Arrhythmias

Objectives:

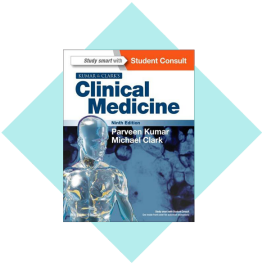
- Identify the common arrhythmias
- Know the differential diagnosis of heart rhythm disorder.
- Learn the treatment modalities and diagnosis of atrial fibrillation.
- Know the risk stratification of atrial fibrillation patient.

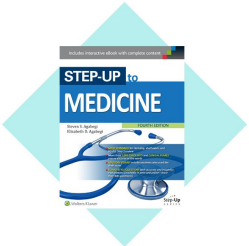
[Color index : Important | Notes | Extra]

[Editing file | Feedback | Share your notes | Shared notes | Twitter]

Resources:

- 435 slides, 434 & 432 teams





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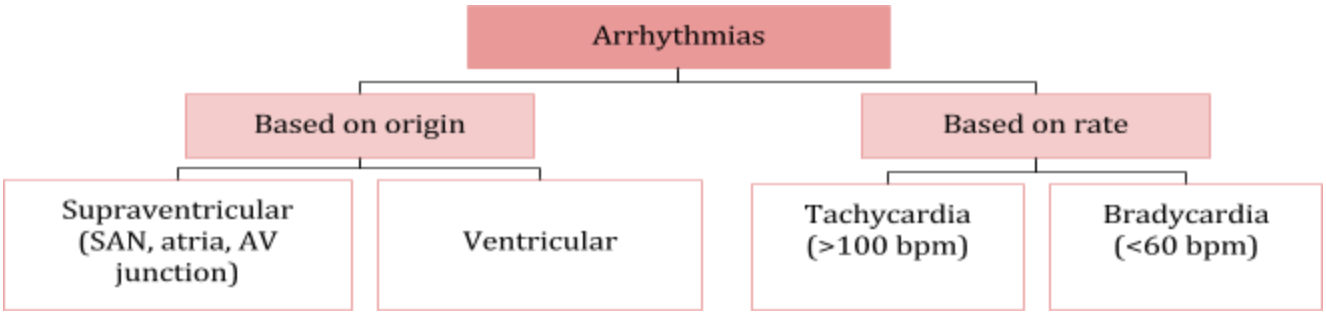
Introduction to Cardiac Arrhythmias

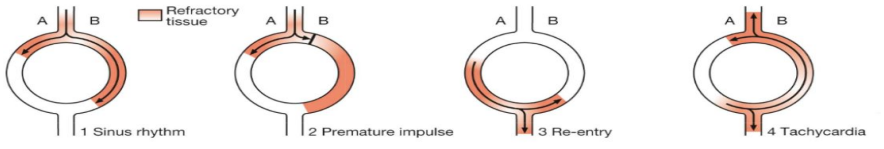
- The heartbeat is normally initiated by an electrical discharge from the sinoatrial (sinus) node. The atria and ventricles then depolarize sequentially as electrical depolarisation passes through specialized conducting tissues.The sinus node acts as a **pacemaker** and its intrinsic rate is regulated by the autonomic nervous system.
- A reduction of parasympathetic tone or an increase in sympathetic stimulation leads to tachycardia; conversely, increased parasympathetic tone or decreased sympathetic stimulation produces bradycardia. The sinus rate in women is slightly faster than in men.
- **A cardiac arrhythmia** is a disturbance of the electrical rhythm of the heart. Arrhythmias are often a manifestation of structural heart disease but may also occur because of abnormal conduction or depolarization in an otherwise healthy heart.
- An arrhythmia may be 'supraventricular' (sinus, atrial or junctional) or ventricular in origin. Supraventricular rhythms usually produce narrow QRS complexes because the ventricles are depolarized in their normal sequence via the AV node and bundle of His. In contrast, ventricular rhythms produce broad, bizarre QRS complexes because the ventricles are activated in an abnormal sequence.



- **There are two main types of arrhythmia :** *(Based on rate)*
  1) ***Bradycardia*:** the heart rate is **slow (<60 b.p.m. during the day or <50 b.p.m. at night)**. Slower heart rates are more likely to cause symptomatic arrhythmias.

  2) ***Tachycardia*:** the heart rate is **fast (>100 b.p.m.).** Tachycardias are more likely to be symptomatic when the arrhythmia is fast and sustained. **There are three main mechanisms of tachycardia :**

Increased automaticity (In response to any kind of stress)	The tachycardia is produced by repeated spontaneous depolarisation of an ectopic focus, often in response to catecholamines.
Re-entry	The tachycardia is initiated by an ectopic beat and sustained by a re-entry circuit . Most tachyarrhythmias are due to re-entry.
Triggered activity	It is a form of secondary depolarisation arising from an incompletely repolarized cell membrane.



Atrial tachyarrhythmias

WE ARE 100% SURE THAT YOU WILL UNDERSTAND EVERYTHING ABOUT AF AFTER WATCHING THIS VIDEO

1) Atrial fibrillation (AF) :

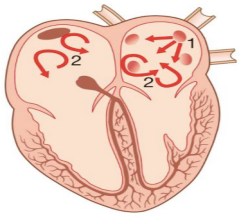
- Atrial fibrillation (AF) is **the most common** sustained cardiac arrhythmia with an overall prevalence of 0.5% in the adult population of the UK.
  - ➔ Atrial fibrillation accounts for 1/3 of all patient discharges with arrhythmia as principal diagnosis.[1]
- The prevalence rises with **age**, affecting 1% of those aged 60–64 years, increasing to 9% of those aged over 80 years.
- **AF is a complex arrhythmia characterised by both :**

1) abnormal **automatic firing**.

2) The presence of multiple interacting **re-entry circuits** looping around the atria.

- ➔ AF becomes sustained because of **re-entrant conduction** within the atria or sometimes because of **continuous ectopic firing**.

- Episodes of atrial fibrillation are initiated by rapid bursts of ectopic beats arising from conducting tissue in the **pulmonary veins** or from diseased atrial tissue.
- Re-entry is more likely to occur in atria that are enlarged, or in which conduction is slow (as is the case in many forms of heart disease).
- During episodes of AF, the atria beat rapidly but in an *uncoordinated and ineffective manner*.
- The ventricles are activated irregularly at a rate determined by conduction through the AV node.



(1) Ectopic beats, often arising from the pulmonary veins, trigger atrial fibrillation.
(2) Re-entry within the atria maintains atrial fibrillation, with multiple interacting re-entry circuits operating simultaneously.

