Arrhythmia

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Cardiac arrhythmia involves a group of conditions in which the heartbeat is irregular, too slow, or too fast.

PATHOPHYSIOLOGY

Atrial fibrillation (AF) has extremely rapid (400–600 atrial beats/min) and disorganized atrial activation. There is loss of atrial contraction ,and supraventricular impulses penetrate the atrioventricular (AV) conduction system to variable degrees, resulting in irregular ventricular activation and an irregularly irregular pulse.

Atrial flutter has rapid (270–330 atrial beats/min) but regular atrial activation. Ventricular response usually has a regular pattern and a pulse of 300 beats/min. This arrhythmia is less common than AF but has similar precipitating factors, consequences, and drug therapy approach.

The predominant mechanism of AF and atrial flutter is reentry, which is usually associated with organic heart disease that causes left atrial distention (eg, ischemia or infarction, hypertensive heart disease, and valvular disorders).

Paroxysmal Supraventricular Tachycardia

PSVT arising by reentrant mechanisms includes arrhythmias caused by AV nodal reentry (AVNRT), AV reentrant tachycardia (AVRT) due to an accessory pathway, sinoatrial (SA) nodal reentry, and intraatrial reentry.

Ventricular Arrhythmias

1.Premature Ventricular Complexes

(PVCs) can occur in patients with or without structural heart disease (SHD). PVCs may be elicited by abnormal automaticity, triggered activity, or reentrant mechanisms.

2.Ventricular Proarrhythmia

Proarrhythmia is the development of a significant new arrhythmia, such as VT, (VF), or TdP, or worsening of an existing arrhythmia. Proarrhythmia results from the same mechanisms that cause other arrhythmias or from an alteration in the underlying substrate due to an antiarrhythmic drug (AAD).

Ventricular Fibrillation VF is electrical anarchy of the ventricle resulting in no cardiac output and cardiovascular (CV) collapse. Sudden cardiac death occurs most commonly in patients with CAD or LV dysfunction.

Bradyarrhythmias

Sinus bradyarrhythmias (heart rate <60 beats/min) are common, especially in young, athletically active individuals, and are usually asymptomatic and do not require intervention. However, some patients have sinus node dysfunction (sick sinus syndrome) because of underlying SHD and the normal aging process, which attenuates SA nodal function. Sinus node dysfunction is usually representative of diffuse conduction disease, which may be accompanied by AV block and by paroxysmal tachycardias such as AF. Alternating bradyarrhythmias and tachyarrhythmias are referred to as the tachy–brady syndrome.

AV block or conduction delay may occur in any area of the AV conduction system. AV block may be found in patients without underlying heart disease (eg, trained athletes) or during sleep when vagal tone is high. It may be transient when the underlying etiology is reversible (eg, myocarditis, myocardial ischemia, after CV surgery, or during drug therapy). β-Blockers, digoxin, or nondihydropyridine (non-DHP) calcium channel blockers (CCBs) may cause AV block, primarily in the AV nodal area. Class I AADs may exacerbate conduction delays below the level of the AV node. AV block may be irreversible if the cause is acute MI, rare degenerative diseases, primary myocardial disease, or congenital heart disease

CLINICAL PRESENTATION

- Patients with AF or atrial flutter may complain of rapid heart rate, palpitations, chest pain, dyspnea, dizziness, and fatigue.

- PSVT: Patients may complain of intermittent episodes of rapid heart rate/palpitations that abruptly start and stop, usually without provocation (but sometimes with exercise). Patients may also complain of chest pressure or a neck sensation. Life-threatening symptoms (syncope, hemodynamic collapse) are associated with an extremely rapid heart rate (eg, >200 beats/min)

- PVCs are non–life-threatening and usually asymptomatic. Patients occasionally complain of palpitations or uncomfortable heartbeats

- The symptoms of sustained VT (monomorphic VT or TdP) can range from nearly asymptomatic to pulseless hemodynamic collapse

- VF results in hemodynamic collapse, syncope, and cardiac arrest. Cardiac output and blood pressure are not recordable.

- Some patients who develop proarrhythmia may be asymptomatic, others may notice worsening symptoms, and some may die suddenly.

