CLINICAL PRACTICE

Supraventricular Tachycardia

Etienne Delacrétaz, M.D.

This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

A 28-year-old woman suddenly has rapid palpitations accompanied by chest pain and dizziness while playing her cello. She is brought to an emergency department. She has a faint regular pulse of 190 beats per minute. Her blood pressure is 82/54 mm Hg. Cardiovascular examination reveals no signs of heart failure. An electrocardiogram shows a regular tachycardia with a narrow QRS complex and no apparent P waves. How should her case be managed?

THE CLINICAL PROBLEM

The term "supraventricular tachycardia" refers to paroxysmal tachyarrhythmias, which require atrial or atrioventricular nodal tissue, or both, for their initiation and maintenance. The incidence of supraventricular tachycardia is about 35 cases per 100,000 persons per year, and the prevalence is about 2.25 per 1000 (excluding atrial fibrillation, atrial flutter, and multifocal atrial tachycardia, which are not covered in this review).[1] Supraventricular tachycardias are often recurrent, occasionally persistent, and a frequent cause of visits to emergency rooms and primary care physicians.

Common symptoms of supraventricular tachycardia include palpitations, anxiety, light-headedness, chest pain, pounding in the neck and chest, and dyspnea. Syncope is uncommon, but some patients have serious psychological distress. Polyuria can occur in prolonged episodes, mainly owing to the release of atrial natriuretic factor.[2]

Most types of tachycardia have a reentry mechanism (Fig. 1), and they are classified according to the location of the reentry circuit (Fig. 2). Approximately 60 percent of cases are due to an atrioventricular nodal reentry circuit, and about 30 percent are due to an atrioventricular reentry circuit mediated by an accessory pathway — a short muscle bundle that directly connects the atria and ventricles.[3] Atrial tachycardia comprises about 10 percent of cases and often has a focal origin.[4] However, paroxysmal or persistent atrial tachycardia occurring long after cardiac surgery that involved a large atrial incision is usually caused by an intraatrial reentry.[5] Sinus-node reentrant tachycardia, inappropriate sinus tachycardia, ectopic junctional tachycardia, and nonparoxysmal junctional tachycardia are rare.[3]

Supraventricular tachycardias are not usually associated with structural heart disease, although there are exceptions (e.g., the presence of accessory pathways associated with hypertrophic cardiomyopathy or Ebstein's anomaly and atrial tachycardias in patients with congenital or acquired heart disease). Reentry arrhythmias are usually induced by premature atrial or ventricular ectopic beats, and precipitating factors — such as excessive intake of caffeine, alcohol, or recreational drugs and hyperthyroidism — can increase the risk of recurrence.
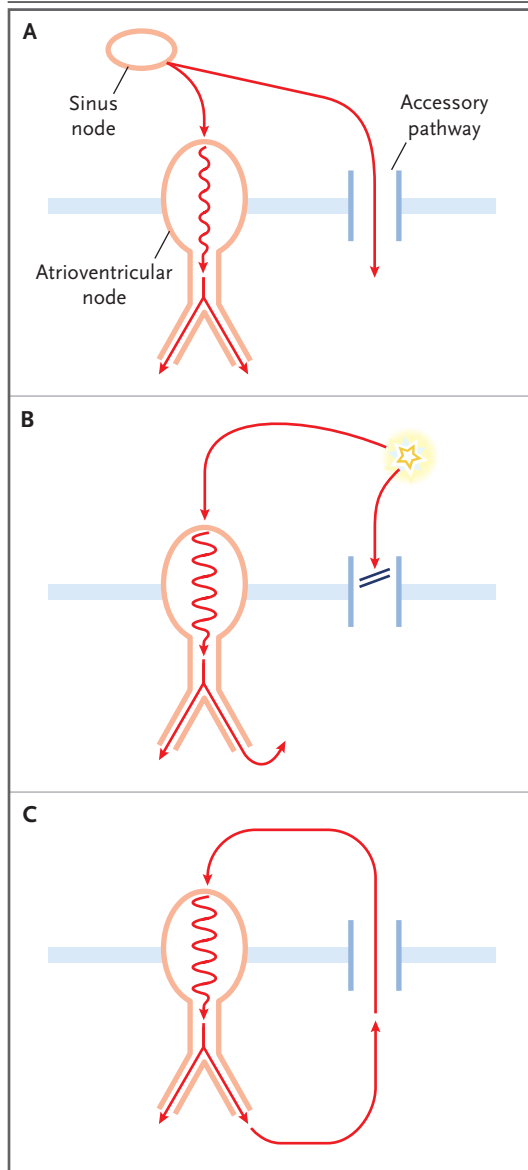
Figure 1. Mechanism of Reentry.

An impulse (Panel A, arrows), initiated normally in the sinus node, passes through two pathways — for example, the atrioventricular nodal connection and an accessory pathway. A premature atrial impulse (Panel B) occurs and reaches the accessory pathway when it is still refractory but conduction can occur in the atrioventricular node. The impulse takes sufficient time to circulate through the atrioventricular node and across the ventricle to allow the accessory pathway to recover its excitability and conduct the impulse back to the atrium (Panel C). The wave front reenters the atrioventricular node, continually encounters excitable tissue, and is perpetuated as a reentry circuit.

Figure 2 (facing page). Main Mechanisms and Typical Electrocardiographic Recordings of Supraventricular Tachycardia.

In patients with atrioventricular (AV) nodal reentrant tachycardia (Panels A and B), the atrioventricular node is functionally divided into two pathways that form the reentrant circuit. In the majority of patients, during this type of tachycardia, antegrade conduction to the ventricle occurs over the slow pathway and retrograde conduction over the fast pathway. The activation of atria and ventricles is synchronous so that the retrograde P wave is buried in the QRS complex, or it may be visible soon after the QRS complex (as pseudo r' in V1 or pseudo s in the inferior leads). Orthodromic AV reentrant tachycardia (Panels A and B) is the most frequent arrhythmia in patients who have an accessory pathway, with antegrade conduction through the AV node, activation of the ventricles, and retrograde conduction through the accessory pathway. Typically, there is a short RP interval, but a long RP interval may be associated with slow-conducting accessory pathways. With the use of adenosine or vagal maneuvers (Panel C), tachycardia often terminates with a retrograde P wave. In the approximately 70 percent of patients with this type of tachycardia who have an obvious accessory pathway, preexcitation may be seen in the ensuing beats; however, it is absent in the approximately 30 percent of patients with a concealed accessory pathway. In antidromic AV reentrant tachycardia (Panels A and B), the activation wave front travels in the opposite direction. On electrocardiography, it is impossible to distinguish antidromic AV reentrant tachycardia from ventricular tachycardia. Atrial tachycardias (Panels A and B) typically have a focal origin (star), and different mechanisms might be involved (reentry within several millimeters, automaticity, and triggered activity). RP intervals are typically long (longer than PR intervals), but this depends on the rate of tachycardia and properties of AV conduction. The PR interval can be prolonged by the use of vagal maneuvers and adenosine (Panel C), which may also produce a transient AV block. A substantial proportion of focal atrial tachycardias are terminated with the use of adenosine. Atrial flutter with 2:1 AV conduction (Panels A and B) may resemble atrial tachycardia or another type of supraventricular tachycardia and can be revealed when vagal maneuvers or adenosine is used (Panel C).