- **Normal heart rhythm is disrupted in AF :**

1) AF is characterized by :

- Rapid **(350–600 beats/min)** and **irregular** atrial rhythm
- **Reduced filling** of the left and right ventricles

2) Conduction of most impulses from the atria to ventricles is blocked at the AV node[2]

3) Contraction of the ventricles can be:

- Irregular and rapid (110–180 beats/min; tachycardia)
- Irregular and slow (<50 beats/min; bradycardia)
- Normal

4) Cardiac output can be reduced.

[1] 34% Atrial Fibrillation ,18% Unspecified, 10% VT ((Ventricular tachycardia), 9% SSS (Sick Sinus Syndrome),8% Conduction Disease, 6% PVCs (Premature ventricular contractions), 6% PSVT (Paroxysmal supraventricular tachycardia), 4% Atrial Flutter, 3% SCD (Sudden cardiac death), 2% VF (Ventricular fibrillation).

[2] Atrial rate is over 400 bpm, but most impulses are blocked at the AV node so ventricular rate ranges between 75 and 175.

- **AF causes remodelling (AF begets** "generate" **AF) :**

1) Electrical remodelling :

Electrophysiological changes occur in the atria within a few hours of the onset of AF that tend to maintain fibrillation (**shortening of refractory period**[3]).

2) Structural:

When AF persists for a period of months, structural remodelling occurs, with **atrial fibrosis** and **dilatation** that further predispose to AF.

➔ Thus early treatment of AF will prevent re-initiation of the arrhythmia.

- Many episodes of AF resolve spontaneously
- Over time AF tends to become persistent or permanent.

- **Classification of atrial fibrillation :**

A. Based on TIME :

First diagnosed	- AF that **has not** been diagnosed before - **Irrespective** of the duration of the arrhythmia or the presence and severity of AF-related symptoms.
Paroxysmal	- Intermittent episodes which self-terminate within 7 days (in most cases within 48 hours). - AF episodes that are cardioverted within 7 days should be considered paroxysmal.
Persistent	- AF that lasts longer than 7 days including episodes that are terminated by cardioversion either by drugs or by direct current cardioversion (7 days or more) prolonged episodes that can be terminated by electrical or chemical cardioversion
Long-standing persistent AF	Continuous AF lasting for 1 year or more when it is decided to adopt a rhythm control strategy.
Permanent	- More than one year and does not respond to cardioversion. - Paroxysmal AF will become permanent as the underlying disease process that disposes to AF progress. - Continuous, with a joint decision between the patient and the physician to cease further attempts to regain sinus rhythm.

B. Based on ETIOLOGY :

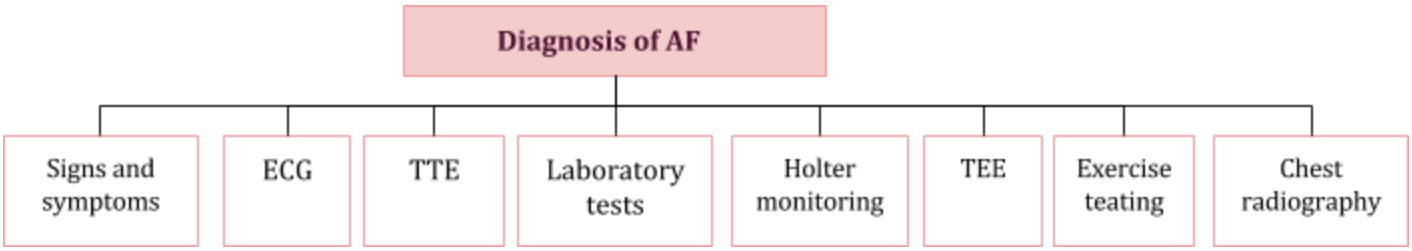
Lone or primary	- AF without clinical/ECG evidence of cardiopulmonary disease. About 50% of all patients with paroxysmal AF and 20% of patients with persistent or permanent AF have structurally normal hearts; this is known as '**lone** atrial fibrillation'.
Secondary	AF **associated** with cardiopulmonary disease (e.g. myocardial infarction or pneumonia)
Non-valvular	AF that is not associated with damage to the heart valves (Absence of rheumatic mitral valve disease, prosthetic heart valve or mitral valve repair). *Any AF EXCEPT prosthetic mechanical valve and mitral stenosis

[3] Refractory period is the amount of time it takes for an excitable membrane to be ready for a second stimulus once it returns to its resting state following excitation.

- **Causes of Atrial fibrillation :**

Cardiac	Non-cardiac
• **Coronary artery disease (including acute MI)** • **Hypertensive heart disease** • **Valvular heart disease** – Rheumatic: mitral stenosis – Non-rheumatic: aortic stenosis And mitral regurgitation. • **Pericarditis and pericardial trauma (e.g., surgery).** • **Cardiac tumors: atrial myxoma** • **Sick sinus syndrome** • **Cardiomyopathy** – Hypertrophic – Idiopathic dilated • **Post-coronary bypass surgery**	• **Pulmonary** – COPD – Pneumonia – Pulmonary embolism • **Metabolic** – Thyroid disease: hyperthyroidism or hypothyroidism. – Electrolyte disorder • **Toxic:** alcohol ('holiday heart' syndrome) • **Stress (e.g., postoperative)** • **Systemic illness (e.g., sepsis, malignancy, DM).**

- **Diagnosis of Atrial fibrillation :**



History and physical examination :

➔ Clinical conditions associated with AF :

– Underlying heart conditions (e.g. valvular heart disease, heart failure, coronary artery disease, HTN)

– Other reversible conditions.

➔ Family history :

– Familial AF (lone AF in a family)

– AF secondary to other genetic conditions (familial cardiomyopathies)

➔ Type of AF :

– First episode, paroxysmal, persistent, permanent

– Triggers – e.g. emotional stress, alcohol, physical exercise, gastroesophageal disease

– Specific symptoms

– Response to any treatments administered

1)Signs and symptoms

- **Symptoms attributable to atrial fibrillation are highly variable. They vary according to :**

➔ Irregularity and rate of ventricular response

➔ Functional status

➔ AF duration

➔ Patient factors

➔ Co-morbidities

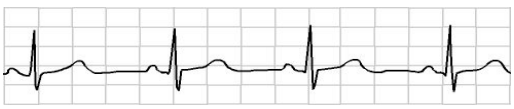
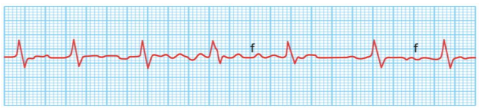
- In some patients (about 30%), it is an **incidental finding**, while others attend hospital as an emergency with rapid palpitations, dyspnoea and/or chest pain following the onset of atrial fibrillation. Most patients with on-going atrial fibrillation experience some deterioration of exercise capacity or well being, but this may be appreciated only once sinus rhythm is restored. When caused by rheumatic mitral stenosis, the onset of atrial fibrillation results in considerable worsening of cardiac failure.

