atrial flutter by this circuit is called typical atrial flutter, although it also has been called common atrial flutter and counterclockwise atrial flutter. A 12-lead ECG during typical atrial flutter with characteristic negative “sawtooth” atrial flutter waves in leads II, III, and aVF and upright flutter waves in V1 as shown in Fig. 2. It is also recognized that impulses can travel in this re-entrant circuit in the opposite direction, so that the impulse travels down the atrial septum and breaks through to the epicardium via the same atrial flutter isthmus to travel up the right atrial free-wall and then re-enter the septum superiorly. This form of atrial flutter is called reverse typical atrial flutter, although it has in the past been called atypical atrial flutter, clockwise atrial flutter, uncommon atrial flutter, and rare atrial flutter. A 12-lead ECG during reverse typical atrial flutter is with characteristic positive flutter waves in leads II, III, and aVF. Other atrial flutter circuits have been described originating in both the left and right atrium, often confined to the right atrium in which the impulse travels up the atrial septum, with epicardial breakthrough superiorly in the right atrium where the impulse then travels inferiorly down the right atrial free-wall to re-enter the atrial septum.

Atrial flutter generally presents as narrow complex tachycardia with a sawtooth baseline. Flutter with 2:1 A:V conduction is common, and so atrial flutter should be considered for any arrhythmia with narrow complexes at the rate of 150 beats per minute. Although atrial flutter can present with nearly any ventricular rate, 1:1 A:V conduction with ventricular rates of up to and above 300 bpm is occasionally encountered in the ER (either with a narrow QRS or with aberrant conduction producing a wide QRS). This constitutes a true medical emergency and rapid treatment with IV AV nodal blocking agents or electrical cardioversion is generally indicated.

**Treatment**

Atrial flutter is treated much like atrial fibrillation. The mainstays of treatment include ventricular rate control and anti-coagulation for stroke prevention. Rate and rhythm control can be generally achieved with the same agents used for atrial fibrillation. Electrical cardioversion is usually effective in the acute conversion of atrial flutter although recurrence is common. Catheter ablation is the only curative treatment for this disorder.

**PSVT**

Paroxysmal supraventricular tachycardia (PSVT) is a common group of arrhythmias that present with NCT. PSVT in the absence of structural heart disease can present at any age but most commonly first presents between ages 12 and 30. Most patients with PSVT due to atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT) do not have associated structural heart disease, although exceptions (e.g., Epstein’s anomaly, familial preexcitation) do exist. Atrial tachycardias are another cause of PSVT and are often associated with structural heart disease. In patients without structural heart disease, the physical exam during PSVT is significant mainly for rapid heart rate although occasionally PSVT with very rapid ventricular rate can cause hemodynamic instability leading to pre-syncpe or syncpe. History, physical exam, and an ECG constitute an appropriate initial evaluation. A 12-lead ECG during tachycardia is helpful for defining the mechanism of PSVT. Patients with panic disorder report symptoms similar to those of PSVT, and an ECG during palpitations shows mostly sinus tachycardia thus aiding in diagnosis.

**PSVT? But what is the mechanism?**

An ECG showing PSVT, but with a stable hemodynamics should be approached by asking the question-what is the mechanism of this arrhythmia? To answer the above question one must examine the P waves. The different forms of PSVT are illustrated in Fig. 3. The P-wave position relative to the QRS complex during AVNRT depends on the types of
Atrial tachycardia is the least common form of PSVT in normal individuals but predominates in patients with significant atrial scarring, especially from prior atrial surgery. Atrial tachycardias may be caused by enhanced or triggered automaticity or by reentry. Because the AV node and ventricle are not required participants in the arrhythmia, AV block commonly occurs. The PR or apparent RP’ intervals depend on AV conduction properties. P-wave morphology depends on the site of origin in the atrium.

We shall now discuss each forms of PSVT in detail to understand their characteristics which will help in recognizing them when they present to the emergency room.

**AV nodal re-entrant tachycardia (AVNRT)**

AVNRT is a common arrhythmia which occurs in patients with two functionally distinct conduction pathways through the atrioventricular node, typically fast and slow pathways. The slow pathway usually has a shorter refractory period than the fast pathway. Both the fast and slow pathways are necessary to maintain AVNRT. The common form of AVNRT is typically initiated when an atrial premature beat blocks in the fast pathway, conducts down the slow pathway, and returns retrograde via the fast pathway to depolarize the atrium, thus creating a re-entrant circuit in the AV node. During the uncommon form of AVNRT the wave front propagates in the opposite direction, conducting down the fast pathway and returning via the slow pathway. The fast pathway is located anteriorly along the septal portion of the tricuspid annulus, near the compact atrioventricular node, whereas the atrial insertion of the slow pathway is located more posteriorly along the tricuspid annulus, closer to the coronary sinus os.