STRATEGIES AND EVIDENCE

GENERAL EVALUATION OF PATIENTS

While considering the patient's history, the clinician should assess the duration and frequency of episodes, the mode of onset, and possible triggers (including the intake of alcohol and caffeine or other drugs) as well as previous cardiac or other
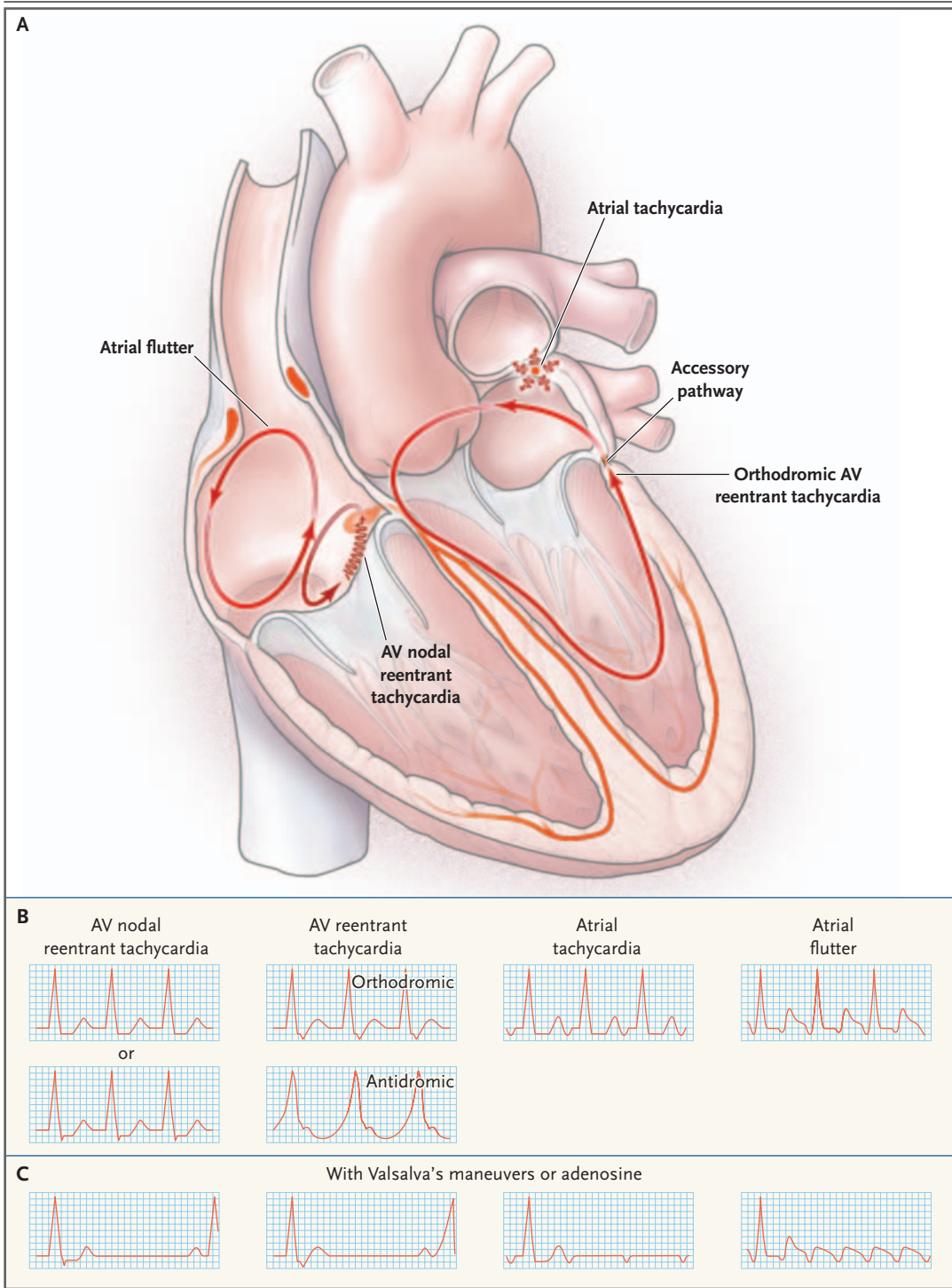
disease. These features are useful in distinguishing supraventricular tachycardia from other tachyarrhythmias (Table 1). Supraventricular tachycardias have a sudden onset and termination, in contrast to sinus tachycardias, which accelerate and decelerate gradually; however, some patients do not perceive the sudden onset of supraventricular tachycardia. It may be misdiagnosed as panic disorder.[6]

Physical examination during episodes may reveal the "frog sign" — prominent jugular venous

Table 1. Clinical Clues to the Differential Diagnosis of Supraventricular Tachycardia (SVT).*

Type of Tachyarrhythmia	Typical Age at Onset of Symptoms	Underlying Condition	Usual Presentation	Findings on Baseline ECG
Paroxysmal SVT	All ages	None (normal heart)	Abrupt onset and termination of regular palpitations, diaphoresis	Preexcitation common in AVRT
Atrial fibrillation, atrial flutter, multifocal atrial tachycardia	≥60 yr	Heart disease common (hypertension, ischemic or valvular heart disease)	Abrupt onset of paroxysmal, irregular palpitations; symptoms sometimes persistent and occasionally mild or absent	Signs of left ventricular hypertrophy; nonspecific repolarization abnormalities common
Sinus tachycardia	10–30 yr	None (normal heart)†	Progressive onset and termination of palpitations	Normal
Ventricular tachycardia‡	≥50 yr	Ischemic heart disease	Abrupt onset and termination of regular palpitations, syncope, or sudden death from cardiac causes	Pathological Q waves common

* Atrial fibrillation and atrial flutter are not included in this category. AVRT denotes atrioventricular reentrant tachycardia.
† In adults, sinus tachycardia is occasionally secondary to hyperthyroidism, anemia, infection, and heart failure. Sinus tachycardia may cause symptoms that can be difficult to differentiate from those due to tachyarrhythmia.
‡ Occasionally, ventricular tachycardia occurs in adults with no structural heart disease and is benign.

A waves due to atrial contraction against the closed tricuspid valve.[7] When sinus rhythm is restored, physical examination is usually normal, but a careful examination is warranted to rule out evidence of structural heart disease.

The usual presentation of supraventricular tachycardia on electrocardiography (ECG) is as a narrow-QRS-complex tachycardia (a QRS interval of less than 120 msec), but in some cases (less than 10 percent), wide-complex tachycardia is the manifestation of supraventricular tachycardia. After the restoration of sinus rhythm, the 12-lead ECG should be examined for the presence of delta waves, which indicate an accessory pathway (Fig. 2C). However, evidence of preexcitation may be minimal or absent if the accessory pathway (e.g., a left lateral accessory pathway) is located far from the sinus node or if, as occurs in approximately 30 percent of patients, the accessory pathways are "concealed" (i.e., they support exclusively retrograde conduction from the ventricle to the atrium and do not cause preexcitation of the ventricle during sinus rhythm). In ambulatory patients with frequent episodes (two or more per month) of supraventricular tachycardia, ECG recordings or event recorders (which record arrhythmias for up to seven days) may be useful to document arrhythmias.

