Atrial Fibrillation

Presented by:

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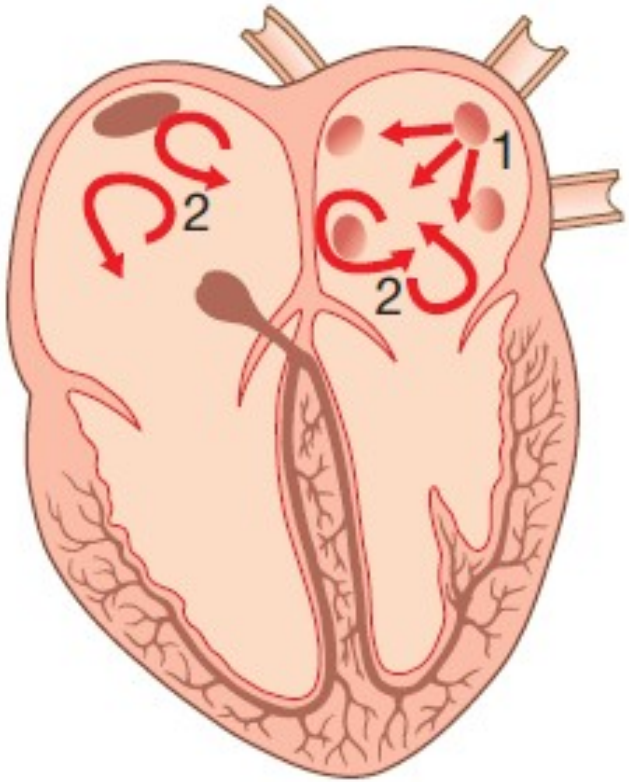
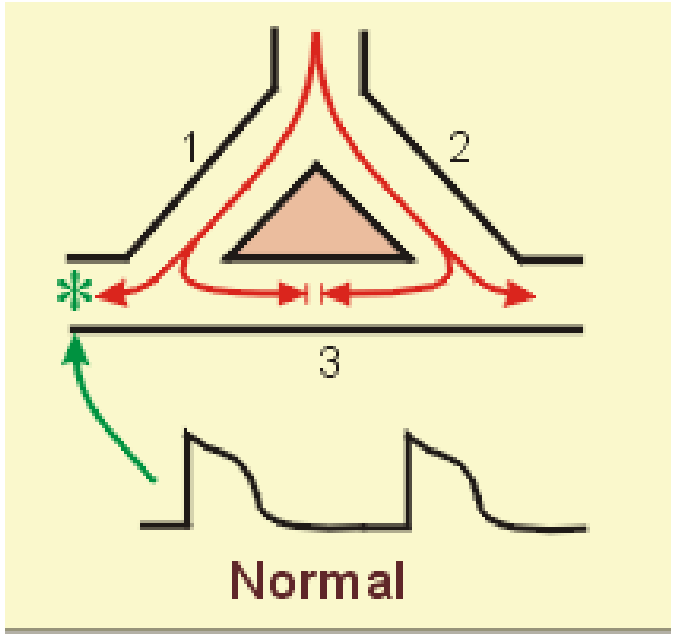
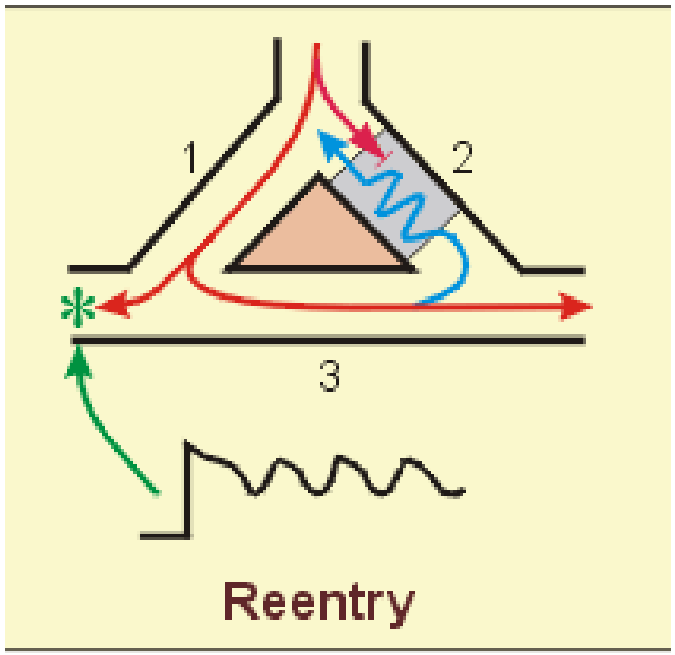
Introduction