**ECG**

Rate is in the range 120-250 beats/minute (commonly 170-200). It presents as a NCT with no visible p waves. (Fig. 4).

**Characteristics**

It starts abruptly due to an atrial premature beat (APB) and ends abruptly. The APB that initiates the tachycardia generally is conducted with a prolonged PR interval.

Carotid massage can either slow or terminate the tachycardia.

P waves are generally not seen. They are buried in the QRS complex and are thus not seen in the most common form of this tachycardia, i.e., the slow-fast variety, occurring in 66% cases. However, if the tachycardia occurs through the
fast-slow pathway, then retrograde p waves may be visible after the QRS complex due to slow conduction over the retrograde pathway. It must be remembered that both the fast and slow pathways are within the AV node.

Clinical features

AVNRT is typically found in patients with no structural heart disease.

It may produce symptoms like palpitations, polyuria, etc. It can also produce angina, syncope, heart failure or even shock.

Tachycardia, typically with a regular pulse.

S1 is of constant intensity.

Prominent jugular venous pulsations due to atrial contraction against closed AV valves may be a clue to AVNRT.

Treatment

Because PSVT will rarely be so poorly tolerated that it requires immediate termination with electrical cardioversion, most patients can be managed with physiological maneuvers or drugs.

Many patients learn to terminate acute episodes of PSVT by using vagal maneuvers early during an episode of PSVT. Valsalva is the most effective technique in adults, but carotid massage may also be effective.29 Facial immersion is the most reliable method in infants. Vagal maneuvers are less effective once a sympathetic response to PSVT has become established, so patients should be advised to try them soon after the onset.

Adenosine and the non-dihydropyridine calcium antagonists verapamil and diltiazem are the intravenous (IV) drugs of choice for termination of PSVT.29 Adenosine is an endogenous purine nucleoside that slows AV nodal conduction and results in transient AV nodal block. Conduction in rapidly conducting accessory pathways is usually not affected, but decremental pathways may exhibit block. Exogenous adenosine is cleared extremely rapidly from the circulation by cellular uptake and metabolism, with an estimated half-life of <5 seconds.30 Adenosine effect is typically seen 5 to 30 seconds after rapid peripheral infusion as a first-pass effect. Administration via a central line requires dose reduction; 3 mg would be the appropriate initial dose. The effective dose range for peripheral administration in adults is 2.5 to 25 mg. If no upper dosage limit is imposed, at least transient termination of AV node-dependent PSVT can be produced in all patients. The recommended adult dosage for peripheral infusion is 6 mg, followed by a 12 mg dose and then an 18 mg dose if needed. In pediatric patients, the dose range is 50 to 250 µg/kg administered via an upward dose titration. Because of the ultrashort duration of action, cumulative effects of sequential doses are not seen.

Minor side effects, including transient dyspnea or chest pain, are common with adenosine. Sinus arrest or bradycardia may occur but typically will resolve quickly if appropriate upward dosing is used. With PSVT termination, atrial and ventricular premature beats are frequently seen, and a few patients with adenosine-induced polymorphic ventricular tachycardia have been reported.29 These patients had long baseline QT intervals and long pauses during adenosine-induced AV block. Adenosine shortens the atrial refractory period, and atrial ectopy may induce atrial fibrillation. This may be dangerous if the patient has an accessory pathway capable of rapid antegrade conduction. Because adenosine is cleared so rapidly, re-initiation of PSVT after initial termination may occur. Either repeat administration of the same dose of adenosine or substitution of a calcium channel blocker will often be effective in this situation.

Adenosine mediates its effects via a specific cell surface receptor, the A1 receptor. Theophylline and other methylxanthines block the A1 receptor. Caffeine levels achieved after beverage ingestion may be overcome by the doses of adenosine used to treat PSVT. Dipyridamole blocks adenosine elimination, thereby potentiating and prolonging its effects. Cardiac transplant recipients are also unusually sensitive to adenosine. If adenosine is chosen in these latter situations, much lower starting doses (i.e., 1 mg) should be selected.

The AV node action potential is calcium channel-dependent, and the non-dihydropyridine calcium channel blockers verapamil and diltiazem are very effective for terminating AV node-dependent PSVT.31,32 The recommended dosage of verapamil is 5 mg IV over 2 minutes, followed in 5 to 10 minutes by a second 5 to 7.5 mg dose. The recommended dosage of diltiazem is 20 mg followed, if necessary, by a second dose of 25 to 35 mg. PSVT termination should occur within 5 minutes of the end of the infusion, and over 90% of patients with AV node-dependent PSVT respond.