An echocardiogram should be considered to rule out structural heart disease, even though it is uncommon. Because electrolyte abnormalities and hyperthyroidism may contribute to supraventricular tachycardia, it is reasonable to check potassium and serum thyrotropin levels; however, the tests for these values appear to have a low yield.

Electrophysiological testing allows for identification of the mechanism of arrhythmia, but this procedure is generally performed only if catheter ablation is considered. Table 2 summarizes conditions for which this testing is generally recommended.

TREATMENT

SHORT-TERM THERAPY

Figure 3 shows an algorithm for the management of acute supraventricular tachycardia. In rare cases, episodes of arrhythmia are so poorly tolerated that they require immediate electrical cardioversion. Most supraventricular tachycardias depend on the atrioventricular node for maintenance of the reentry circuit and can be interrupted by vagal maneuvers or pharmacologic agents that slow conduction through the atrioventricular node.

Vagal Maneuvers
Massage of the carotid sinus stimulates baroreceptors, which trigger a reflexive increase in the activity of the vagal nerve and sympathetic with-

Table 2. Conditions Warranting Referral
to an Electrophysiologist.

Tachycardia with a wide QRS complex

Supraventricular tachycardia

    In a patient with syncope or severe symptoms

    In a patient with drug resistance or intolerance

    In a patient who prefers to be free of drug therapy

Preexcitation syndrome (with or without supraventricu-
lar tachycardia)

drawal, slowing conduction through the atrioven-
tricular node. If the physical examination does not
reveal a carotid bruit and there is no history sug-
gesting carotid artery disease, pressure may be
applied at the level of the cricoid cartilage for
about five seconds with a firm circular movement.
If the tachyarrhythmia persists, the procedure
may be repeated on the opposite side. Other ap-
proaches to increasing vagal tone include having
the patient perform a Valsalva maneuver or (pri-
marily in children) apply an ice pack to the face.

A continuous 12-lead ECG recording of the
episode should be obtained during vagal maneu-
vers, since the way in which arrhythmias end may
provide clues to their mechanism (Fig. 2).[9]

Adenosine

As with vagal maneuvers, treatment with intrave-
nous adenosine has both diagnostic and thera-
peutic value. Data from randomized trials show
that supraventricular tachycardia is terminated in
60 to 80 percent of patients treated with 6 mg of
adenosine and in 90 to 95 percent of those treat-
ed with 12 mg.[10] In patients with atrial tachycar-
dias, adenosine causes a transient atrioventricular
nodal block or interrupts the tachycardia (Table 3
and Fig. 2).[10-12] ECG monitoring is required dur-
ing the administration of adenosine, and resusci-
tation equipment should be available in the event
that the rare complications of bronchospasm or
ventricular fibrillation occur. Adenosine is contra-
indicated in heart-transplant recipients and should
be used cautiously in patients with severe obstruc-
tive lung disease. Adenosine is also contraindi-
cated in patients with tachycardia with a wide
QRS complex (unless the diagnosis of supraven-
tricular tachycardia with aberrancy is certain).

Other Agents

If supraventricular tachycardia is refractory to
adenosine or rapidly recurs, clinical experience

indicates that the tachycardia can usually be ter-
minated by the administration of intravenous
verapamil or a beta-blocker.[11,13,14] As a next step,
procainamide, ibutilide, propafenone, or flecainide
can be given intravenously if the patient's blood
pressure is stable.[15] However, sequential trials
with different antiarrhythmic agents should be un-
dertaken only after careful consideration of their
possible negative hypotensive, bradycardic, and
proarrhythmic effects. At any point, electrical car-
dioversion is an alternative, but this technique
is generally considered in patients in hemodyna-
mically stable condition only if atrioventricular
nodal-blocking agents fail. Table 3 reviews medi-
cations used for acute supraventricular tachycar-
dia. These agents are contraindicated in patients
with severe hypotension, a history of heart block,
or congestive heart failure.

Atrial fibrillation with rapid ventricular con-
duction can occur spontaneously in patients with
the Wolff–Parkinson–White syndrome or during
treatment for supraventricular tachycardia. Emer-
gency-resuscitation equipment should be available,
since the arrhythmia can degenerate into ven-
tricular fibrillation if the accessory pathway has
a short refractory period (250 msec or less).[16]
Treatment with an electrical shock is a safe option.
If the patient's condition is hemodynamically sta-
ble, procainamide, ibutilide, propafenone, or fle-
cainide may be used; all have a rapid onset of
action, lengthen antegrade refractoriness of the
accessory pathway, and terminate atrial fibrilla-
tion in the majority of cases.[15]

Wide-QRS-Complex Supraventricular Tachycardia

Supraventricular tachycardia presents infrequently
as a wide-complex tachycardia, in which there is
an associated bundle-branch block or conduction
over an accessory pathway. Wide-QRS-complex,
regular tachycardia should routinely be treated as
ventricular tachycardia, unless the diagnosis of
supraventricular tachycardia with aberrancy or of
supraventricular tachycardia with preexcitation
is certain. Adenosine and other atrioventricular-
nodal–blocking agents are ineffective and poten-
tially deleterious in patients with ventricular tachy-
cardia.

