Management of Cardiac Arrhythmias

With many types of cardiac arrhythmias presenting in the clinical setting, family physicians must be able to distinguish each in order to refer dangerous cases to a specialist.

By Anne M. Gillis, MD, FRCPC

Many different types of cardiac arrhythmias are encountered in clinical practice, ranging from benign premature beats to sustained, potentially life-threatening ventricular tachyarrhythmias. Treatment is dependent on the magnitude of symptoms and the potential risk of significant morbidity or mortality.

This review article will offer a practical approach to the management of the most common cardiac arrhythmias encountered in clinical practice.

Atrial Fibrillation/Atrial Flutter

Atrial fibrillation (AF) is the most common sustained arrhythmia encountered in clinical practice. The initial approach to treating AF is illustrated in Figure 1.1 The goals of therapy are:

1. Maintain sinus rhythm using Class I or Class III antiarrhythmic drugs, or achieve adequate ventricular rate control using Class II or IV antiarrhythmic drugs;1,2 and
2. Prevent thromboembolic events.

Currently, there is no data to support the superiority of the maintenance of sinus rhythm...
approach over the heart-rate control approach. Therapy, therefore, should be individualized. The initial approach is to control the ventricular rate by using intravenous beta blockers or calcium channel blockers (Figure 2). If the patient is hemodynamically compromised, urgent synchronized cardioversion may be required. Once heart-rate control has been achieved, and if the patient has been in AF for less than 48 hours, or has been on chronic anticoagulant therapy, Class I/III antiarrhythmic drug therapy can be initiated with the intent of achieving a pharmacologic conversion to sinus rhythm.

Ibutilide administered intravenously may rapidly terminate AF. There is a risk of ventricular proarrhythmia (torsade de pointes VT) and patients should be monitored for four hours following drug administration, even if they convert to sinus rhythm. A loading dose of propafenone or flecainide also may promote early pharmacologic conversion. The loading and maintenance doses of commonly used Class I/III drugs are summarized in Table 1.

Sotalol is less effective at promoting pharmacologic conversion compared to propafenone or flecainide. Although the Class III drug dofetilide has been shown to be safe and efficacious for the management of AF in patients with heart failure, it is not yet approved in Canada and its use is restricted to cardiac electrophysiologists.

Chronic prophylactic antiarrhythmic drug therapy may be required for patients with frequent paroxysmal AF, or patients with recurrent persistent AF requiring cardioversion. A heart-rate controlling drug must be continued since Class I drugs may convert AF to atrial flutter with a slower atrial rate, which allows 1:1 conduction via the AV node at rates up to 220 beats per minute.
Cardiac Arrhythmias

Individualize heart rate control and anti-thrombotic therapy

Restore and maintain sinus rhythm?

**YES**
- No heart disease
  - Class III AAD
  - Electrical cardioversion (if necessary)

**NO**
- LV dysfunction
  - Amiodarone
  - Caution with Class I drugs or sotalol

Continue anti-thrombotics + initiate drugs to control heart rate

Figure 1. Management of atrial fibrillation/flutter.

**Patient unstable**
- Electrical cardioversion 100 to 400 J, repeat if necessary

**Patient stable**
- Achieve heart rate control via:
  - Metoprolol 5mg to 15 mg IV infusion in 5 mg boluses q 5 min
  - Verapamil 5mg to 20 mg IV push in 5 mg boluses q 5 min
  - Diltiazem 0.25 mg/kg IV infusion over 15 min, then 0.35 mg/kg IV prn
  - Procainamide 10 mg to 15 mg/kg at 25 mg/min IV infusion, then 2mg to 4 mg/min (WPW)
  - Digoxin 0.5-0.75 mg IV infusion over 30 min, then 0.75 mg in divided doses over next 24 hr

Figure 2. Acute therapy of atrial fibrillation/flutter.
If the patient has been in AF for longer than 48 hours, anticoagulation should be commenced and maintained for at least four weeks prior to planned elective cardioversion or initiation of pharmacologic therapy to restore sinus rhythm. Alternatively, if a transesophageal echocardiogram (ECG) shows no cardiac thrombus, efforts to restore sinus rhythm can be started immediately. The indications for long-term anticoagulation and choice of antithrombotic agent in patients with AF are summarized in Table 2.

### Catheter Ablation for AF
Many patients with AF will have recurrence of AF despite antiarrhythmic drug therapy. Furthermore, many patients do not tolerate drug therapy over the long term. Implantation of a pacemaker followed by radiofrequency catheter ablation of the AV junction is a very effective treatment option for such patients. Antiarrhythmic drugs can then be discontinued, but the majority of patients will develop chronic AF and will need chronic anticoagulation therapy.

Catheter ablation for the cure of AF is undergoing clinical investigation. Isolation and ablation of foci in one or more pulmonary veins that initiate AF has been reported to prevent AF.
However, long-term success rates are unknown and there are risks of thromboembolism and pulmonary vein stenosis. This approach currently is reserved for select patients.