As with adenosine, transient arrhythmias, including atrial and ventricular ectopy, atrial fibrillation, and bradycardia, may be seen after PSVT termination with calcium channel blockers. Hypotension may occur with calcium channel blockers, particularly if the PSVT does not terminate. Calcium channel blockers are not recommended in infants and neonates with PSVT because of reports of cardiovascular collapse.33

Adenosine and verapamil have been shown to have equivalent efficacy in several randomized clinical trials.29,30,34 Most PSVT patients can be acutely managed with either agent. To minimize the potential for adverse effects, adenosine should be selected in patients with severe hypotension or heart failure, in infants and neonates, and in those at risk for severe bradycardia. Verapamil and diltiazem should be chosen for patients with poor venous access, patients with bronchospasm, and those taking agents that interfere with adenosine action or metabolism.

It should be noted that while pharmacological therapy can be useful for acute control of symptoms of PSVT and chronic arrhythmia suppression, catheter ablation is the only curative therapy for recurrent arrhythmia.

Atrioventricular reentrant tachycardia (AVRT)

AVRT can present as a narrow complex tachycardia (orthodromic AVRT) or as a wide complex tachycardia
Atrial tachycardias

These include different forms of tachycardia occurring due to automatic foci or reentry in the atrium. P-wave morphology depends on the site of origin in the atrium. If the site of origin is within or involves the sinus node region, sinus node reentrant or inappropriate sinus tachycardia is identified. Left atrial tachycardia can be identified by positive p wave morphology in lead V1 and a negative or biphasic p in aVL (although AVRT via a left-sided accessory pathway, producing retrograde P waves originating in the left atrium must also be considered). Frequently there is AV block evident in cases of atrial tachycardia and thus more P waves are seen than QRS complexes, thus simplifying diagnosis.

Treatment

Limited data are available on acute pharmacological therapy of atrial tachycardias. Automatic or triggered tachycardias and sinus node reentry may respond to adenosine, verapamil, diltiazem, or β-adrenergic blockers. Other atrial tachycardias may respond to class I or class III antiarrhythmic drugs given orally or parenterally. The response of atrial tachycardia to electrical cardioversion is variable. Typically re-entrant forms of atrial tachycardia will terminate with electrical cardioversion (although they may recur), while automatic forms generally will not terminate with cardioversion. It is often difficult to determine whether a particular atrial tachycardia is re-entrant or automatic in origin before initiating therapy. In the majority of cases, AV nodal blocking agents (beta blockers and calcium channel blockers) will slow the ventricular rate of patients presenting with atrial tachycardia and rapid ventricular rates. Anti-arrhythmic drugs including Type I and Type III agents and amiodarone can be effective in suppressing atrial tachycardias when other therapies are ineffective, and catheter ablation can prove curative for refractory cases.

Fig. 5: A. 12-lead ECG during tachycardia in a patient with WPW syndrome. Note the NCT. B. Same patient in sinus rhythm. Note the delta waves.

(antidromic AVRT or orthodromic AVRT with aberrant ventricular conduction). The position of p waves helps to differentiate AVRT from AVNRT. In typical AVNRT, no p waves are seen as they are tucked inside the QRS; at times they also may be found in the terminal part of the QRS producing a pseudo S’ in lead III and a pseudo R’ in V1. In cases of atypical AVNRT, p waves can be seen with a long R-P interval which can be difficult to distinguish from AVRT.

In case of AVRT the RP interval is generally at least 100 msec.

It is important in emergency room practice to identify patients with anterograde accessory pathway conduction (typically visualized by a delta wave) (Fig. 5) as use of adenosine or calcium channel blockers can paradoxically increase conduction through the accessory pathway (which has faster conducting properties than the normal AV node in which conduction remains blocked due to adenosine) resulting in dangerous or fatal arrhythmias. Although AV nodal blocking agents can be effective in terminating both AVNRT and AVRT, these drugs can prove dangerous in patients with anterograde conduction through an accessory pathway who develop rapid atrial rhythms (typically atrial fibrillation or atrial flutter). These atrial rhythms may be conducted to the ventricles at extremely rapid rates via the accessory pathway in the presence of these agents, potentially leading to hemodynamic decompensation or ventricular fibrillation. In patients with manifest accessory pathways as identified by a delta wave, Class IA or IC agents will generally inhibit accessory pathway conduction, and so are safer in these patients, either when used alone or in combination with AV nodal blocking agents. As these pre-excited tachycardias produce a wide QRS complex, they will be discussed in greater detail in the chapter on wide complex tachycardias.

References