- Most common sustained cardiac arrhythmia
- A complex arrhythmia characterised by
  - abnormal automatic firing and the
  - presence of multiple re-entry circuits
- Disorganized atrial depolarisation without effective atrial contraction
- The prevalence rises with increasing age
  - 1% & 9% of those aged 60-64 years and over 80 years
- Responsible for 15% strokes



Usually initiated by rapid bursts of ectopic beats arising from conducting tissue in the pulmonary veins or from diseased atrial tissue





Pathophysiology

- Electrophysiological changes occur in the atria within a few hours of the onset of AF that tend to maintain fibrillation: **electrical remodelling**

- 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 this and reinitiating of the arrhythmia

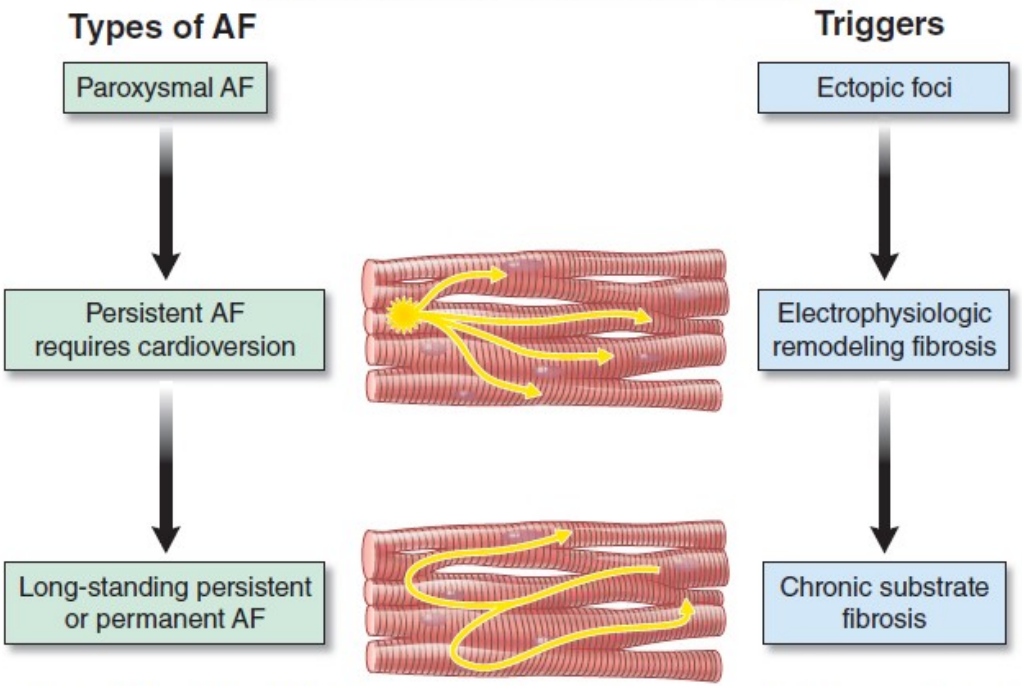


FIGURE 276-12 A rhythm strip of atrial fibrillation (AF) showing no distinct P-wave morphology and irregular ventricular response. Diagram depicts atrial fibrillation types. Paroxysmal AF is initiated by premature beats, as shown in the rhythm strip (*arrow*) after two sinus beats. Triggering foci are often an important cause of this arrhythmia. Persistent AF is associated with atrial structural and electrophysiologic remodeling, as well as with triggering foci in many patients. Long-standing persistent AF is associated with greater structural remodeling with atrial fibrosis and electrophysiologic remodeling.