Signs and symptom	Cause
- Irregularly irregular pulse - Palpitations	Irregular heartbeat
- Fatigue - Diminished exercise capacity - Breathlessness (dyspnoea) - Weakness (asthenia)	Decreased cardiac output
- Dizziness and fainting (syncope)	Hypotension
- Chest pain (angina)	Cardiac ischaemia
- Thromboembolic TIA, stroke	Increased risk of clot formation

2)Electrocardiogram (ECG)

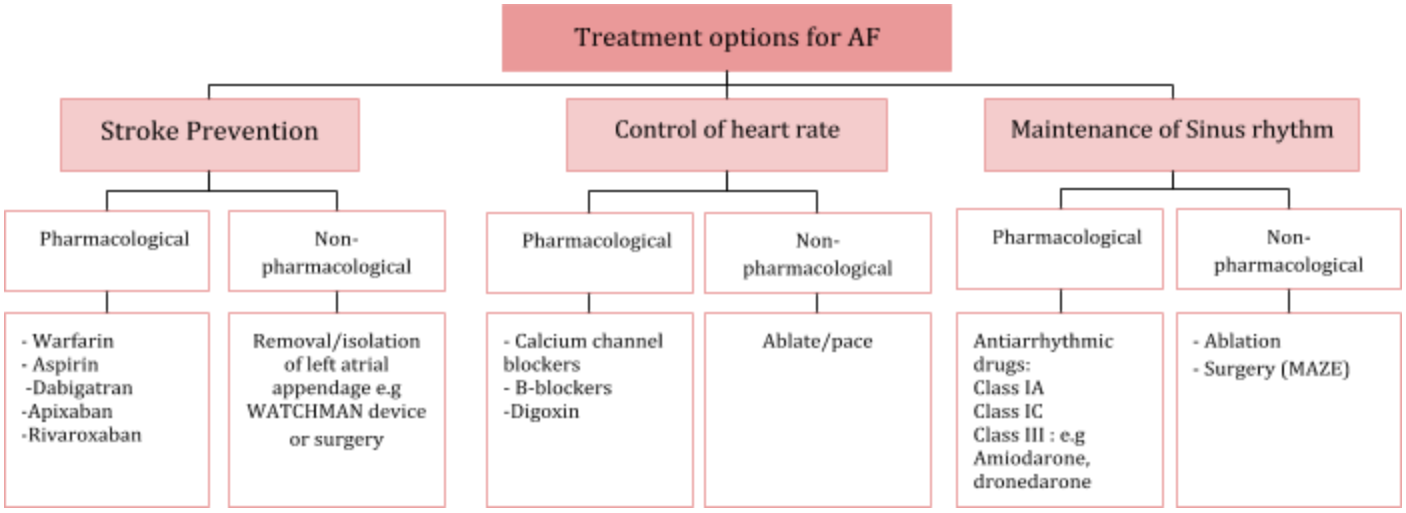
- **Assesses the electrical activity of the heart**
- **Essential for all patients with suspected AF, to identify :**

[Abnormal heart rhythm (verify AF), Left ventricular hypertrophy, Pre-excitation, Bundle-branch block, Prior MI, Differential diagnosis of other atrial arrhythmias].

Normal sinus rhythm	AF
- Normal heart rate - Regular rhythm - P Waves 	– Heart rate increased (tachyarrhythmia) Reduced heart rate (bradyarrhythmia) may also be observed – Irregular rhythm (Irregularly irregular) – No P wave – Irregular baseline (f waves) 

3)Transthoracic Echocardiography (TTE)	- Non-invasive - Used to identify :(Size and functioning of atria and ventricles, Ventricle hypertrophy, Pericardial disease and Valvular heart disease).
4) Laboratory tests	• Routine blood tests should be carried out at least once in patients with AF • **Important parameters to assess include:** **– Thyroid function** – Renal function – Hepatic function – Serum electrolytes – Complete blood count
5) Holter monitor	• Portable ECG device (Diagnostic, follow up) • Continuous monitoring for a short period of time (typically 24 hours) • **Useful for :** – Detecting asymptomatic AF – Evaluating patients with paroxysmal AF – Associating symptoms with heart rhythm disturbance – Assessing response to treatment
6)Transesophageal-echocardiogram (TEE) **THE ONLY TEST THAT CAN RULE OUT THROMBUS EXISTENCE!!**	• Ultrasound transducer positioned close to the heart using an endoscope-like device • High quality images of cardiac structure and function particularly the **left atrial appendage,** the most common site of thrombi in patients with AF Appendage is the only muscular part of the atrium so 95% of thrombosis occur there. • **Not routinely used but useful for:** – Accurate assessment of risk of stroke – Sensitive detection of atrial thrombi - Detection of low flow velocity ('smoke' effect)
7) Chest radiography	• When clinical findings suggest an abnormality chest radiography may be used to : – Evaluate pulmonary pathology and vasculature – Detect congestive heart failure – Assess enlargement of the cardiac chambers

- **Treatment and management of AF (3 strategies) :**
  1. Prevention of thromboembolism
  2. Rate control
  3. Restoration and maintenance of sinus rhythm



★ قاعدة ذهبية مهم ترسخ ببالكم :

★ **Valvular Afibs** are Caused by: **1-Mitral Stenosis 2- Mechanical Prosthetic** valve and they have 10x risk of coagulation, thus Anticoagulants must be used irrespectively of CHA_2DS_2VASc score.

★ Other Afibs which are caused by other Valvular conditions (Eg. MR, AS, AR..) are called **Non-Valvular Afib**

➔ Anticoagulation is indicated in patients with atrial fibrillation related to rheumatic mitral stenosis or in the presence of a mechanical prosthetic heart valve. In patients with **non-valvular atrial fibrillation** (in the absence of mitral stenosis, artificial heart valves or mitral valve repair), a scoring system known as CHA_2DS_2VASc is used as the first step in determining the need for anticoagulation.

