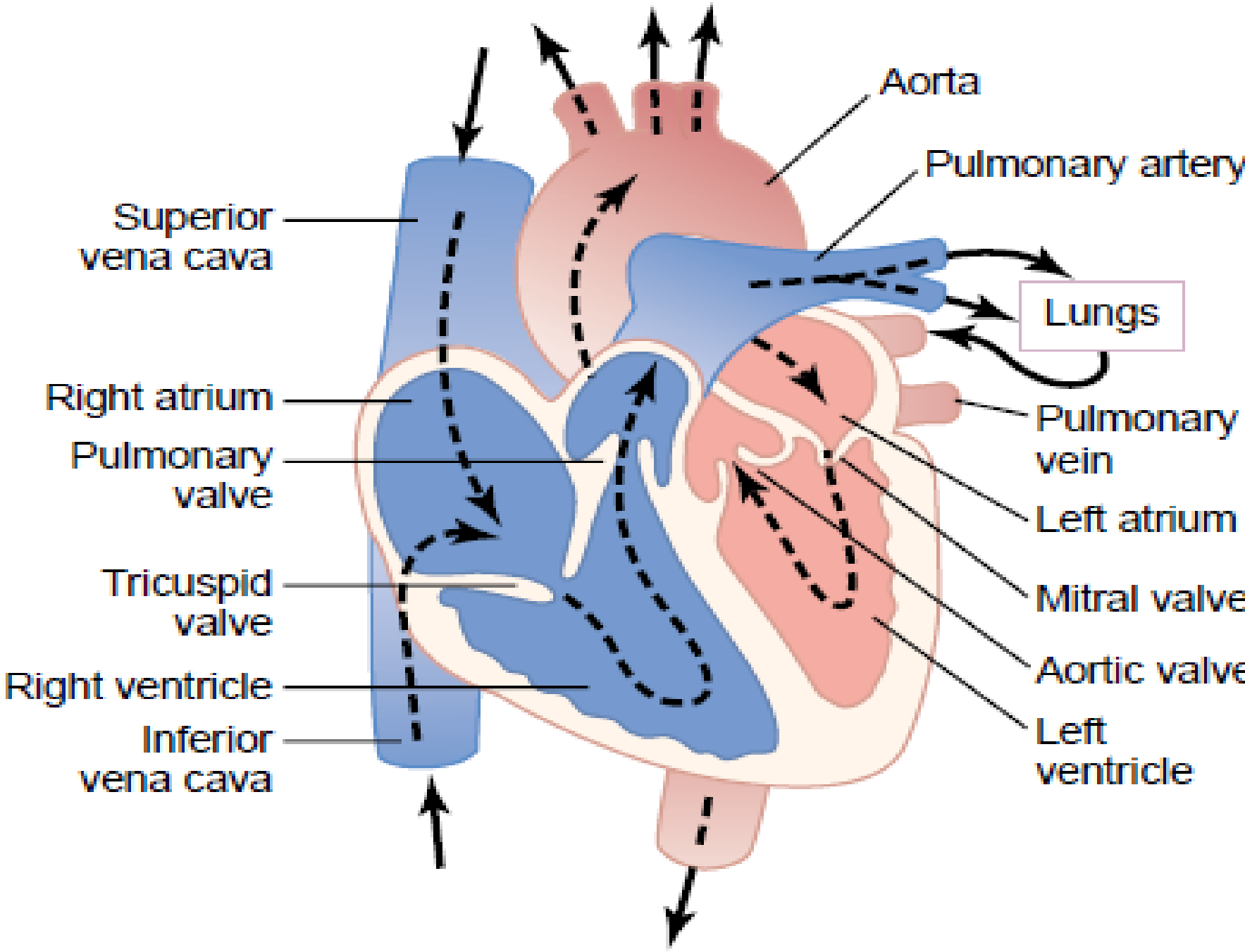
# ARRHYTHMIAS

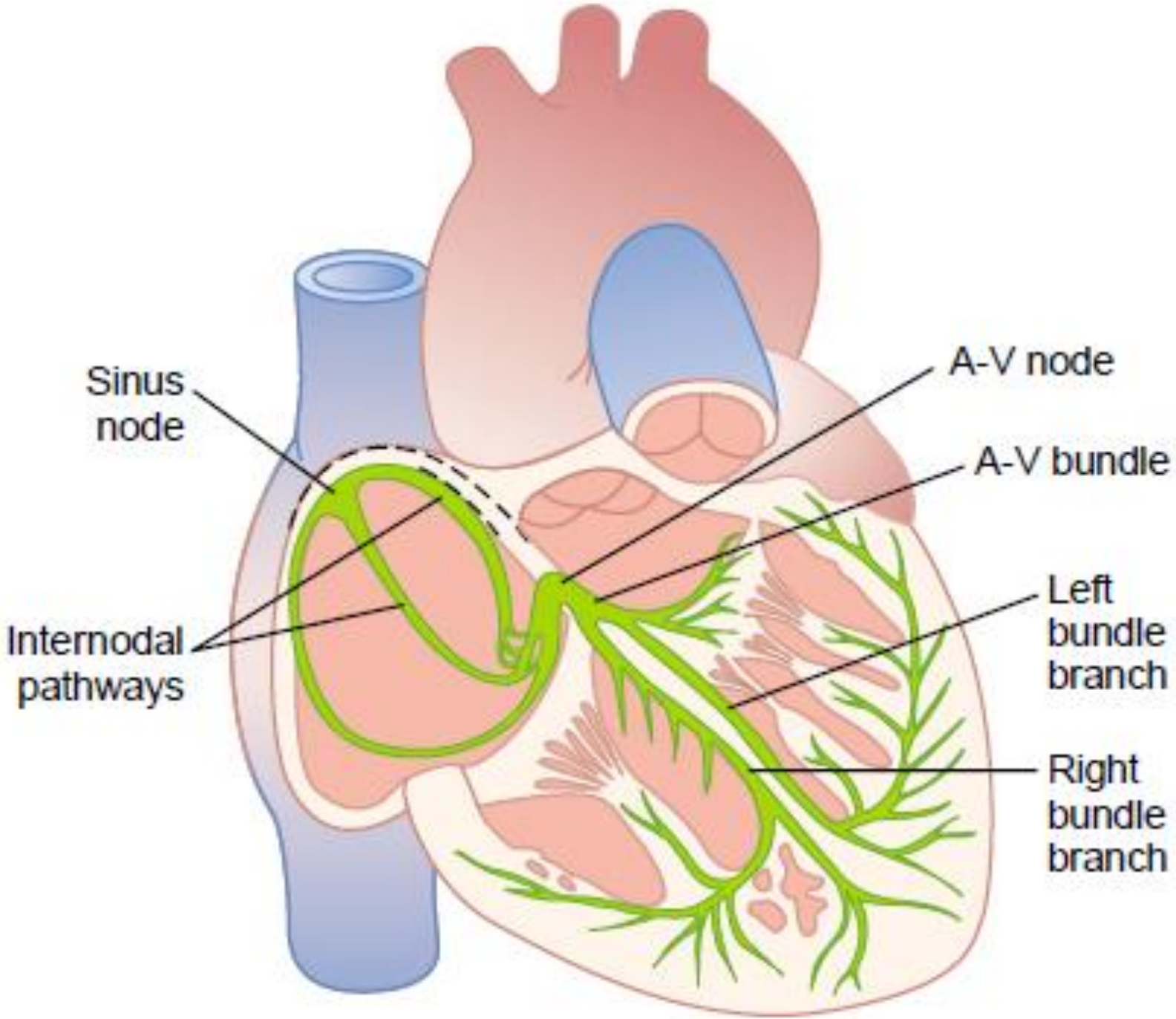
*Applied Therapeutics*

# DEFINITION

- Arrhythmia is defined as loss of cardiac rhythm, especially irregularity of heartbeat.
- A group of conditions caused by an abnormality in the rate, regularity, or sequence of cardiac activation.




TRUNK AND LOWER EXTREMITY


Sinus node
A-V node
A-V bundle
Internodal pathways
Left bundle branch
Right bundle branch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- The heart is endowed with a special system for

(1)generating rhythmical electrical impulses to cause rhythmical contraction of the heart muscle .

(2)conducting these impulses rapidly through the heart .

- When this system functions normally, the atria contract about one sixth of a second ahead of ventricular contraction, which allows filling of the ventricles before they pump the blood through the lungs and peripheral circulation. Another special importance of the system is that it allows all portions of the ventricles to contract almost simultaneously, which is essential for most effective pressure generation in the ventricular chambers.
- This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The result is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death.

# PATHOPHYSIOLOGY

- **SUPRAVENTRICULAR ARRHYTHMIAS**
  - Common supraventricular tachycardias requiring drug treatment are .