Etiologies of AF

CARDIAC

Hypertensive heart disease

Valvular heart disease

Ischaemic heart disease

Cardiomyopathy

Pericarditis

Congenital heart disease

Post Cardiac surgery

Etiologies of AF contd:

NON CARDIAC

1. Pulmonary : Pneumonia, COPD,PE
2. Hyperthyroidism
3. Excess catecholamine /sympathetic activity
4. Drugs and alcohol
5. Significant electrolyte imbalance

LONE ATRIAL FIBRILLATION

- Younger patients < 60
- No underlying cause
- Usually not much symptoms
- Normal heart structure
- No associated co-morbidities

About 50% of all patients with paroxysmal AF and 20% of patients with persistent or permanent AF have structurally normal hearts

Classification of AF

Terminology	Clinical features	
Initial event (first detected episode)	Symptomatic Asymptomatic Onset unknown	Rhythm/Rate
Paroxysmal	Spontaneous termination <7 days and most often <48 hours	Rhythm Control
Persistent	Not self-terminating Lasting >7 days or prior cardioversion	Rhythm or Rate control
Permanent ('accepted')	Not terminated Terminated but relapsed No cardioversion attempt	Rate Control

Paroxysmal (intermittent)

- Recurrent AF (≥2 episodes)

- Terminates spontaneously in less than seven days, usually less than 24 hours
- It will **become permanent as the underlying disease process that predisposes to AF progresses**

Persistent

- **Fails to self-terminate within seven days**
- Episodes often require pharmacologic or electrical cardioversion to restore sinus rhythm
- Persistent AF can have later episodes of paroxysmal AF as AF is generally considered a progressive disease

Permanent

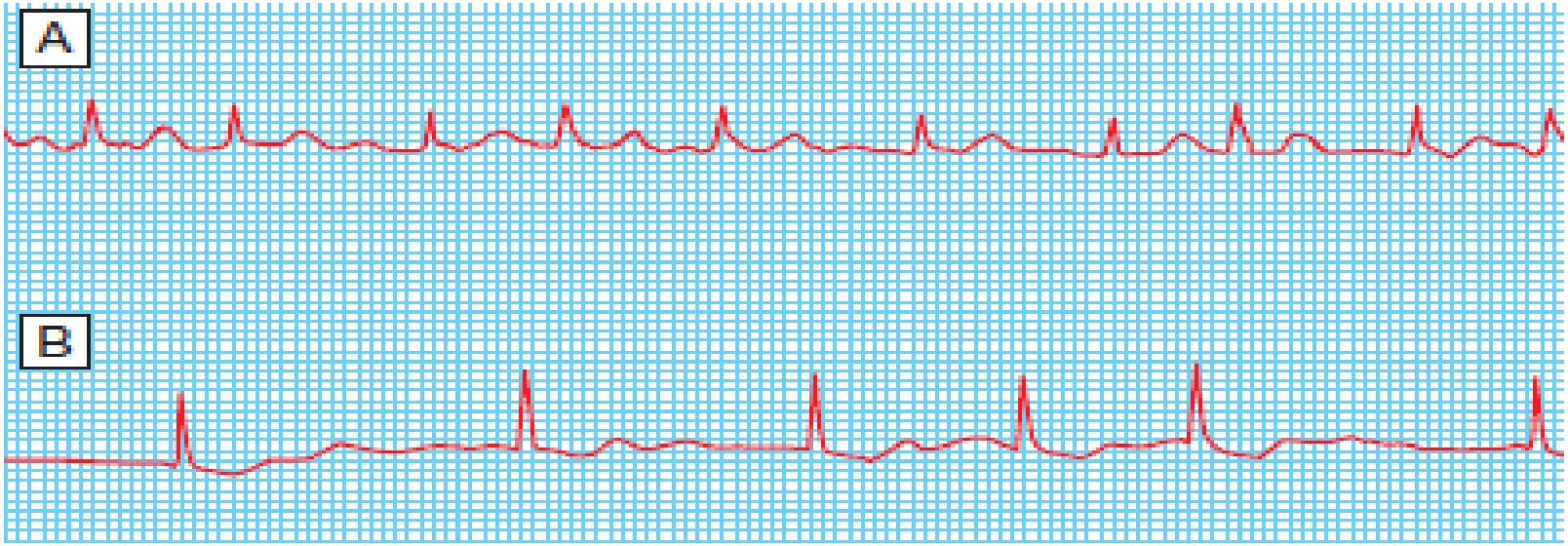
- Lasts for **more than one year and cardioversion either has not been attempted or has failed**