LONG-TERM MANAGEMENT

The risk of recurrence after a single episode of
supraventricular tachycardia is not well defined,
and a single episode is not an indication for long-
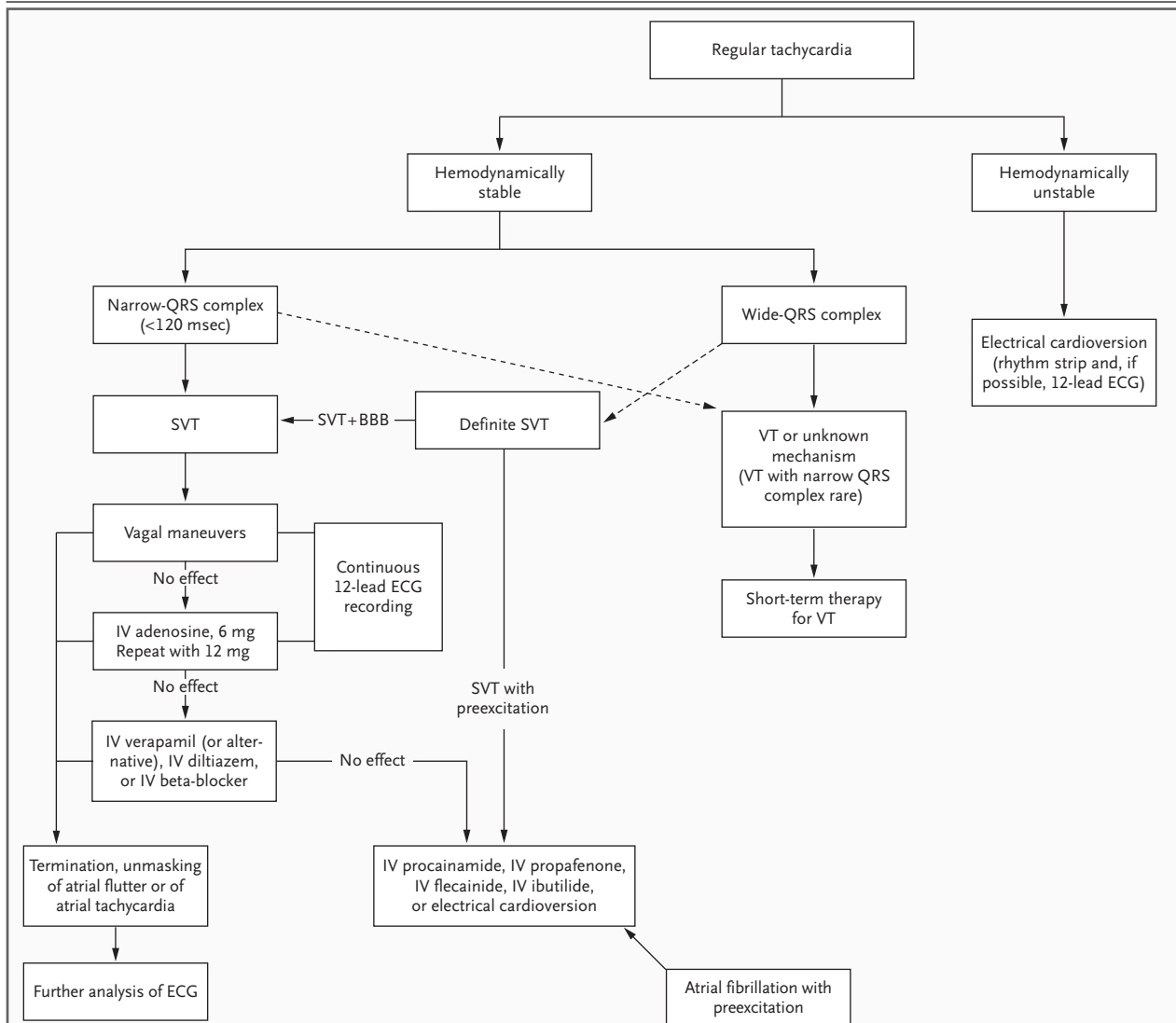
Figure 3. Algorithm for the Short-Term Management of Supraventricular Tachycardia (SVT).

If the diagnosis of SVT with aberration or SVT with preexcitation is not certain, tachycardia with a wide QRS complex must be considered as an unknown mechanism and treated as such. SVT with preexcitation can be the result of either antidromic atrioventricular reentry or, uncommonly, another type of SVT (e.g., atrial tachycardia) with an accessory pathway that is not critical for the maintenance of the arrhythmia. BBB denotes bundle-branch block, VT ventricular tachycardia, IV intravenous, and ECG electrocardiogram. Adapted from Blomstrom-Lundqvist et al.[8]

term therapy. For patients with recurrent episodes, options for long-term treatment include medication and ablation therapy. However, not all patients with recurrent supraventricular tachycardia need treatment. The severity of the symptoms and patient preferences should be considered in decision making. Referral to an electrophysiologist is warranted for the conditions listed in Table 2 and should be considered in other cases to assist in decisions regarding therapy.[17,18]

Figure 4 shows a decision algorithm for the long-term care of patients with supraventricular tachycardia. In cases in which the precise mechanism of tachycardia is uncertain, management is based on the presence or absence of preexcitation on the baseline ECG (Table 3 and Fig. 4).

PHARMACOLOGIC THERAPY

Patients with recurrent episodes of supraventricular tachycardia without preexcitation may be

Table 3. Pharmacologic Agents for Short-Term Treatment of Supraventricular Tachycardia (SVT).*

Drug	Usual Intravenous Dose	Major Side Effects	Cautions, Contraindications
Regular tachycardia with narrow-QRS complex			
First-line agents			
Adenosine†	6 mg rapid bolus followed by fluid bolus; if no response within 1–2 min, administer 12 mg (half-life of less than 5 sec, no risk of accumulation)	Facial flushing, chest pain, and hypotension common; asystole, lasting a few seconds, possible Bronchospasm, atrial fibrillation (in patients with Wolff–Parkinson–White syndrome, can degenerate into ventricular fibrillation), nonsustained ventricular tachycardia uncommon	Contraindicated in heart-transplant recipients because of risk of prolonged asystole (supersensitive response); must be used cautiously in patients with severe reactive airway disease
Verapamil	5 mg every 3–5 min, to maximum 15 mg	Hypotension, heart block, negative inotropic effect	
Alternative agents			
Diltiazem	0.25 mg/kg of body weight over a 2-min period; if no response, additional dose of 0.35 mg/kg over a 2-min period; maintenance infusion of 5–15 mg/hr	Hypotension, heart block, negative inotropic effect	
Beta-blockers		Hypotension, heart block, bradycardia, bronchospasm, negative inotropic effect	Asthma
Metoprolol	5 mg over a 2-min period; up to 3 doses in 15 min		
Esmolol	250–500 μg/kg over a 1-min period; can be followed by a 4-min maintenance infusion of 50–200 μg/kg/min; short half-life of 8 min		Because of short half-life, preferred for use in patients at risk for complications associated with beta-blockers
Propranolol	0.15 mg/kg over a 2-min period		Careful monitoring warranted; dose not to exceed 1 mg/min, to avoid lowering blood pressure and causing severe bradycardia
SVT and atrial fibrillation with preexcitation and SVT refractory to drugs listed above			
Procainamide	30 mg/min continuous infusion to a maximal dose of 17 mg/kg (maintenance infusion of 2–4 mg/min)	Hypotension, widening of QRS complex, torsades de pointes	Infusion should be stopped if arrhythmia suppressed or hypotension or ≥50% widening of QRS complex occurs
Flecainide	2 mg/kg over a 10-min period	Negative inotropic effect, rapidly conducting atrial flutter, widening of QRS complex	
Propafenone	2 mg/kg over a 10-min period		
Ibutilide	If ≥60 kg: 1 mg over a 10-min period; If <60 kg: 0.01 mg/kg over a 10-min period; Repeat once if no response after 10 additional min	Prolongation of QT interval, torsades de pointes	Contraindicated in patients with hypokalemia; careful monitoring required for at least 4 hr after administration