Stroke prevention :

- Regular measurement of INR levels is an essential component in the management of patients receiving warfarin treatment. Warfarin has drug- drug and food-drug interaction (e.g. food containing Vitamin K).
- Aspirin (Antiplatelet) :Antiplatelet therapy with aspirin plus clopidogrel (or, less effectively, aspirin only) should be considered in patients who refuse any oral anticoagulant therapy or cannot tolerate anticoagulants for reasons unrelated to bleeding.
- Dabigatran.Apixaban and Rivaroxaban are oral anticoagulants with No food-drug interaction and minimize drug-drug interaction, no need for monitoring. They are AS EFFECTIVE AS WARFARIN AND THEY REPLACE WARFARIN COMPLETELY **(new oral anticoagulant agents (NOACs)).**

➔ When oral anticoagulation is required, either warfarin (dose adjusted to maintain an INR between 2 and 3) or one of the **new oral anticoagulant agents (NOACs)** can be used. These latter agents fall into two classes: direct thrombin inhibitors (e.g. dabigatran) and oral direct factor Xa inhibitors (e.g. rivaroxaban and apixaban). NOACs specifically block a single step in the coagulation cascade, in contrast to warfarin, which blocks several vitamin K-dependent factors (II, VII, IX and X). Unlike warfarin, the NOACs have a rapid onset of action, shorter half-life and fewer food and drug interactions, and do not require INR testing. Trial data have shown them to be equally effective as, and safer than, warfarin. However, these agents require dose reduction or avoidance in patients with renal impairment, the elderly or those with low body weight.

- If there are contraindications to use anticoagulants > catheterization > Watchman device: The device is meant to capture blood clots and prevent them from migrating into the circulation and causing a stroke. Watchman would provide an alternative to warfarin and other blood thinners for preventing stroke and systemic embolism.

Control of HR :

- B blockers are contraindicated in patients with Asthma so Calcium channel blockers are used instead and these two drugs have the same efficacy while Digoxin is the least efficient and used for patients who cannot tolerate CCB or beta blockers, it could also be used as (Post-operative medication).
- Uncontrolled heart rate = pacemaker.

Maintenance of sinus rhythm :

- Options for the long-term management of cardiac tachyarrhythmias include: antiarrhythmic drug therapy,ablation therapy or device therapy.
- Antiarrhythmic drugs : Amiodarone causes harmful side effects and usually given to elderly. Class IC (Flecainide, Propafenone) are safer for young patients.

Prevention of Thromboembolism :

- Stroke Risk Score for Atrial Fibrillation : for estimating the risk of stroke in patients with **non-rheumatic atrial fibrillation :**

- **The CHADS2 Index:**

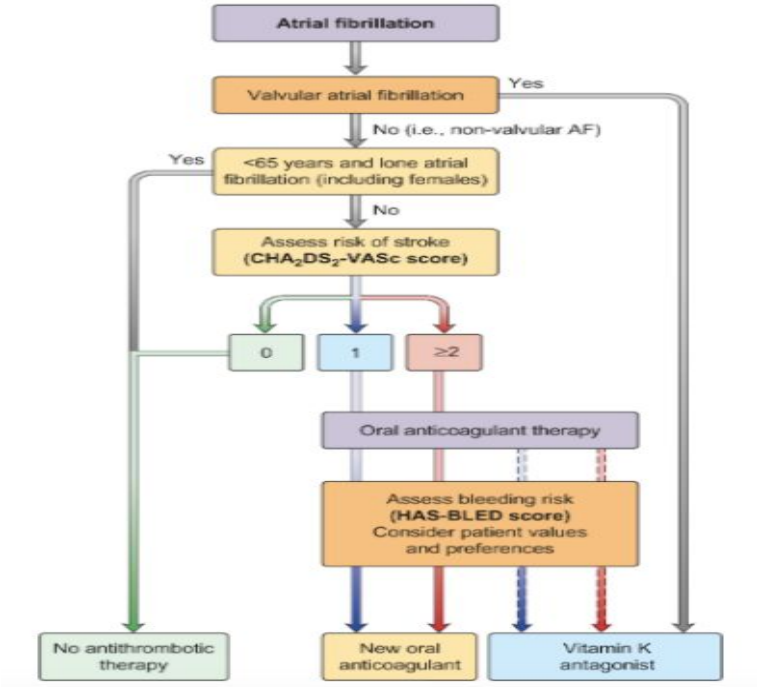
	Score (Points)	Prevalence (%)
Congestive Heart failure	1	32
Hypertension	1	65
Age >75 years	1	28
Diabetes mellitus	1	18
Stroke or TIA	2	10
Moderate-High risk	≥ 2	50-60
Low risk	0-1	40-50

-If patient was at a high risk according to CHADS2 score then he must take Warfarin or one of "ban" family drugs.
-If patient was at low risk then Aspirin is given

- **The $CHA_2DS_2VASc^4$ Index** : more accurate than $CHADS_2$

	Weight (Points)
Congestive Heart failure or left ventricular ejection fraction equals or less than 35% (LVEF)	1
Hypertension	1
Age >75 years	**2**
Diabetes mellitus	1
Stroke /TIA/ Systemic embolism	**2**
Vascular disease (MI/PAD/Aortic plaque)	**1**
Age 65-74 years	**1**
Sex category	**1**
Moderate-High risk	**≥ 2**
Low risk	**0-1**

[4] هذي اللي بتسألون عنها في الاختبار!



Restoration of Sinus rhythm:

- **Rhythm-control therapies :**

- The objective of rhythm-control therapy is to **restore (cardioversion)** and **maintain** normal sinus rhythm.
- **Cardioversion can be achieved by:**
  - Pharmacotherapy with *antiarrhythmic agents*
  - Electrical shocks *(direct-current cardioversion)*
- Direct-current cardioversion is generally more effective than pharmacotherapy
- Likelihood of successful cardioversion <u>decreases</u> with the **duration** of AF :

➔ Pharmacological cardioversion is most effective when initiated within 7 days of AF onset