- It also describes patients where a decision has been made to no longer pursue a rhythm control strategy

Signs and Symptoms

- Asymptomatic
- Incidental finding (30% of cases)
- Palpitation, breathlessness and fatigue.
- Patient with poor ventricular function or valve disease, it may precipitate or aggravate cardiac failure because of Loss of atrial function and heart rate control.
- Fall BP may cause lightheadedness and chest pain.

ECG changes

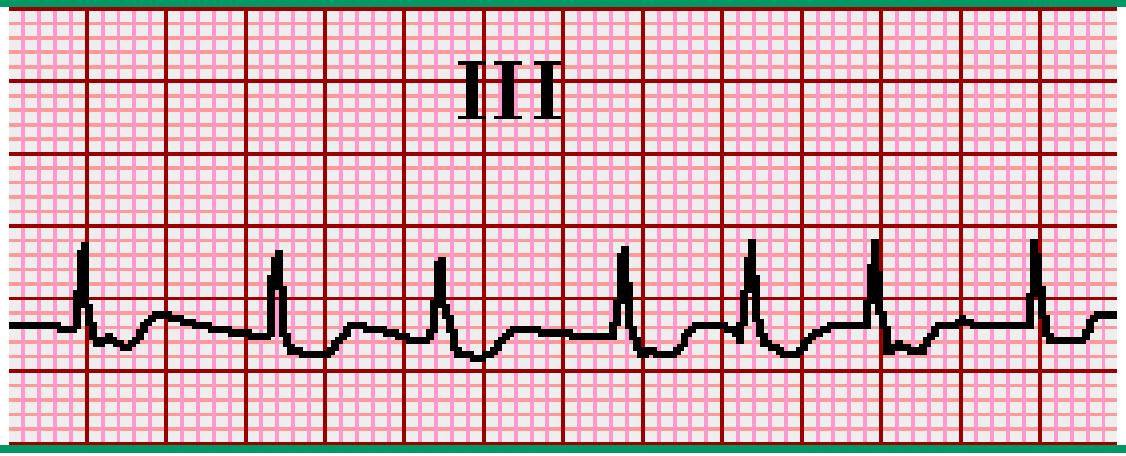


The QRS complexes are irregular and there are no P waves.

A. There is usually a fast ventricular rate, e.g. between 120 and 160/min, at the onset of atrial fibrillation.

B. In chronic atrial fibrillation, the ventricular rate may be much slower, due to the effects of medication and AV nodal fatigue

Atrial fibrillation



Here is an example/review of what an EKG may look like in the previous patient with afib. Atrial activity is rapid (>320 bpm) without any organized activity, and of various amplitudes. No discrete P waves are seen in this image. There is also irregularly irregular ventricular response which shows up as variable R-R intervals. The Ventricular response is usually 130-200.