* These agents have been tested in randomized trials. Electrocardiographic monitoring and blood-pressure monitoring are required during treatment. Emergency-resuscitation equipment should always be available. If the diagnosis is certain, SVT with bundle-branch block may be treated as tachycardia with a narrow QRS complex.
† Adenosine is administered through rapid intravenous injection over a period of one to two seconds at a peripheral site, followed by an infusion of 0.9 percent saline.
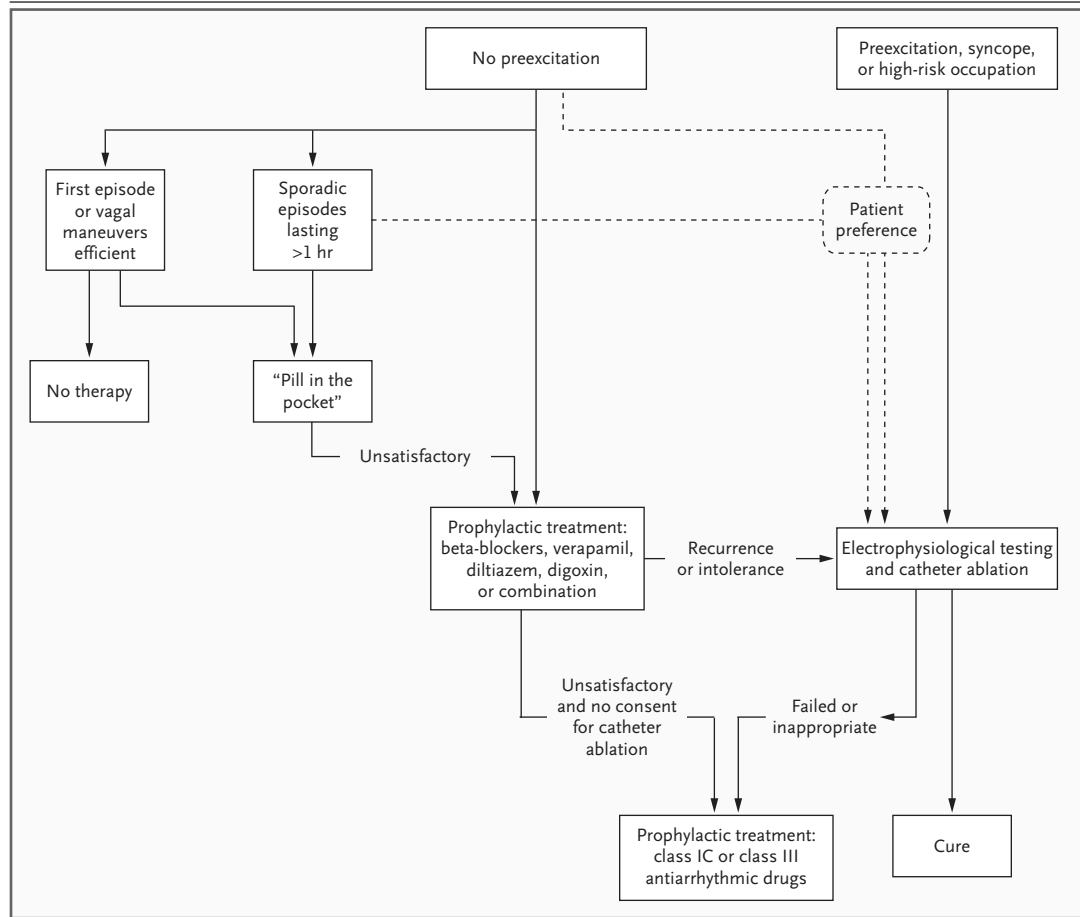
Figure 4. Algorithm for the Long-Term Management of Supraventricular Tachycardia.

In many circumstances, patient preference is an important consideration in the selection of therapy. Referral to an electrophysiologist should be considered for discussion of the risks and benefits of catheter ablation.

treated with prophylactic antiarrhythmic agents. Patients with atrioventricular nodal reentrant tachycardia and atrioventricular reentrant tachycardia mediated by a concealed accessory pathway should primarily receive atrioventricular-node–blocking agents such as verapamil, beta-blockers, or digoxin. Clinical experience indicates that these agents decrease the frequency of the episodes and the severity of symptoms in an estimated 30 to 60 percent of patients, but complete suppression of supraventricular tachycardia is uncommon.[19] A randomized, double-blind trial in which verapamil, propranolol, and digoxin were compared failed to demonstrate the superiority of any one drug over the others.[19] If treatment with the above-mentioned agents proves unsatisfactory, pharmacologic options include a combination of two atrioventricular node-blocking agents or a class IC or class III antiarrhythmic drug (e.g., propafe-

none, sotalol, or amiodarone). In randomized, placebo-controlled trials, class IC and class III antiarrhythmic drugs have prevented the recurrence of supraventricular tachycardia in up to 80 percent of patients over a 60-day period of follow-up; these agents also appear to be more effective in preventing supraventricular tachycardia than are the atrioventricular-node–blocking drugs, although data from comparative trials are lacking.[20,21] Despite the apparent safety of class IC antiarrhythmic drugs in patients with supraventricular tachycardia,[22] long-term therapy with these drugs is generally not recommended because of their potential adverse effects (Table 4); catheter ablation is usually preferred if the patient agrees to this approach.

The pharmacologic management of atrial tachycardias has not been well evaluated in controlled trials. Depending on the mechanism causing the

Table 4. Pharmacologic Agents for Prophylactic Treatment of Supraventricular Tachycardia (SVT).*

Drug	Usual Maintenance Dose	Major Side Effects	Cautions, Contraindications
SVT without preexcitation			
Beta-blockers†		Hypotension, heart block, bradycardia	Asthma, congestive heart failure
Metoprolol	50–200 mg daily		
Bisoprolol	2.5–10 mg daily		
Atenolol	50–100 mg daily		
Propranolol‡§	80–240 mg daily		
Calcium-channel blockers		Hypotension, heart block, negative inotropic effect	Congestive heart failure
Diltiazem‡	180–360 mg daily		
Verapamil‡	120–480 mg daily	Interaction with digoxin, constipation	
Digoxin	0.125–0.375 mg daily	Toxic effects of digitalis, bradycardia	Serum levels should be monitored
SVT with preexcitation and SVT refractory to atrioventricular-node–blocking agents			
First-line agents			
Class IC drugs		Ventricular tachycardia, enhanced atrioventricular nodal conduction, negative inotropic effect	Ischemic and structural heart disease
Flecainide	100–300 mg daily	In addition to above-mentioned side effects of class IC drugs, interaction with digoxin	
Propafenone‡	450–900 mg daily		Drug accumulation in 5–10% of patients with cytochrome P-450 2D6 deficiency
Alternative agents			
Amiodarone	200 mg daily	Skin discoloration, hypothyroidism or hyperthyroidism, gastrointestinal upset, hepatotoxic effects, corneal deposits, tremor, optic neuropathy, pulmonary toxicity	Interaction with oral anticoagulants
Sotalol	160–320 mg daily	Hypotension, heart block, bradycardia, torsades de pointes (latter is dose-dependent; increased risk in women, in patients with left ventricular hypertrophy, and in those with low potassium plasma levels)	Asthma, congestive heart failure; dose reduction in elderly patients and those with renal failure; most studied and used antiarrhythmic drug during pregnancy (class B)

* The maintenance dose should be titrated.
† Many beta-blockers have been studied as antiarrhythmic agents; those listed are selected examples. Beta-blockers with intrinsic sympathomimetic activity have no clear advantages over other beta-blockers in most circumstances; an exception is the concomitant presence of the sick sinus syndrome.
‡ A sustained-release preparation is available.
§ Propranolol is noncardioselective.

arrhythmia, beta-blockers, calcium-channel blockers, and class I or class III antiarrhythmic drugs may reduce or eliminate symptoms.