**Supraventricular Tachycardia**

Supraventricular tachycardia (SVT) is most commonly due to AV node re-entry or re-entry involving an accessory AV connection. An approach to the acute management of SVT is summarized in Table 3.1 If vagal maneuvers are ineffective, adenosine often is the first-line drug used due to its extremely short half-life.

Metoprolol or verapamil given intravenously are also very effective therapies. If the ECG shows a wide complex tachycardia (QRS > 100 ms), this should raise suspicion of Wolff Parkinson White syndrome.

Adenosine, verapamil and digoxin have been reported to precipitate VF when SVT or AF conducts antegradely over the accessory connection.7 In this setting, the safest approach is to administer procainamide or proceed to synchronized cardioversion.

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**Table 3**

**Acute Therapy For Hemodynamically Stable Supraventricular Tachycardia**

<table>
<thead>
<tr>
<th>QRS ≤ 0.1s or patient known to have bundle branch block</th>
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<tr>
<td>• Vagal maneuvers (e.g., Valsalva maneuver, carotid sinus massage, orbital pressure, diving reflex)</td>
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<tr>
<td><strong>AND</strong></td>
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<tr>
<td>• Adenosine 6 mg to 12 mg IV push (max 24 mg)</td>
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<tr>
<td><strong>OR</strong></td>
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<tr>
<td>• Metoprolol 5 mg to 15 mg IV infusion in 5 mg increments q 5 min.</td>
</tr>
<tr>
<td><strong>OR</strong></td>
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<tr>
<td>• Verapamil 5 mg to 20 mg IV push in 5 mg boluses q 5 min.</td>
</tr>
<tr>
<td><strong>OR</strong></td>
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<tr>
<td>• Diltiazem 0.25 mg/kg IV infusion over 15 min.; then increase to 0.35 mg/kg if necessary</td>
</tr>
<tr>
<td><strong>OR</strong></td>
</tr>
<tr>
<td>• Digoxin 0.5 mg to 0.75 mg IV infusion over 15-30 min., followed by additional 0.25 mg to 0.75 mg over next 24 hr.</td>
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<tr>
<th>QRS &gt; 0.1s and no previous electrocardiogram or patient known to have QRS &lt; 0.1s</th>
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<tr>
<td>• Suspect Wolff Parkinson White syndrome: Procainamide 15 mg/kg in 25 mg/min. infusion; then maintenance dose 2-4 mg/min.</td>
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<tr>
<td><strong>OR</strong></td>
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<tr>
<td>• Electrical cardioversion</td>
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Chronic prophylactic antiarrhythmic therapy for SVT is based on the frequency and duration of episodes, as well as on associated symptoms. Beta blockers or calcium channel blockers are frequently effective in preventing SVT, but may not be well tolerated over the long term. The dose ranges of commonly used drugs are summarized in Table 4.

Sotalol has Class III antiarrhythmic drug effects in addition to beta-blocking actions and, thus, prolongs the QT interval. This drug should be used cautiously, particularly in female patients with cardiac hypertrophy or ventricular dysfunction, in patients on diuretics or in patients with renal insufficiency. An ECG should be obtained at baseline and after each dosage change. If the QT interval is greater than 500 ms, sotalol should be discontinued or the dosage reduced.

Catheter Ablation for SVT

Many patients elect to undergo radiofrequency catheter ablation of the electrophysiologic substrate contributing to SVT. This procedure involves: the insertion of several electrode catheters into the heart; the induction of SVT-programmed stimulation of the atrium and/or ventricle; the identification of the arrhythmia mechanism; and mapping and radiofrequency ablation of the substrate for SVT. Success rates are 99% for AV node re-entrant tachycardia, 95% for accessory pathways, and between 80% and 90% for true atrial tachycardias or atrial flutter. The risk of the procedure is in the range of 1% to 2% and includes vascular injury/hemorrhage, thromboembolism, cardiac perforation/tamponade or complete heart block, requiring a permanent pacemaker insertion.

Premature Beats in the Absence of Structural Heart Disease

Most individuals experience some SVPBs or VPBs, with the frequency increasing with age. Some patients are asymptomatic whereas others will experience symptoms, such as a “flip-flopping” sensation, pounding in the chest or neck, light-headedness or awareness of slow or irregular beats. In the absence of structural heart disease, atrial premature beats or VPBs are benign and no specific treatment is required. The patient should be reassured that they are not at risk for a serious cardiac event and be encouraged to continue normal activities.

If specific triggers exist, such as excessive caffeine or alcohol consumption, they should be eliminated. If patients are extremely symptomatic, beta blockers or calcium channel antagonists (i.e., verapamil, diltiazem) may effectively suppress premature beats. Because of the poten-
tial risk of ventricular proarrhythmia, however, Class I/III antiarrhythmic drugs, including sotalol, are contraindicated. In the absence of structural heart disease, referral to a cardiologist is not generally required.