Management of AF

Diagnosis

History, Physical Ex,Labs

- Underlying heart disease, thyroid, alcohol

ECG

CXR

- Pneumonia

Echocardiogram

Approach

- When AF complicates an acute illness (e.g. chest infection, pulmonary embolism), effective **treatment of the primary disorder** will often restore sinus rhythm
- Otherwise, the main objectives are
  - restoration of sinus rhythm (when possible),
  - prevention of recurrent AF,
  - optimisation of the heart rate during periods of AF,
  - reduction of the risk of thromboembolism, and
  - treatment of underlying disease

Paroxysmal AF

- Occasional attacks that are well tolerated do not necessarily require treatment

- β-blockers as first-line therapy if troublesome, and if associated with IHD, HTN and CCF

  - Beta-blockers reduce the ectopic firing that normally initiates AF

- Class IC drugs, such as propafenone or flecainide, also effective (C/I: CAD or Left Ventricular dysfunction)

Catheter ablation

- When anti-arrhythmic drug therapy is ineffective or causes side-effects
- Used to **disconnect the pulmonary veins from the LA electrically**, preventing ectopic triggering of AF
- Lines of conduction block can be created within the atria to prevent re-entry
- Prevents AF in approximately 70% of patients although a repeat procedure is sometimes required
- Associated with a risk of cardiac tamponade or embolic stroke

Persistent and Permanent AF

There are two options:-

- Rhythm control

- Attempting to restore and maintain sinus rhythm

- Rate control

- Accepting that AF will be permanent and using treatments to control the ventricular rate and to prevent embolic complications

Rhythm control

- Attempts successful if AF present for < 3 months, the patient is young and there is no important structural heart disease
- Pharmacologic or by electro cardioversion
- Electrical cardioversion successful in 75%, but relapse frequent
- Immediate DC cardioversion after iv heparin if AF present for < 48 hrs.

Cont…

- Infusing IV flecainide (2 mg/kg over 30 minutes, maximum dose 150 mg) is a safe alternative to electrical cardioversion if no underlying structural heart disease

- In other situations, DC cardioversion should be deferred until

  - On warfarin, with an INR > 2.0 for a minimum of 4 weeks, and

  - Any underlying problems, such as HTN or alcohol excess, have been eliminated

- Anticoagulation should be maintained for at least 3 months following successful cardioversion

Rate control

- If sinus rhythm cannot be restored , treatment should be directed at maintaining an appropriate heart rate
- Digoxin, β-blockers or rate-limiting CCB such as verapamil, diltiazem