- Symptoms of bradyarrhythmias generally result from decreased cardiac output and may be associated with hypotension (eg, dizziness, syncope, fatigue, confusion). If LV dysfunction exists, patients may experience worsening HF symptoms. Except for recurrent syncope, symptoms associated with bradyarrhythmias are often subtle and nonspecific.

DIAGNOSIS

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On ECG, AF is an irregularly irregular rhythm with no discernible, consistent atrial activity (P waves). Ventricular rate is usually 90–170 beats/min and the pulse is irregular

The ECG in patients with PSVT commonly shows a rapid, narrow QRS tachycardia (regular in rhythm) that starts and stops abruptly

Proarrhythmia can be difficult to diagnose because of the variable nature of underlying arrhythmias.

TREATMENT

- Goals of Treatment: The desired outcome depends on the underlying arrhythmia. For example, the goals of treating AF or atrial flutter are
- restoring sinus rhythm
- preventing thromboembolic complications
- and preventing further recurrences.

General Approach

- Use of AADs has declined because clinical trials showed increased mortality with their use due to proarrhythmic side effects.
- AADs have been increasingly replaced by nonpharmacologic approaches such as ablation and the implantable cardioverter-defibrillator (ICD).

Antiarrhythmic Drugs

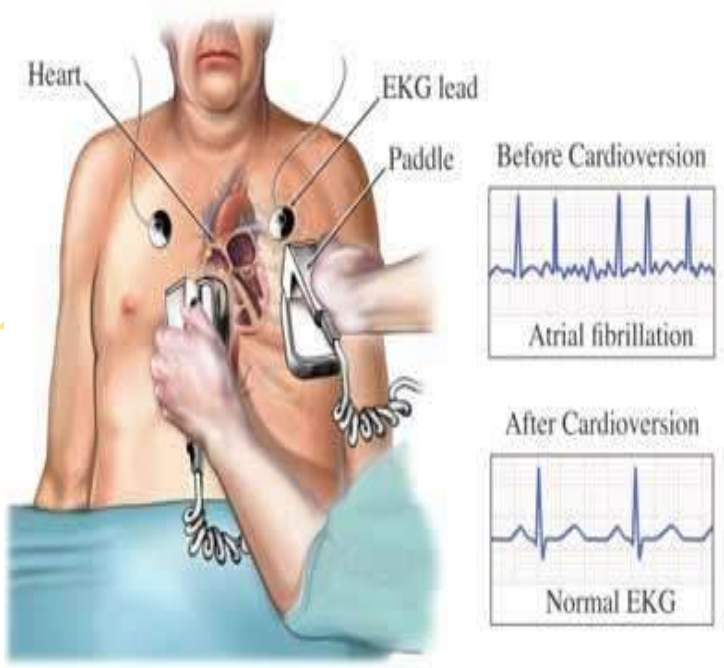
- Drugs may depress the automatic properties of abnormal pacemaker cells by decreasing the slope of phase 4 depolarization and/or by elevating threshold potential. Drugs may alter conduction characteristics of the pathways of a reentrant loop

Table 18. Vaughn-Williams Antiarrhythmic Drug Classes

Class/Ion Affected	Agents	Physiological Effect	Result on Electrophysiological Parameters	Clinical Utility
Class I/Na^+ channel blockers				
Ia (intermediate)	Disopyramide quinidine, procainamide	↓ Conduction velocity; ↑ refractory period	↑ QRS complex and ↑ QT interval	Atrial and ventricular arrhythmias
Ib (fast)	Lidocaine, mexiletine, phenytoin	↓ Conduction velocity; ↓↑ refractory period	↓ QT interval	Ventricular arrhythmias
Ic (slow)	Flecainide, propafenone	↓↓↓ Conduction velocity; Ø refractory period	↑ QRS complex	Supraventricular arrhythmias and ventricular arrhythmias
Class II β-blockers	Metoprolol, esmolol, atenolol	↓ Conduction velocity; ↑ refractory period	↓ HR and ↑ PR interval	Atrial and ventricular arrhythmias
Class III K^+ channel blockers	Amiodarone,[a] dronedarone,[a] sotalol,[b] dofetilide, ibutilide	Ø Conduction velocity; ↑↑↑ refractory period	↑ QT interval	Atrial and ventricular arrhythmias
Class IV Ca^{2+} channel blockers	Diltiazem, verapamil	↓ Conduction velocity; ↑ refractory period	↓ HR and ↑ PR interval	Atrial and ventricular arrhythmias

Hemodynamically Unstable AF

- In patients with new-onset AF or atrial flutter with signs and/or symptoms of hemodynamic instability (eg, severe hypotension, angina, and/or pulmonary edema),
- direct-current cardioversion (DCC) is indicated to restore sinus rhythm immediately (without regard to the risk of thromboembolism).