**VPBs and Structural Heart Disease**

VPBs in the setting of structural heart disease are a risk factor for sudden cardiac death. Class I antiarrhythmic drugs (i.e., quinidine, procainamide, disopyramide, propafenone, flecainide) are contraindicated in patients with a prior myocardial infarction (MI) because of the increased risk of ventricular proarrhythmia. By extension, these drugs also are relatively contraindicated in patients with significant left ventricular (LV) dysfunction. Amiodarone has been reported to reduce arrhythmic death in patients with frequent VPBs post-MI. It has not been shown to substantially reduce overall cardiac mortality in this setting.

The potential adverse effects associated with amiodarone, and the lack of a substantial mortality benefit, have limited its use in this population. Currently, the best approach to reducing arrhythmic death in this population is the utilization of beta blockers, angiotensin-converting enzyme (ACE) inhibitors, spironolactone and other therapies shown to prevent ventricular remodeling following MI.

**Nonsustained VT in the Absence of Structural Heart Disease**

Nonsustained VT in the absence of structural heart disease usually originates in the right ventricular outflow tract. This arrhythmia is not associated with an increased risk of sudden death, but individuals may experience palpitations or syncope. This arrhythmia is usually catecholamine-sensitive and can be suppressed with beta blockers. Catheter ablation is successful in eliminating the arrhythmogenic substrate, but should be reserved for highly symptomatic patients.
Figure 3. Management of hemodynamically stable monomorphic VT.

Monomophic VT
- Treat reversible causes
  - Normal LV function
    - Procainamide 20 mg/min (total 17 mg/kg), then 1 mg to 4 mg/min
  - Depressed LV function
    - Amiodarone 150 mg IV bolus, then 1 mg to 2 mg/min over 6 hr, then 0.5 mg to 1 mg/min over 18 hrs
    - OR
      - Lidocaine 0.5 mg to 0.75 mg/kg IV push, then 1 mg to 4 mg/min

Figure 4. Pharmacologic approach to polymorphic VT.

Normal QT interval
- Treat ischemia correct electrolytes
  - Normal LV function
    - Beta blockers (e.g., metoprolol 5 mg IV push)
    - OR
      - Lidocaine
      - OR
      - Amiodarone
  - Depressed LV function
    - Amiodarone
    - OR
      - Lidocaine

QT interval prolongation (torsade de pointes VT)
- Correct electrolytes
  - Magnesium 1 g to 4 g IV
  - OR
    - Overdrive pacing (80 bpm to 100 bpm)
    - OR
      - Isoproterenol (80 bpm to 100 bpm)
      - OR
        - Lidocaine
Nonsustained VT in the Setting of Structural Heart Disease

Nonsustained VT following MI is associated with an increased risk of sudden cardiac death. The Multicentre Unsustained Tachycardia Trial (MUSTT) investigators reported that patients with coronary artery disease, left ventricular (LV) dysfunction and asymptomatic, nonsustained VT in whom SVTs could not be induced during programmed stimulation of the ventricle, had a significantly lower risk of sudden death or cardiac arrest, as well as a lower overall mortality than similar patients with inducible sustained tachyarrhythmias.11

Treatment with an implantable cardioverter defibrillator, but not antiarrhythmic drugs, reduced the risk of sudden death in this population. Accordingly, patients with nonsustained VT in the setting of LV dysfunction and coronary artery disease should be referred to an expert.

Ventricular Tachycardia

Sustained VT or VF are the most common causes of sudden cardiac death and usually occur in the setting of structural heart disease. The American Heart Association recently revised the guidelines for advanced cardiac life support.12 Figures 3 and 4 illustrate the approach to the management of sustained monomorphic VT and sustained polymorphic VT.

The initial approach is aimed at identifying and treating reversible causes, such as ischemia, electrolyte disorders or drugs. Importantly, lidocaine is no longer the first drug of choice. The treatment of polymorphic VT is based on whether the QT interval is normal or prolonged. If the QT is prolonged, the VT is torsade de pointes VT. This condition is usually associated with a reversible cause, such as hypokalemia, hypomagnesemia, severe bradycardia, antiarrhythmic drug therapy or other drugs known to prolong the QT interval (e.g., tricyclic antidepressants, phenothiazines, erythromycin). The treatment for torsade de pointes VT is magnesium and/or overdrive pacing. Class I/III antiarrhythmic drug therapy will exacerbate the condition.

Summary

The long-term management of VT or VF depends on a number of factors. If feasible, revascularization should be performed in
patients with coronary artery disease. The treatment of the underlying heart disease should be optimized, and reversible causes should be identified and treated. All patients with sustained VT or VF should be assessed by a cardiologist. Patients with sustained VT or VF in the absence of reversible causes should be referred to a cardiac electrophysiologist for an expert opinion on management. The majority of these patients will likely receive an implantable cardioverter defibrillator or chronic amiodarone therapy. The choice of therapy is based on a number of factors, including life expectancy, LV function and frequency of arrhythmias.

References