"Pill-in-the-Pocket" Approach
For patients with infrequent (i.e., no more than a few per year) but prolonged (i.e., lasting more than one to two hours) episodes of supraventricular tachycardia that are well tolerated hemodynamically, or for patients who have had only a single episode of supraventricular tachycardia, another option is to prescribe single-dose pharmacologic therapy (the "pill in the pocket") to be taken when needed for an arrhythmic event. Drugs administered in this fashion include calcium-channel blockers (e.g., 40 to 160 mg of verapamil), exclusively for patients without preexcitation; various beta-blockers; flecainide (100 to 300 mg); and propafenone (150 to 450 mg). In one study, 80 percent of episodes of supraventricular tachycardia were in-

terrupted within two hours with a combination of diltiazem and propanolol or with flecainide.[23]

Supraventricular Tachycardia with the Wolff–Parkinson–White Syndrome

Verapamil and digoxin are contraindicated in patients with the Wolff–Parkinson–White syndrome, unless the accessory pathway has been shown to have a long refractory period (300 msec or more), because these drugs may increase the risk of rapid ventricular response, causing ventricular fibrillation in patients with atrial fibrillation.[24,25] Although catheter ablation is considered the treatment of choice for these patients, both flecainide and propafenone are effective and have been approved by the Food and Drug Administration for the prevention of paroxysmal supraventricular tachycardias mediated by an accessory pathway (with or without antegrade conduction).[26-28]

Catheter Ablation

Since the early 1990s, catheter ablation (Fig. 5) has increasingly been used in the management of supraventricular tachycardia on the basis of its observed efficacy and overall safety when performed at centers with experienced clinicians. Observational studies of catheter ablation of tachycardia mediated by an accessory pathway indicate that success rates exceed 95 percent and recurrence rates are less than 5 percent during the first few months after the procedure is performed. Late recurrences are the exception.[29-31] In cases in which the accessory pathway is close to a His bundle, the application of radiofrequency current can be complicated by atrioventricular block that requires pacemaker therapy. Data from observational studies suggest that in this situation, the use of cryothermal ablation is similarly effective and reduces the potential for atrioventricular block, although studies directly comparing these approaches are lacking.[32] Other complications associated with accessory-pathway ablation, occurring in less than 2 to 3 percent of patients, include damage to an artery, bleeding, arteriovenous fistula, venous thrombosis, pulmonary embolism, myocardial perforation, valvular damage, systemic embolism (in the case of a left-sided accessory pathway), and rarely, death.[33,34]

In patients with atrioventricular nodal reentrant tachycardia, the atrioventricular nodal slow pathway is targeted by catheter ablation in the posteroseptal region of the tricuspid annulus.[35] Success rates are higher than 95 percent.[34,36] Serious complications are uncommon but include pulmonary embolism (in up to 0.2 percent of patients) and the development of atrioventricular block requiring pacemaker therapy (in up to 1 percent of patients).[33,34] Tachycardia recurs in 3 to 7 percent of patients.[36]

Catheter ablation of focal atrial tachycardias has slightly lower success rates (about 85 percent) and higher recurrence rates (about 8 percent).[36,37] Procedural risks are slightly increased for the treatment of left atrial tachycardia, which requires a transseptal puncture. For reentrant atrial tachycardias, radiofrequency ablation has high success rates and is often used as first-line therapy.[37-39]

AREAS OF UNCERTAINTY

Limited data suggest that, as compared with antiarrhythmic therapy, catheter ablation improves the quality of life and is more cost effective in the long term.[40,41] However, there is a lack of large randomized trials with prolonged follow-up to guide the choice between radiofrequency ablation and medical therapy.

The appropriate treatment strategy for patients with asymptomatic preexcitation syndromes is controversial.[42-44] The incidence of sudden death due to rapid conduction of atrial fibrillation that leads to ventricular fibrillation is estimated at between 0.15 and 0.45 percent per patient-year.[45-47] Attempts to stratify the risk according to the use of noninvasive methods or invasive measurements of the refractory period of the accessory pathway have been advocated but may be misleading.[17,44,48] A task force of the American College of Cardiology, the American Heart Association, and the European Society of Cardiology concluded that the positive predictive value of invasive electrophysiologic testing is too low to justify its routine use in asymptomatic patients and that the decision to ablate accessory pathways in persons with high-risk occupations or those who engage in high-risk recreational activities should be made on an individual basis.[8]

GUIDELINES

Comprehensive guidelines for the management of supraventricular tachycardia were published by an expert committee of the American College of Cardiology, the American Heart Association, and the
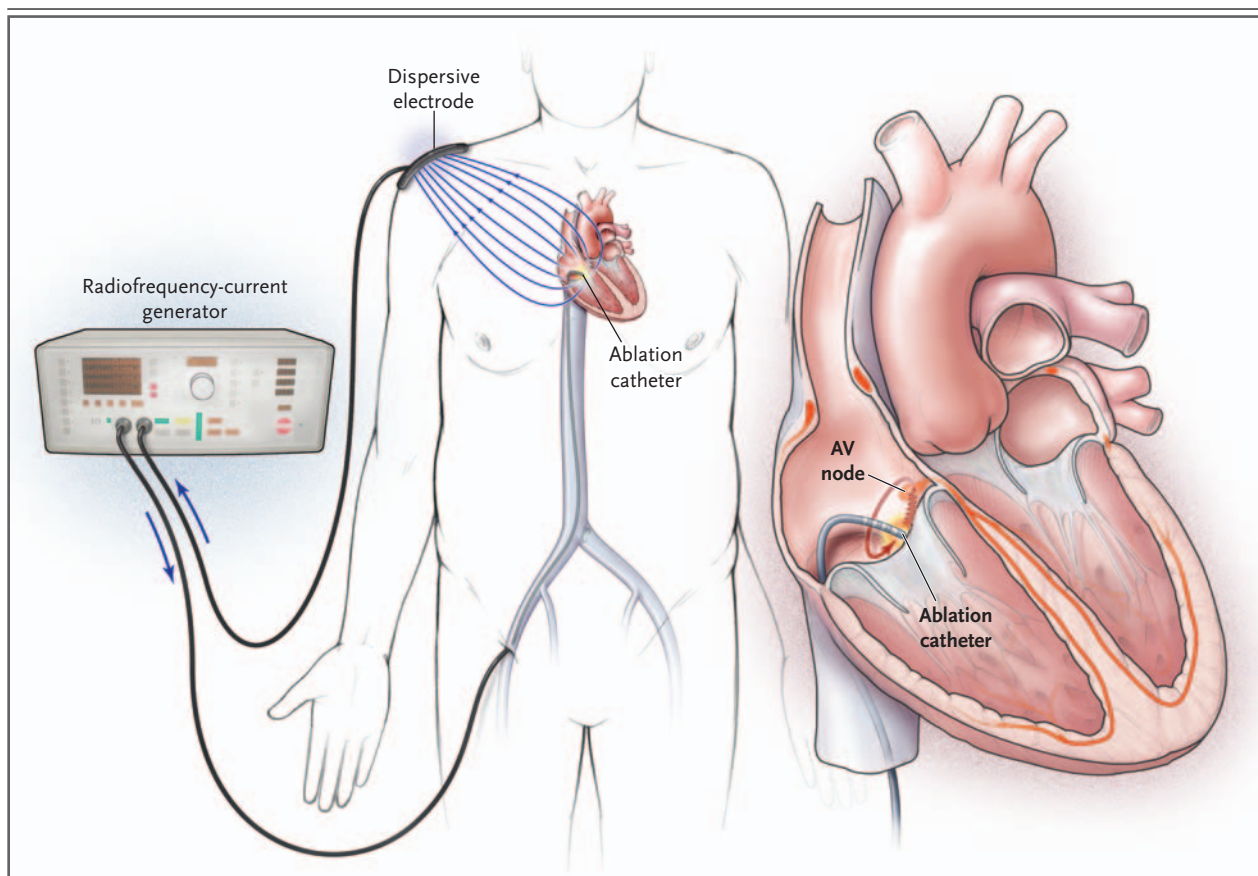
Figure 5. Catheter Ablation of Cardiac Arrhythmias.