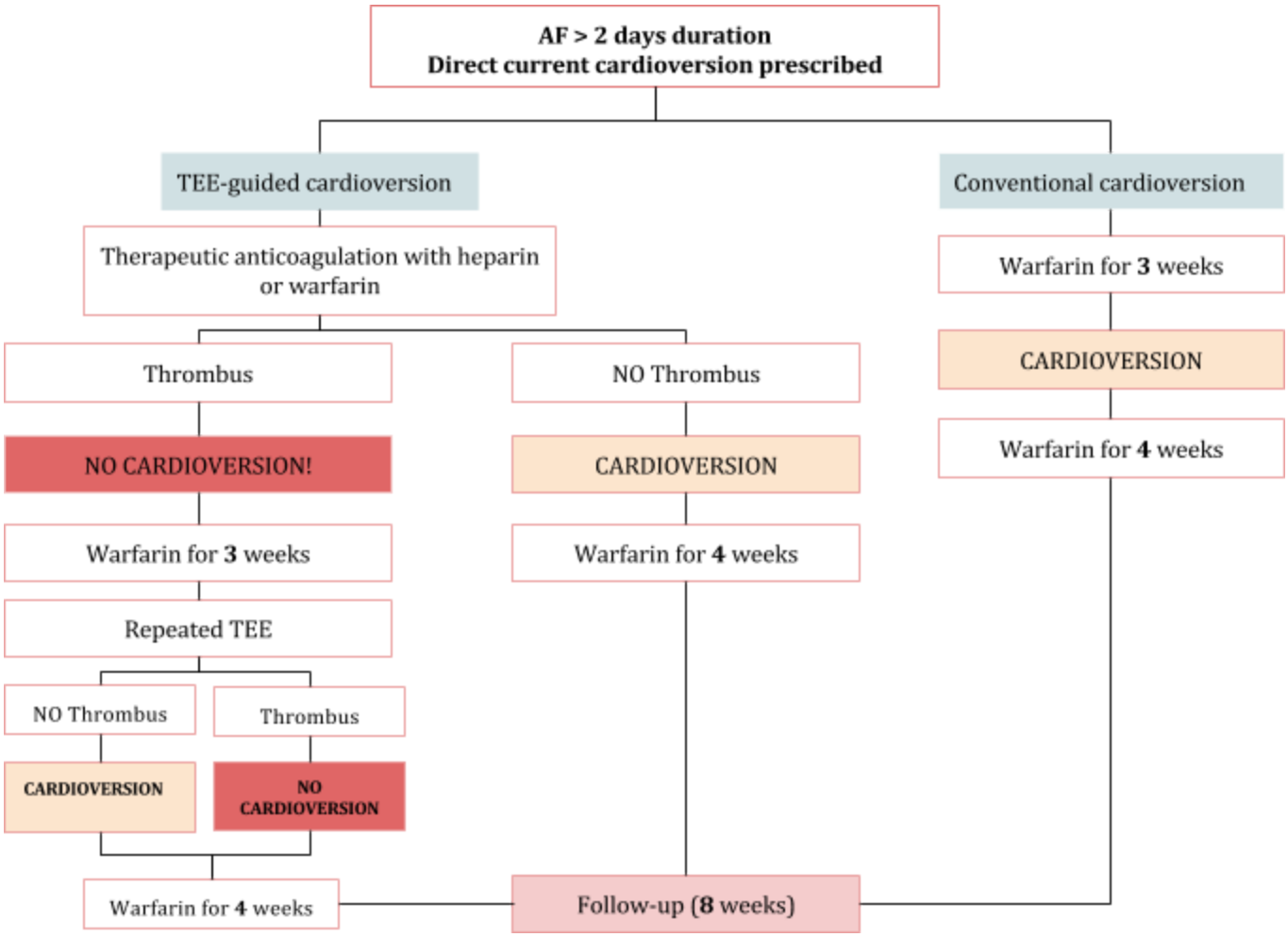
- Cardioversion can dislodge thrombi in the atria, increasing the risk of stroke

➔ **Thromboprophylaxis is recommended for 3 weeks before and for at least 4 weeks after cardioversion in patients with AF that has persisted for 48 hrs !!!!!!!!!**

After shocking the patient, anticoagulants must be given for 4 weeks because patient might develop a clot. REGARDLESS OF LOW RISK FACTORS. After 4 weeks of anticoagulants, evaluate the risk factors, if they are **high** continue with anticoagulants.

<u>TEE-guided cardioversion: ACUTE study design:</u>

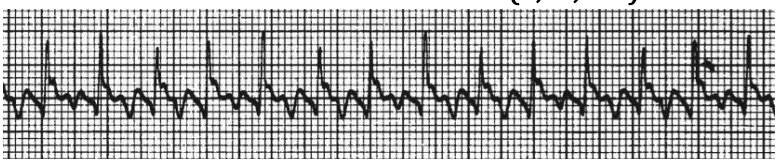
To avoid waiting 3 weeks for anticoagulation, obtain a transesophageal echocardiogram (TEE) to image the left atrium. If no thrombus is present, start IV heparin and perform cardioversion within 24 hours. Patients still require 4 weeks of anticoagulation after cardioversion.



- **Consequences of AF :**

Event	Association with AF
Death	Increased mortality especially cardiovascular mortality due to sudden death, heart failure or stroke.
Stroke	20-30% of all strokes are due to AF. A growing number of patients with stroke are diagnosed with "silent", paroxysmal AF.
Hospitalization	10-40% of patients are hospitalized every year.
Left ventricular dysfunction & HF	LVD is found in 20-30% of all AF patients. AF causes or aggravates LVD In many AF patients, while others have completely preserved LV function despite long-standing AF.
Cognitive decline and vascular dementia	Cognitive decline and vascular dementia can develop even in anticoagulated AF patients. Brain white matter lesions are more common in AF patients than patients without AF.

2) Atrial Flutter:

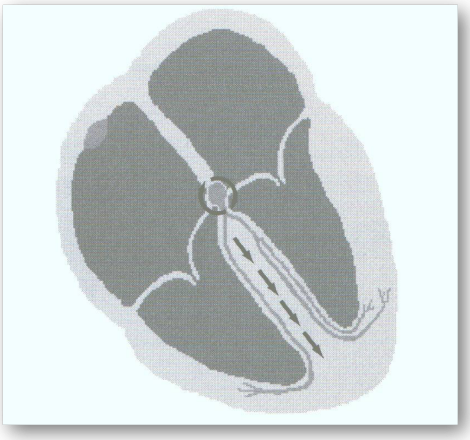
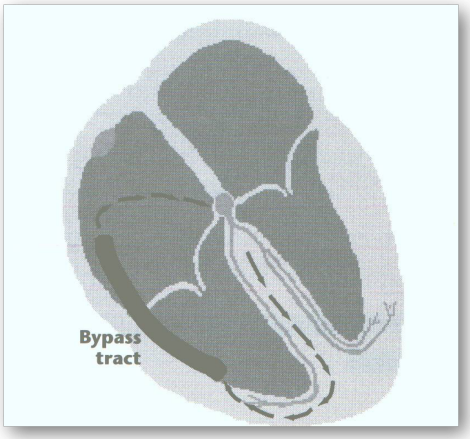
<table>
<tr><th colspan="4">Atrial Flutter</th></tr>
<tr><td>Pathophysiology</td><td colspan="3">One irritable automaticity focus in the atria fires at about 250 to 350 bpm (typically very close to 300 bpm), giving rise to regular atrial contractions.
Atrial rate between around 300 bpm
Ventricular rate is one-half to one-third of the atrial rate
The long refractory period in the AV node allows only one out of every two or three flutter waves to conduct to the ventricles.</td></tr>
<tr><td>Causes</td><td>Heart disease: Heart failure (most common association), rheumatic heart disease, CAD</td><td>COPD</td><td>Atrial septal defect (ASD)</td></tr>
<tr><td>Diagnosis (ECG)</td><td colspan="3">ECG provides a saw-tooth baseline
QRS complex appearing after every second or third “tooth” (P wave)
Saw-tooth flutter waves are best seen in the inferior leads (II, III, aVF).  </td></tr>
<tr><td>Treatment</td><td colspan="3">Similar to treatment for AFib</td></tr>
</table>

3) Multifocal atrial tachycardia :

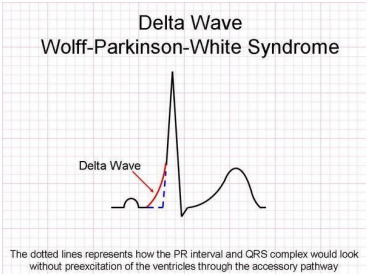
- Usually occurs in patients with **severe pulmonary disease (e.g., COPD)**
- **ECG findings:** Variable P wave morphology and variable PR and RR intervals. At leasT three different P wave morphologies are required to make an accurate diagnosis.
- The diagnosis of wandering atrial pacemaker is identical except that the heart rate is between 60 and 100 bpm **(i.e., not tachycardic).**
- Can also be diagnosed by use of vagal maneuvers or adenosine to show AV blocK without disrupting the atrial tachycardia
- Treatment involves improving oxygenation and ventilation (strong association between MAT and lung disease). If left ventricular function is preserved, acceptable treatments include calcium channel blockers, β-blockers, digoxin, amiodarone, IV flecainide, and IV propafenone. If LV function is not preserved, use digoxin, diltiazem, or amiodarone. Electrical cardioversion is ineffective and should not be used.

Supraventricular Tachycardia (SVT)

SVT Video (6:23 minutes) Arrhythmias of supraventricular origin using a re-entrant mechanism with abrupt onset & termination [AVNRT (60%) ,AVRT (30%), Atrial tachycardia (10%)].

	AV nodal reentrant tachycardia (AVNRT)	Orthodromic AV reentrant tachycardia (AVRT)
Pathophysiology	• Two pathways **(one fast and the other slow)** within the AV node, so the reentrant circuit is within the AV node. • Most common cause of supraventricular tachyarrhythmia (SVT) • Initiated or terminated by **PACs** 	• An accessory pathway between the atria and ventricles that conducts anterogradely or retrogradely, Called a "**concealed bypass tract**" • A common cause of SVTs • Initiated or terminated by **PACs or PVCs** 
ECG	**Narrow QRS** complexes with **no discernible P waves** (P waves are buried within the QRS complex) Why? This is because the circuit is short and conduction is rapid, so impulses exit to activate atria and ventricles simultaneously	**Narrow QRS** complexes with P waves which **may or may not be discernible**, depending on the rate. Why? This is because the accessory pathway is at some distance from the AV node (reentrant circuit is longer), and there is a difference in the timing of activation of the atria and ventricles.
Causes	➔ Ischemic heart disease ➔ Digoxin AV node reentry ➔ Atrial flutter with rapid ventricular response ➔ AV reciprocating tachycardia (accessory pathway) ➔ Excessive caffeine or alcohol consumption	
Treatment and Prevention	Maneuvers that **stimulate the vagus delay AV conduction** and thus block the reenetry mechanism: ❖ The Valsalva maneuver ❖ Carotid sinus massage breath holding ❖ Head immersion in cold water (or placing an ice bag to the face) **Acute Treatment:** ★ **IV adenosine:** Only used in SVT and has a short duration of action. ONLY USED WITH SVT ★ **CCB** (IV Verapamil) and β -blocker (IV Esmolol) or Digoxin are appropriate in Patients with preserved Left Ventricular function ★ **Direct Current Cardioversion** **Prevention:** • **Digoxin** is usually the drug of choice. • **Verapamil or β -blockers** are alternatives. • Radiofrequency catheter ablation of either the AV node or the accessory tract	

- **WPW Syndrome:** it's a subtype of AVRT



- An accessory pathway **(Kent bundles)** connecting the atria and the ventricles, will lead to premature depolarization of the ventricles, therefore a shorter PR interval (since we lost the properties of the AV node to delay conduction) and a **delta wave** (which is a slurring of the QRS complex).

So we can say that WPW Syndrome is a symptomatic AVRT.

- As the signal bypasses the AV node, this can develop a reentrant circuit, causing tachycardia
- If Afib occurs in these patients, this could predispose to a fatal situation. Where the AF fires through the accessory pathway, This should be treated with DC cardioversion.
- The first-line therapy is to ablate the accessory pathway. (curative)

- WPW can lead to paroxysmal tachycardia, by two possible mechanisms:

Orthodromic reciprocating tachycardia	**Supraventricular tachycardias (AFib or atrial flutter)**
• The impulse travels through the AV node (anterograde limb) and depolarizes the ventricles. Then it travels back through the accessory pathway (the retro grade limb) and redepolarizes the atria, creating a reentry loop. • No delta waves (because conduction occurs retrograde over the accessory pathway).	Usually, AV node only allows certain impulses to get to ventricles. With an accessory pathway, all or most of the impulses may pass to the ventricles. A fast ventricular rate may occur and cause hemodynamic compromise

Bradyarrhythmias

- **Bradycardia could be due to :**
  2. *Reduced automaticity*, e.g. sinus bradycardia.
  3. *Blocked or abnormally slow conduction*, e.g. AV block.

• Sinus bradycardia:

Sinus bradycardia is due to :

1. **Extrinsic factors that influence the normal SA node :**

Such as: Hypothermia, hypothyroidism, raised intracranial pressure, drugs (eg, beta blockers).