  1- atrial fibrillation

  2- atrial flutter

  3- paroxysmal supraventricular tachycardia

  4- automatic atrial tachycardias.
- **VENTRICULAR ARRHYTHMIAS**
  - Premature ventricular complexes (PVCs)
  - Ventricular tachycardia (VT)
- **BRADYARRHYTHMIAS**

# Atrial Fibrillation and Atrial Flutter

- **Atrial fibrillation** is characterized as an extremely rapid (400 to 600 atrial beats/min) and disorganized atrial activation. There is a loss of atrial contraction (atrial kick), and supraventricular impulses penetrate the atrioventricular (AV) conduction system in variable degrees, resulting in irregular ventricular activation and irregularly irregular pulse (120 to 180 beats/min).
- **Atrial flutter** is characterized by rapid (270 to 330 atrial beats/min) but regular atrial activation. The ventricular response usually has a regular pattern and a pulse of 300 beats/min. This arrhythmia occurs less frequently than atrial fibrillation but has similar precipitating factors, consequences, and drug therapy.

# Atrial Fibrillation and Atrial Flutter

- The predominant mechanism of atrial fibrillation and atrial flutter is reentry, which is usually associated with organic heart disease that causes atrial distention (e.g., ischemia or infarction, hypertensive heart disease, valvular disorders).
- Additional associated disorders include acute pulmonary embolus and chronic lung disease, resulting in pulmonary hypertension and cor pulmonale; and states of high adrenergic tone such as thyrotoxicosis, alcohol withdrawal, sepsis, or excessive physical exertion.

# Paroxysmal Supraventricular Tachycardia Caused by Reentry

- Paroxysmal supraventricular tachycardia (PSVT) arising by reentrant mechanisms includes.

1- arrhythmias caused by AV nodal reentry.

2- AV reentry incorporating an anomalous AV pathway.

3-sinoatrial (SA) nodal reentry.

4- intra-atrial reentry.

# VENTRICULAR ARRHYTHMIAS

- **Premature Ventricular Complexes**
- Premature ventricular complexes (PVCs) are common ventricular rhythm disturbances that occur in patients with or without heart disease and may be elicited experimentally by abnormal automaticity, triggered activity, or reentrant mechanisms.

# Ventricular Tachycardia

- Ventricular tachycardia (VT) is defined by three or more repetitive PVCs occurring at a rate greater than 100 beats/min. It occurs most commonly in acute myocardial infarction (MI); other causes are severe electrolyte abnormalities (e.g., hypokalemia), hypoxemia, and digitalis toxicity.
- The chronic recurrent form is almost always associated with underlying organic heart disease (e.g., idiopathic dilated cardiomyopathy or remote MI with left ventricular [LV] aneurysm).

# BRADYARRHYTHMIAS

- Asymptomatic sinus bradyarrhythmias (heart rate less than 60 beats/min) are common especially in young, athletically active individuals. However, some patients have sinus node dysfunction (sick sinus syndrome) because of underlying organic heart disease and the normal aging process, which attenuates SA nodal function.
- 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 (e.g., trained athletes) or during sleep when vagal tone is high. It may be transient when the underlying etiology is reversible (e.g., myocarditis, myocardial ischemia, during drug therapy).

# CLINICAL PRESENTATION

- **Supraventricular tachycardias** may cause a variety of clinical manifestations ranging from no symptoms to minor palpitations and/or irregular pulse to severe and even life-threatening symptoms.
- Patients may experience dizziness or acute syncopal episodes; symptoms of heart failure; anginal chest pain; or, more often, a choking or pressure sensation during the tachycardia episode.

# CLINICAL PRESENTATION

- **Atrial fibrillation or flutter** may be manifested by the entire range of symptoms associated with other supraventricular tachycardias, but syncope is not a common presenting symptom.
- An additional complication of atrial fibrillation is arterial embolization resulting from atrial stasis and poorly adherent mural thrombi, which accounts for the most devastating complication: embolic stroke.

# DIAGNOSIS

- The surface electrocardiogram (ECG) is the cornerstone of diagnosis for cardiac rhythm disturbances.

# DESIRED OUTCOME

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

# TREATMENT

- GENERAL PRINCIPLES
- The use of antiarrhythmic drugs is declining because of major trials that showed increased mortality with their use in several clinical situations, the realization of proarrhythmia as a significant side effect, and the advancing technology of nondrug therapies such as ablation and the internal cardioverter-defibrillator.