Ventricular Rate Control

If patients are hemodynamically stable, the focus should be directed toward controlling ventricular rate. Use drugs that slow conduction and increase refractoriness in the AV node as initial therapy

In patients with normal LV function (left ventricular ejection fraction [LVEF] >40% [0.40]), an IV β-blocker (propranolol, metoprolol, esmolol) or non-DHP CCB (diltiazem, verapamil) is recommended as first-line therapy .
If a high adrenergic state is the precipitating factor, IV β-blockers should be considered first

In patients with LVEF ≤40% (0.40), avoid IV diltiazem and verapamil and use IV β-blockers with caution. In patients having an exacerbation of HF symptoms, use IV digoxin or amiodarone as first-line.therapy for ventricular rate control. IV amiodarone can also be used in patients who are refractory or have contraindications to β-blockers, non-DHP CCBs, and digoxin.

Anticoagulation Prior to Cardioversion

If sinus rhythm is to be restored, initiate anticoagulation **prior** to cardioversion because return of atrial contraction may dislodge poorly adherent thrombi and increase the risk of thromboembolism. Initiating anticoagulation prior to cardioversion prevents clot growth and formation of new thrombi and allows existing thrombi to become organized and well adherent to the atrial wall. Patients become at increased risk of thrombus formation and a subsequent embolic event if the duration of AF **exceeds 48** hours.

Patients undergoing elective cardioversion for AF lasting longer than 48 hours or an unknown duration should receive warfarin (target international normalized ratio [INR] 2–3) or a direct oral anticoagulant (DOAC; apixaban, dabigatran, edoxaban, or rivaroxaban) for at least **3 weeks prior** to cardioversion. If cardioversion is successful, continue anticoagulation for at **least 4 weeks.**

Patients with AF less than 48 hours in duration do not require a prolonged period of anticoagulation prior to cardioversion because there has not been sufficient time to form atrial thrombi. Recommendations regarding short-term anticoagulation therapy given immediately prior to cardioversion differ. The CHEST guidelines recommend anticoagulation with either IV unfractionated heparin (UFH) or subcutaneous (LMWH) (with doses used for treating venous thromboembolism) at the time the patient presents with AF that is known to be less than 48 hours in duration. If the patient is successfully cardioverted to sinus rhythm, continue therapeutic anticoagulation with warfarin (INR target range 2–3) or a DOAC for at least 4 weeks. Decisions about long-term antithrombotic therapy beyond 4 weeks should be based on the patient's risk for stroke and not whether the patient is in sinus rhythm.

Conversion to Sinus Rhythm

After anticoagulation needs have been addressed (or after transesophageal echocardiography [TEE] demonstrated absence of a thrombus, obviating need for anticoagulation), methods for restoring sinus rhythm are pharmacologic cardioversion and DCC. Disadvantages of pharmacologic cardioversion are the risk of significant side effects (eg, drug-induced TdP, drug–drug interactions) and lower cardioversion rate for AADs compared with DCC.

DCC is **quick** and more often successful (80%–90% success rate), but it requires prior sedation or anesthesia and has a small risk of serious complications, such as sinus arrest or ventricular arrhythmias. Clinicians often elect to use AADs first, and then resort to DCC if these drugs fail. Pharmacologic cardioversion is most effective when initiated within 7 days after the onset of AF.

Chronic Anticoagulation for Stroke Prevention

When initiating chronic antithrombotic therapy to prevent stroke in patients with AF, selection of the appropriate regimen is based on the patient's stroke risk as determined by the CHA2DS2-Vasc risk scoring system. Patients are given 2 points each if they have a history of a previous stroke, transient ischemic attack, or thromboembolism, or if they are at least 75 years old. Patients are given 1 point each for age 65–74 years; having hypertension, diabetes, HF, or vascular disease; and being female.

✓Low risk : No antithrombotic therapy is recommended for males with a CHA2DS2-VASc score of 0 and females with a score of 1 because they are at low risk for stroke.