One to four catheter electrodes are introduced into the cavities of the heart through femoral (or, alternatively, internal jugular or sub-clavian) venous access after local anesthesia is administered. Radiofrequency current — a low-voltage, high-frequency (500 kHz) form of electrical energy used for electrocautery in surgery — is delivered through a catheter electrode to create small lesions through ther-mal injury in the myocardial tissue, the conduction system, or both, which have been identified as critical for mediating the cardiac ar-rhythmia. In patients with arrhythmias mediated by an abnormal accessory pathway, the catheter is positioned so that it is in contact with the pathway, and the application of radiofrequency current blocks conduction over the accessory pathway within a few seconds. For left-sided accessory pathways, a retrograde approach through the femoral artery and the aortic valve can be used. Alternatively, a trans-septal puncture can be performed to gain access to the left atrium. Cryothermal ablation is an effective approach in patients with atrio-ventricular (AV) nodal reentrant tachycardia or an accessory pathway close to a His bundle because of the reversibility of the initial effect and the negligible risk of AV block. Most ablation procedures take one to three hours. Catheter ablation of supraventricular tachycardia can be performed as a one-day outpatient procedure, or it may require overnight hospitalization. Treatment with aspirin is often recom-mended for several weeks after ablation that has been performed in the left side of the heart to reduce the potential risk of emboli. Patients need no special follow-up after the intervention.

European Society of Cardiology.[8] Doses of anti-arrhythmic drugs and their adverse effects are discussed in these societies' guidelines for the management of patients with atrial fibrillation.[49] The recommendations in this review are in gen-eral agreement with these guidelines. Generally, radiofrequency ablation is recommended as pri-mary therapy for patients in whom the preexcita-tion syndrome or hemodynamic instability occurs during their arrhythmias. In other cases, patient preference is an important consideration in the selection of therapy.

CONCLUSIONS
AND RECOMMENDATIONS

For a patient such as the one described in the vi-gnette, I would first try carotid sinus pressure or other vagal maneuvers, followed by intravenous adenosine if the maneuvers are ineffective.

For patients in whom supraventricular tachycardia recurs, preventive therapy is generally warranted if there are frequent, prolonged, or highly symptomatic episodes that cannot easily be terminated by the patient's use of vagal maneuvers. If the tachycardia is associated with preexcitation or syncope, electrophysiological evaluation is warranted. In the absence of preexcitation or syncope, atrioventricular-node–blocking agents are usually recommended as first-line treatment, even though there is a lack of data from large trials to compare these drugs with other approaches to management. However, many patients may have adverse effects or find it inconvenient to take medication over the long term. For patients in whom recurrences are infrequent but prolonged, pill-in-the-pocket treatment (e.g., 100 to 200 mg of flecainide) at the onset of supraventricular tachycardia is a reasonable approach. Catheter ablation, when performed at a center with experienced clinicians, is appropriate for supraventricular tachycardia associated with preexcitation or hemodynamic instability or if antiarrhythmic drugs are not effective or are poorly tolerated. Catheter ablation may also be used as primary therapy in other cases if the patients, informed of the risks and benefits, prefer this approach.

REFERENCES

1. Orejarena LA, Vidaillet H Jr, DeStefano F, et al. Paroxysmal supraventricular tachycardia in the general population. J Am Coll Cardiol 1998;31:150-7.
2. Tikkanen I, Metsarinne K, Fyhrquist F. Atrial natriuretic peptide in paroxysmal supraventricular tachycardia. Lancet 1985; 2:40-1.
3. Wellens HJ. 25 Years of insights into the mechanisms of supraventricular arrhythmias. Pacing Clin Electrophysiol 2003; 26:1916-22.
4. Chen SA, Chiang CE, Yang CJ, et al. Sustained atrial tachycardia in adult patients: electrophysiological characteristics, pharmacological response, possible mechanisms, and effects of radiofrequency ablation. Circulation 1994;90:1262-78.
5. Cosio FG, Martin-Penato A, Pastor A, Nunez A, Goicolea A. Atypical flutter: a review. Pacing Clin Electrophysiol 2003;26: 2157-69.
6. Lessmeier TJ, Gamperling D, Johnson-Liddon V, et al. Unrecognized paroxysmal supraventricular tachycardia: potential for misdiagnosis as panic disorder. Arch Intern Med 1997;157:537-43.
7. Gursoy S, Steurer G, Brugada J, Andries E, Brugada P. The hemodynamic mechanism of pounding in the neck in atrioventricular nodal reentrant tachycardia. N Engl J Med 1992;327:772-4.
8. Blomstrom-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias — executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for

the Management of Patients with Supraventricular Arrhythmias). Circulation 2003; 108:1871-909.
9. Josephson ME, Wellens HJ. Differential diagnosis of supraventricular tachycardia. Cardiol Clin 1990;8:411-42.
10. DiMarco JP, Miles W, Akhtar M, et al. Adenosine for paroxysmal supraventricular tachycardia: dose ranging and comparison with verapamil: assessment in placebo-controlled, multicenter trials. Ann Intern Med 1990;113:104-10. [Erratum, Ann Intern Med 1990;113:996.]
11. Glatter KA, Cheng J, Dorostkar P, et al. Electrophysiologic effects of adenosine in patients with supraventricular tachycardia. Circulation 1999;99:1034-40.
12. Kall JG, Kopp D, Olshansky B, et al. Adenosine-sensitive atrial tachycardia. Pacing Clin Electrophysiol 1995;18:300-6.
13. Rinkenberger RL, Prystowsky EN, Heger JJ, Troup PJ, Jackman WM, Zipes DP. Effects of intravenous and chronic oral verapamil administration in patients with supraventricular tachyarrhythmias. Circulation 1980;62:996-1010.
14. Anderson S, Blanski L, Byrd RC, et al. Comparison of the efficacy and safety of esmolol, a short-acting beta blocker, with placebo in the treatment of supraventricular tachyarrhythmias. Am Heart J 1986; 111:42-8.
15. Glatter KA, Dorostkar PC, Yang Y, et al. Electrophysiological effects of ibutilide in patients with accessory pathways. Circulation 2001;104:1933-9.
16. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff–Parkinson–White syndrome. N Engl J Med 1979;301:1080-5.
17. Schilling RJ. Which patient should be