# Classification of Antiarrhythmic Drugs

| Type | Drug | Conduction Velocity[a] | Refractory Period | Automaticity | Ion Block |
|---|---|---|---|---|---|
| Ia | Quinidine<br>Procainamide<br>Disopyramide | ↓ | ↑ | ↓ | Sodium (intermediate)<br>Potassium |
| Ib | Lidocaine<br>Mexiletine<br>Tocainide | 0/↓ | ↓ | ↓ | Sodium (fast on/off) |
| Ic | Flecainide<br>Propafenone<br>Moricizine | ↓↓ | 0 | ↓ | Sodium (slow on/off)<br>Potassium |
| II | β-Blockers | ↓ | ↑ | ↓ | Calcium (indirect) |
| III | Amiodarone<br>Bretylium<br>Dofetilide<br>Sotalol<br>Ibutilide | 0 | ↑↑ | 0 | Potassium |
| IV | Verapamil<br>Diltiazem | ↓ | ↑ | ↓ | Calcium |

# ATRIAL FIBRILLATION OR ATRIAL FLUTTER

- Many methods are available for restoring sinus rhythm, preventing thromboembolic complications, and preventing further recurrences however, treatment selection depends in part on onset and severity of symptoms.
- If symptoms are severe and of recent onset, patients may require direct-current cardioversion (DCC) to restore sinus rhythm immediately.
- If patients are hemodynamically stable, the focus should be directed toward control of ventricular rate.
- Drugs that slow conduction and increase refractoriness in the AV node should be used as initial therapy. In patients with normal LV function (left ventricular ejection fraction >40%), IV β blockers (**propranolol, metoprolol, esmolol**), **diltiazem**, or **verapamil** is recommended.

# ATRIAL FIBRILLATION OR ATRIAL FLUTTER

- After treatment with AV nodal blocking agents and a subsequent decrease in ventricular response, the patient should be evaluated for the possibility of restoring sinus rhythm if atrial fibrillation persists.
- If sinus rhythm is to be restored, anticoagulation should be initiated prior to cardioversion because return of atrial contraction increases risk of thromboembolism. Current recommendations are to initiate warfarin (international normalized ratio [INR] 2 to 3) for at least 3 weeks prior to cardioversion and continuing for at least 1 month after effective cardioversion. Anticoagulation may not be necessary in patients with atrial fibrillation of less than 48 hours' duration and in the absence of atrial thrombus or severe stasis on transesophageal echocardiography (TEE).

# Paroxysmal Supraventricular Tachycardia (SVT)

- The choice between pharmacologic and nonpharmacologic methods for treating PSVT depends on symptom severity.
- Synchronized DCC is the treatment of choice if symptoms are severe (e.g., syncope, near syncope, anginal chest pain, severe heart failure).
- Nondrug measures that increase vagal tone to the AV node (e.g., unilateral carotid sinus massage, Valsalva maneuver) can be used for mild to moderate symptoms. If these methods fail, drug therapy is the next option.

# Paroxysmal Supraventricular Tachycardia (SVT)

- The choice among drugs is based on the QRS complex . Drugs can be divided into three broad categories:

(1) those that directly or indirectly increase vagal tone to the AV node (e.g., digoxin);

(2) those that depress conduction through slow, calcium-dependent tissue (e.g., adenosine, β blockers, calcium channel blockers);

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

- Adenosine has been recommended as the drug of first choice in patients with PSVT because its short duration of action will not cause prolonged hemodynamic compromise in patients with wide QRS complexes who actually have VT rather than PSVT.

# Paroxysmal Supraventricular Tachycardia (SVT)

- After acute PSVT is terminated, long-term preventive treatment is indicated if frequent episodes necessitate therapeutic intervention or if episodes are infrequent but severely symptomatic.
- Chronic antiarrhythmic drug treatment in young, otherwise healthy patients is problematic because of possible need for lifelong daily medication, poor tolerability, occasional severe side effects, and frequent lack of efficacy.
- Transcutaneous catheter ablation using radiofrequency current on the PSVT substrate should be considered in any patient who would have previously been considered for chronic antiarrhythmic drug treatment.

# VENTRICULAR TACHYCARDIA

- If severe symptoms are present, DCC should be instituted to restore sinus rhythm immediately. Precipitating factors should be corrected if possible.
- Patients with mild or no symptoms can be treated initially with antiarrhythmic drugs. IV ***amiodarone*** is usually the first step in this situation. ***Procainamide*** or ***lidocaine*** given IV are suitable alternatives.
- DCC should be instituted or a transvenous pacing wire should be inserted if the patient's status deteriorates, VT degenerates to VF or drug therapy fails.