✓ Intermediate risk: For patients with one non sex stroke risk factor (ie, CHA2DS2-VASc score of 1 in males or 2 in females), oral anticoagulation is recommended over aspirin monotherapy, aspirin plus clopidogrel, or no antithrombotic therapy.

✓ High risk: For patients with more than one non sex stroke risk factor (ie, CHA2DS2-VASc score of ≥2 in males or ≥3 in females), oral anticoagulation is also recommended.

Chronic Antiarrhythmic Therapy

Use of AADs to prevent AF recurrences is controversial; AADs may be reasonable in patients who remain symptomatic despite having adequate ventricular rate control or for patients in whom adequate ventricular rate control cannot be achieved.

In patients with no SHD, dofetilide, dronedarone, flecainide, propafenone, or sotalol should be considered initially. Amiodarone is second line if the patient fails or does not tolerate one of these drugs. In patients with HF, amiodarone or dofetilide is first-line therapy, with catheter ablation as second-line. In patients with CAD dofetilide, dronedarone, or sotalol are first-line, with amiodarone as second-line therapy. Flecainide and propafenone should be avoided in the presence of SHD because of the risk of proarrhythmia.

Paroxysmal Supraventricular Tachycardia

Both pharmacologic and nonpharmacologic methods have been used to treat PSVT. Drugs can be divided into three broad categories:

(1) those that directly or indirectly increase vagal tone to the AV node (eg, digoxin),

(2) those that depress conduction through slow, calcium-dependent tissue (eg, adenosine, β-blockers, and non-DHP CCBs), and

(3) those that depress conduction through fast, sodium-dependent tissue (eg, quinidine,procainamide, disopyramide, and flecainide).

Premature Ventricular Complexes

In apparently healthy individuals without SHD, drug therapy is unnecessary because PVCs carry little or no risk. In addition, AADs should not be used to suppress asymptomatic PVCs. In patients with symptomatic PVCs who have risk factors for arrhythmic death (recent MI, LV dysfunction, or complex PVCs), limit chronic therapy to β-blockers. β-Blockers can also be used to suppress symptomatic PVCs in patients without underlying heart disease.

Ventricular Tachycardia

Acute Ventricular Tachycardia

If severe symptoms are present, institute synchronized DCC immediately to restore sinus rhythm and correct precipitating factors if possible.

Patients with mild or no symptoms can be treated initially with AADs. IV procainamide, amiodarone, or sotalol may be considered in this situation;lidocaine is an alternative agent

Sustained Ventricular Tachycardia

Patients with chronic recurrent sustained VT are at high risk for death. Use of invasive electrophysiologic studies and serial Holter monitoring with drug testing have been largely abandoned. These findings and the side-effect profiles of antiarrhythmic agents have led to nondrug approaches. The automatic ICD is a highly effective method for preventing sudden death due to recurrent VT or VF.

Ventricular Proarrhythmia

The proarrhythmia caused by the class Ic AADs is often resistant to resuscitation with cardioversion or overdrive pacing. IV lidocaine or sodium bicarbonate (which reverses the excessive sodium channel blockade) has been used successfully by some clinicians.

Torsade de Pointes

For an acute episode of TdP, most patients require and respond to DCC. However, TdP tends to be paroxysmal and often recurs rapidly after DCC.

IV magnesium sulfate is the drug of choice for preventing recurrences of TdP.

Bradyarrhythmias

Asymptomatic sinus bradyarrhythmias usually do not require treatment.

Symptomatic carotid sinus hypersensitivity also should be treated with permanent pacemaker therapy.

Patients who remain symptomatic may benefit from adding an α-adrenergic stimulant such as midodrine.

Atrioventricular Block

If patients with second- or third-degree AV block develop signs or symptoms of poor perfusion (eg, altered mental status, chest pain, hypotension, shock) administer atropine (0.5 mg IV given every 3–5 minutes, up to 3 mg total dose). Transcutaneous pacing can be initiated in patients unresponsive to atropine. Infusions of epinephrine (2–10 mcg/min) or dopamine (2–10 mcg/kg/min) can also be used in the event of atropine failure.

Chronic symptomatic AV block warrants insertion of a permanent pacemaker. Patients without symptoms can sometimes be followed closely without the need for a pacemaker