referred to an electrophysiologist: supraventricular tachycardia. Heart 2002;87:299-304.
18. Epstein LM, Chiesa N, Wong MN, et al. Radiofrequency catheter ablation in the treatment of supraventricular tachycardia in the elderly. J Am Coll Cardiol 1994;23: 1356-62.
19. Winniford MD, Fulton KL, Hillis LD. Long-term therapy of paroxysmal supraventricular tachycardia: a randomized, double-blind comparison of digoxin, propranolol and verapamil. Am J Cardiol 1984;54:1138-9.
20. Henthorn RW, Waldo AL, Anderson JL, et al. Flecainide acetate prevents recurrence of symptomatic paroxysmal supraventricular tachycardia. Circulation 1991; 83:119-25.
21. Pritchett EL, McCarthy EA, Wilkinson WE. Propafenone treatment of symptomatic paroxysmal supraventricular arrhythmias: a randomized, placebo-controlled, crossover trial in patients tolerating oral therapy. Ann Intern Med 1991;114:539-44.
22. Hellestrand KJ. Efficacy and safety of long-term oral flecainide acetate in patients with responsive supraventricular tachycardia. Am J Cardiol 1996;77:83A-88A.
23. Alboni P, Tomasi C, Menozzi C, et al. Efficacy and safety of out-of-hospital self-administered single-dose oral drug treatment in the management of infrequent, well-tolerated paroxysmal supraventricular tachycardia. J Am Coll Cardiol 2001;37: 548-53.
24. Sellers TD Jr, Bashore TM, Gallagher JJ. Digitalis in the pre-excitation syndrome: analysis during atrial fibrillation. Circulation 1977;56:260-7.
25. Glatter K, Yang Y, Chatterjee K, et al.

Chemical cardioversion of atrial fibrillation or flutter with ibutilide in patients receiving amiodarone therapy. Circulation 2001;103:253-7.

26. Ludmer PL, McGowan NE, Antman EM, Friedman PL. Efficacy of propafenone in Wolff-Parkinson-White syndrome: electrophysiologic findings and long-term follow-up. J Am Coll Cardiol 1987;9:1357-63.

27. Kim SS, Lal R, Ruffy R. Treatment of paroxysmal reentrant supraventricular tachycardia with flecainide acetate. Am J Cardiol 1986;58:80-5.

28. Anderson JL, Platt ML, Guarnieri T, Fox TL, Maser MJ, Pritchett EL. Flecainide acetate for paroxysmal supraventricular tachyarrhythmias. Am J Cardiol 1994;74:578-84.

29. Jackman WM, Wang XZ, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff–Parkinson–White syndrome) by radiofrequency current. N Engl J Med 1991;324:1605-11.

30. Kuck KH, Schluter M, Geiger M, Siebels J, Duckeck W. Radiofrequency current catheter ablation of accessory atrioventricular pathways. Lancet 1991;337:1557-61.

31. Calkins H, Sousa J, el-Atassi R, et al. Diagnosis and cure of the Wolff–Parkinson–White syndrome or paroxysmal supraventricular tachycardias during a single electrophysiologic test. N Engl J Med 1991;324:1612-8.

32. Friedman PL, Dubuc M, Green MS, et al. Catheter cryoablation of supraventricular tachycardia: results of the multicenter prospective "Frosty" trial. Heart Rhythm 2004;1:129-38.

33. Ganz LI, Friedman PL. Supraventricular tachycardia. N Engl J Med 1995;332:162-73.

34. Scheinman MM, Huang S. The 1998 NASPE prospective catheter ablation registry. Pacing Clin Electrophysiol 2000;23:1020-8.

35. Jackman WM, Beckman KJ, McClelland JH, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of slow-pathway conduction. N Engl J Med 1992;327:313-8.

36. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Role of radiofrequency ablation in the management of supraventricular arrhythmias: experience in 760 consecutive patients. J Cardiovasc Electrophysiol 1993;4:371-89.

37. Lesh MD, Van Hare GF, Epstein LM, et al. Radiofrequency catheter ablation of atrial arrhythmias: results and mechanisms. Circulation 1994;89:1074-89.

38. Natale A, Newby KH, Pisano E, et al. Prospective randomized comparison of antiarrhythmic therapy versus first-line radiofrequency ablation in patients with atrial flutter. J Am Coll Cardiol 2000;35:1898-904.

39. Cosio FG, Lopez-Gil M, Goicolea A, Arribas F, Barroso JL. Radiofrequency ablation of the inferior vena cava-tricuspid valve isthmus in common atrial flutter. Am J Cardiol 1993;71:705-9.

40. Bathina MN, Mickelsen S, Brooks C, Jaramillo J, Hepton T, Kusumoto FM. Radiofrequency catheter ablation versus medical therapy for initial treatment of supraventricular tachycardia and its impact on quality of life and healthcare costs. Am J Cardiol 1998;82:589-93.

41. Lau CP, Tai YT, Lee PW. The effects of radiofrequency ablation versus medical therapy on the quality-of-life and exercise capacity in patients with accessory pathway-mediated supraventricular tachycardia: a treatment comparison study. Pacing Clin Electrophysiol 1995;18:424-32.

42. Klein GJ, Prystowsky EN, Yee R, Sharma AD, Laupacis A. Asymptomatic Wolff-Parkinson-White: should we intervene? Circulation 1989;80:1902-5.

43. Lerman BB, Basson CT. High-risk patients with ventricular preexcitation — a pendulum in motion. N Engl J Med 2003;349:1787-9.

44. Todd DM, Klein GJ, Krahn AD, Skanes AC, Yee R. Asymptomatic Wolff-Parkinson-White syndrome: is it time to revisit guidelines? J Am Coll Cardiol 2003;41:245-8.

45. Munger TM, Packer DL, Hammill SC, et al. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989. Circulation 1993;87:866-73.

46. Pappone C, Santinelli V, Rosanio S, et al. Usefulness of invasive electrophysiologic testing to stratify the risk of arrhythmic events in asymptomatic patients with Wolff-Parkinson-White pattern: results from a large prospective long-term follow-up study. J Am Coll Cardiol 2003;41:239-44.

47. Fitzsimmons PJ, McWhirter PD, Peterson DW, Kruyer WB. The natural history of Wolff-Parkinson-White syndrome in 228 military aviators: a long-term follow-up of 22 years. Am Heart J 2001;142:530-6.

48. Boahene KA, Klein GJ, Sharma AD, Yee R, Fujimura O. Value of a revised procainamide test in the Wolff-Parkinson-White syndrome. Am J Cardiol 1990;65:195-200.

49. Fuster V, Ryden LE, Asinger RW, et al. ACC/AHA/ESC guidelines for the management of patients with atrial fibrillation: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines and Policy Conferences (Committee to Develop Guidelines for the Management of Patients with Atrial Fibrillation) developed in collaboration with the North American Society of Pacing and Electrophysiology. Circulation 2001;104:2118-50.

Copyright © 2006 Massachusetts Medical Society.

COLLECTIONS OF ARTICLES ON THE *JOURNAL*'S WEB SITE
The *Journal*'s Web site (**www.nejm.org**) sorts published articles into more than 50 distinct clinical collections, which can be used as convenient entry points to clinical content. In each collection, articles are cited in reverse chronologic order, with the most recent first.