2. **Intrinsic sinus node disease :**

Such as : Acute ischemia or infarction of sinus node(complication of MI), sick sinus syndrome(chronic degenerative idiopathic fibrosis of SA node)

• Heart block:

Degree of heart block:	**First degree**	**Second degree (Beat drops in both types)**		**Third degree**
		Mobitz type I (Wenkebach)	**Mobitz type II**	
Characteristics	PR interval (>0.2ms) , rarely causes symptoms	Progressive PR prolongation and beat drop	Constant PR prolongation and beat drop	Complete dissociation, ventricle and atria contract contract irrespective to each other
Treatment	No Treatment		Pacemaker	

Ventricular arrhythmias

Type	Ventricular tachycardia	Ventricular Fibrillation
Characteristics	• Rapid and repetitive firing at rate of more than 120 bpm, with broad abnormal QRS complexes. • Usually occurs in patients with CHD or cardiomyopathies • May cause : 1/ hemodynamic instability 2/ or degenerate into V fib	• Usually preceded by V tach • Very rapid and irregular ventricular activation without mechanical effect. • Patient is pulseless , becomes unconscious , can get into cardiac arrest.
Treatment	• In cases of emergency with a systolic BP less than 90 mmhg > DC cardioversion is required. • But if the patient is stable give IV amiodarone	• Immediate defibrillation and CPR, if persistent> IV epinephrine or vasopressin If fails >antiarrhythmics. • ICD when cardioversion is successful

Summary (Step-up to medicine)

- **Treatment of Atrial fibrillation :**

1. Acute AFib in a hemodynamically *unstable* patient: Immediate electrical cardioversion to sinus rhythm

2. Acute AFib in a hemodynamically stable patient:

a. Rate control

• Determine the pulse in a patient with AFib. If it is too rapid, it must be treated. Target rate is 60 to 100 bpm.

• β -blockers are preferred. Calcium channel blockers are an alternative.

• If left ventricular systolic dysfunction is present, consider digoxin or amioda-rone (useful for rhythm control).

b. Cardioversion to sinus rhythm (after rate control is achieved)

• Candidates for cardioversion include those who are hemodynamically unstable, those with worsening symptoms, and those who are having their first ever case of AFib.

• Electrical cardioversion is preferred over pharmacologic cardioversion. Attempts should be made to control ventricular rate before attempting DC cardioversion.

• Use pharmacologic cardioversion only if electrical cardioversion fails or is not feasible: Parenteral ibutilide, procainamide, flecainide, sotalol, or amiodarone are choices.

c. Anticoagulation to prevent embolic cerebrovascular accident (CVA)

• If AFib present >48 hours (or unknown period of time), risk of embolization during cardioversion is significant (2% to 5%). Anticoagulate patients for 3 weeks before and 4 weeks after cardioversion.

• An INR of 2 to 3 is the anticoagulation goal range.

• To avoid waiting 3 weeks for anticoagulation, obtain a transesophageal echocardiogram (TEE) to image the left atrium. If no thrombus is present, start IV heparin and perform cardioversion within 24 hours. Patients still require 4 weeks of anticoagulation after cardioversion.

3. Chronic AFib

a. Rate control with a β -blocker or calcium channel blocker

b. Anticoagulation

• Patients with "lone" AFib (i.e., AFib in the absence of underlying heart disease or other cardiovascular risk factors) under age 60 do not require anticoagulation because they are at low risk for embolization (aspirin may be appropriate).

• Treat all other patients with chronic anticoagulation (warfarin).

CLINICAL PEARL 1-12

Cardioversion Versus Defibrillation

Cardioversion

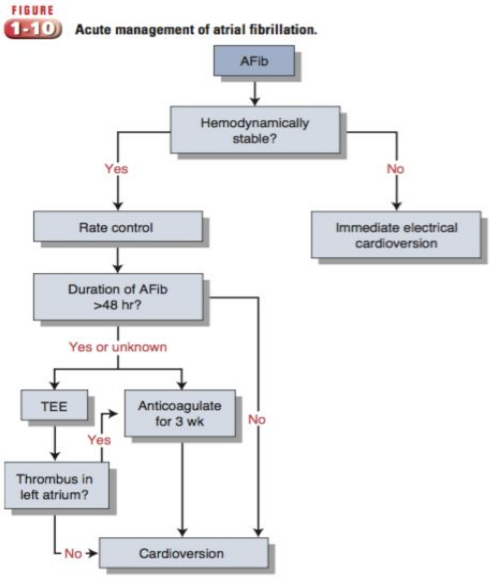
- Delivery of a shock that is in synchrony with the QRS complex: Purpose is to terminate certain dysrhythmias such as PSVT or VT; an electric shock during T wave can cause Vfib, so the shock is timed not to hit the T wave.
- Indications: AFib, atrial flutter, VT with a pulse, SVT

Defibrillation

- Delivery of a shock that is not in synchrony with the QRS complex: Purpose is to convert a dysrhythmia to normal sinus rhythm
- Indications: VFib, VT without a pulse

Automatic Implantable Defibrillator

- Device that is surgically placed: When it detects a lethal dysrhythmia, it delivers an electric shock to defibrillate. It delivers a set number of shocks until the dysrhythmia is terminated.
- Indications: VFib and/or VT that is not controlled by medical therapy



Cases

1. **In the ICU, a patient suddenly becomes pulseless and unresponsive, with cardiac monitor indicating ventricular tachycardia. The crash cart is immediately available. What is the best first therapy?**
    A. Amiodarone
    B. Lidocaine
    C. Epinephrine
    D. Defibrillation

2. **A 60-year-old woman develops chest pain, respiratory distress, and confusion after right hip replacement surgery. She is confused and appears in respiratory distress. Blood pressure is 80/50, heart rate of 155/minute. ECG reveals atrial fibrillation. Which of the following is the best management of this patient's arrhythmia?**
    A. immediate defibrillation
    B. Intravenous amiodarone
    C. Intravenous adenosine
    D. Immediate electric cardioversion

3. **An 18-year-old man military recruit reports several episodes of palpitation and syncope over the past several years. Physical examination is unremarkable. His ECG shows shortening of PR interval and delta waves What is the most likely diagnosis?**

    A. Congenital prolonged QT syndrome
    B. Hypertrophic obstructive cardiomyopathy (HOCM)
    C. Preexcitation syndrome (Wolff-Parkinson-White)
    D. Rheumatic mitral stenosis

4. **A 65 year old male with a history of hypertension, stage IV kidney disease on dialysis presents to the emergency department with dizziness and palpitations for 1 hour. He denies chest pains. His temperature is 37.0 C, blood pressure 80/40, heart rate 150, respirations 24, and oxygen saturation 85% on room air. Physical examination reveals pulmonary rales throughout his lung fields, elevated jugular venous pressure, a regular tachycardic rhythm without murmurs, and trace lower extremity pitting edema. Laboratory studies are normal. His ECG is shows saw-tooth baseline. Which of the following is the most appropriate initial therapy?**
    A. Intravenous amiodarone
    B. Intravenous beta-blockers
    C. Intravenous calcium channel blockers
    D. direct current cardioversion

5. Which is the preferred method to detect left atrial appendage thrombus?

A. TEE
B. MRI
C. TTE
D. CT

6. A 79-year-old woman is admitted to the coronary care unit (CCU) with unstable angina. She is started on appropriate medication to reduce her cardiac risk. She is hypertensive, fasting glucose is normal and cholesterol is 5.2. She is found to be in atrial fibrillation. What is the most appropriate treatment?

A. Aspirin and clopidogrel
B. Digoxin
C. Cardioversion
D. Aspirin alone
E. Warfarin

7. While on call you are called by a nurse to a patient on the ward complaining of light headedness and palpitations. When you arrive the patient is not conscious but has a patent airway and is breathing with oxygen saturation at 97 per cent. You try to palpate a pulse but are unable to find the radial or carotid. The registrar arrives and after hearing your report of the patient decides to shock the patient who recovers. What is the patient most likely to have been suffering?

A. Torsades de Pointes
B. Ventricular fibrillation
C. Sustained ventricular tachycardia
D. Non-sustained ventricular tachycardia
E. Normal heart ventricular tachycardia

8.A 21-year-old man is on his way home from a party when he experiences the sudden onset of rapid palpitations. He feels uncomfortable but not short of breath and has no chest pain. He goes to the nearest accident and emergency department, where he is found to have a supraventricular tachycardia (SVT) at a rate of 170/minute. Carotid sinus massage produced transient reversion to sinus rhythm, after which the tachycardia resumed. What would be the next step in your management?

A. Repeat carotid sinus massage
B. IV verapamil
C. IV propranolol
D. IV adenosine

E. Synchronized DC cardioversion

9.A 62-year-old male presents with palpitations, which are shown on ECG to be atrial fibrillation with a ventricular rate of approximately 130/minute. He has mild central chest discomfort but is not acutely distressed. He first noticed these about 3 hours before coming to hospital. As far as is known this is his first episode of this kind. Which of the following would you prefer as first-line therapy?

A. Anticoagulate with heparin and start digoxin at standard daily dose
B. Attempt DC cardioversion
C. Administer bisoprolol and verapamil, and give warfarin
D. Attempt cardioversion with IV flecainide
E. Wait to see if there is spontaneous reversion to sinus rhythm

ANSWERS

1.D

The standard approach to ventricular fibrillation or hypotensive ventricular tachycardia involves defibrillation, followed by epinephrine if needed. Therapy with lidocaine, amiodarone, or procainamide may be warranted if prior interventions fail.

2.D

This woman has hemodynamically unstable atrial fibrillation which requires immediate electrical cardioversion.

3.C

The ECG reveals shortened PR interval and a delta wave causing widening of the QRS. The delta wave is a "slurring" of the upstroke of the R wave caused by the early depolarization of ventricular myocardium. This is consistent with an accessory conduction pathway or WPW.

4.D

The ECG reveals atrial flutter with a rapid ventricular rate. The patient is not tolerating it from a hemodynamic standpoint (hypotensive), thus immediate restoration of sinus rhythm is indicated with direct current cardioversion (a shock).

5.A

Because the left atrium is most clearly visualized through the TEE.

6. E

Ideally this patient should be started on antihypertensives, a beta blocker and a statin. There is no indication for hypoglycaemics at present. There is no indication that this is acute atrial fibrillation and she does not seem to be compromised in a female of this age, cardioversion (C) is unlikely to be successful. She should be rate-controlled but the beta blockade is more appropriate in light of her ischaemic heart disease. Whether to start anticoagulation (A) is a decision that has to be tailor-made for each individual patient. The CHAD2 score is a quick and dirty but very useful way of predicting risk of subsequent stroke as a result of atrial fibrillation and helps guide the prescription of prophylactic antiplatelets or anticoagulants. Other factors, such as ease of taking and monitoring warfarin, risk of falls and important risk factors, such as vascular disease, should be taken into account.

7.B

This patient is suffering from a life-threatening ventricular tachyarrhythmia of which there are two types, sustained ventricular tachycardia and ventricular fibrillation. In ventricular fibrillation (B) the patient is pulseless and cardioversion is required. A sustained ventricular tachycardia (C) is usually recognized by cannon 'a' waves on JVP and broad QRS complexes if an ECG is available. If stable, patients can be cardioverted with amiodarone, if unstable, electrocardioversion is required. Torsades de pointes (A) presents with irregular QRS complexes and prolonged QT interval, a non-sustained ventricular tachycardia (D) is defined by more than five consecutive heart beats within 30 seconds, while a normal heart ventricular tachycardia (E) is a benign tachyarrhythmia. Answers (A), (D) and (E) are not shockable rhythms.

8.D

IV adenosine (D) has a very high likelihood of success, with rapid onset and offset. It may cause very brief chest pain (which is not ischaemic) and very occasionally bronchospasm. Verapamil (B) and beta-blockers (C) may also be effective but have a longer duration of action which is unnecessary here, may cause excessive bradycardia, and are in any case less effective than adenosine. If the patient has severe haemodynamic compromise, DC cardioversion (E) could be considered but would be excessive here. Carotid sinus massage (A) is likely to remain ineffective. SVT is common in young people and may be associated with excessive nicotine, caffeine and alcohol and patients should be advised about this, although they may not take much notice!

9.B

The onset of the arrhythmia is recent, and there is a good chance of successful cardioversion (B) at this point without the need for anticoagulation. Conservative management (E) is also reasonable, though the patient is in some discomfort. 'Chemical' cardioversion (D) may be somewhat less likely to succeed than DC cardioversion but may be preferred by the patient. Digoxin (A) may eventually control resting, but not ambulant heart rate, but would probably take several days before it did so. Option (C) is certainly suitable in cases of persistent or permanent atrial fibrillation where it is decided to opt for rate control.