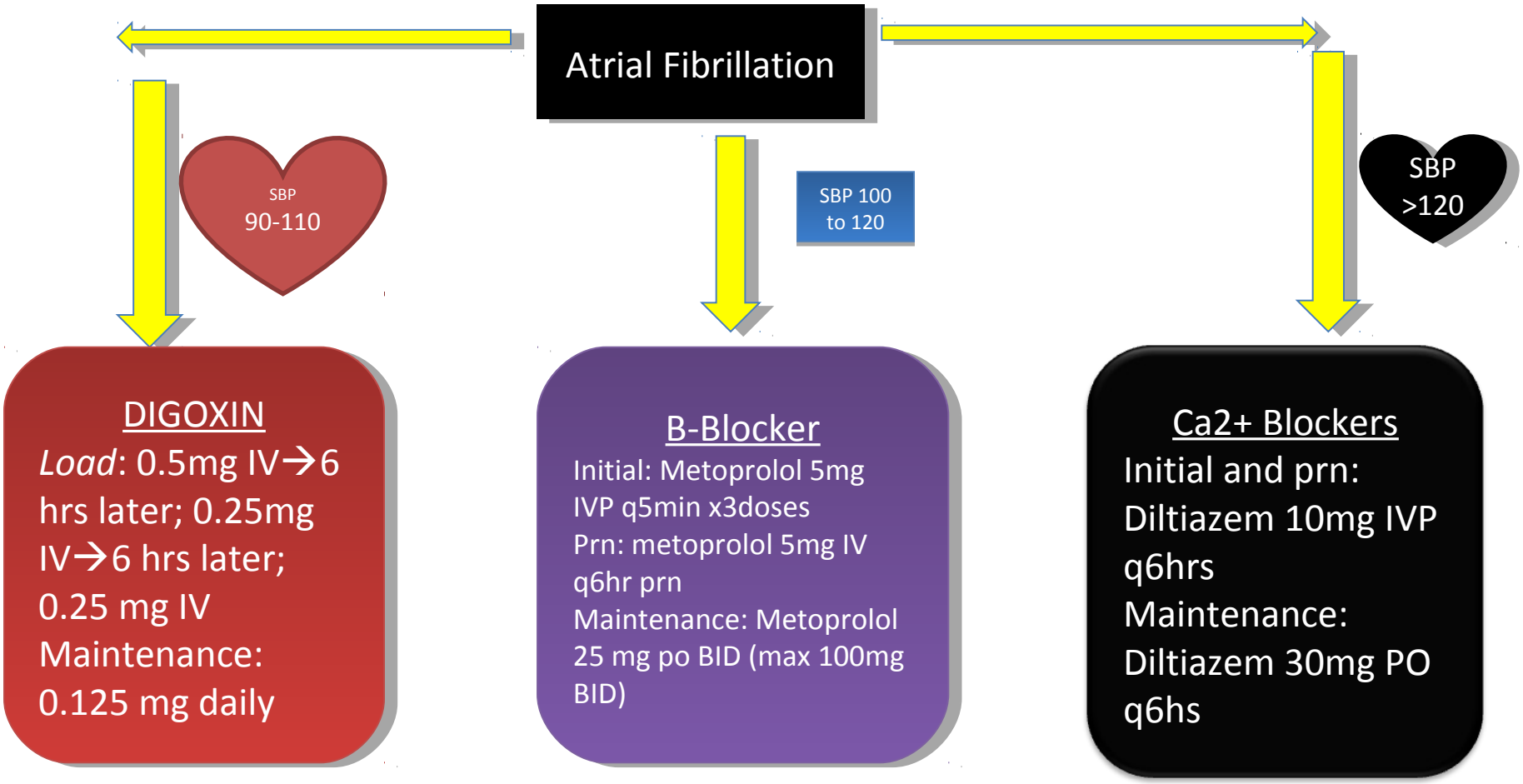
  - reduce the ventricular rate by increasing the degree of AV block

  - β-blockers or rate-limiting CCB often more effective than digoxin in controlling HR during exercise and have additional benefits in pt's with HTN and structural heart disease

Cont..

- Combination therapy (e.g. digoxin + atenolol) is often advisable
- Cather ablation and permanent pacemaker is useful in poorly controlled symptoms “pace and ablate” strategy

RATE CONTROL



16.23 CHA_2DS_2-VASc stroke risk scoring system for non-valvular atrial fibrillation

	Parameter	Score
C	Congestive heart failure	1 point
H	Hypertension history	1 point
A_2	Age ≥ 75 years	2 points
D	Diabetes mellitus	1 point
S_2	Previous stroke or transient ischemic attack (TIA)	2 points
V	Vascular disease	1 point
A	Age 65–74 years	1 point
Sc	Sex category female	1 point
	Maximum total score	9 points

Annual stroke risk

0 points = 0% (no prophylaxis required)

1 point = 1.3% (oral anticoagulant recommended in males only)

2+ points = > 2.2% (oral anticoagulant recommended)

CHA_2DS_2-VASc Score	Estimated Annual Stroke Rate[a]
0	0
1	1.3%
2	2.2%
3	3.2%
4	4.0%
5	6.7%
6–9	>9%

Anticoagulants	Mechanism	Excretion	Dosing Considerations	Risk/Benefit
Warfarin	Vitamin K antagonist	Liver	Adjusted to INR 2–3 Days to therapeutic effect Multiple drug/food interactions (e.g., amiodarone)	Major hemorrhage: 1% per year Intracranial hemorrhage: 0.1–0.6% per year Risk of bleeding increases with INR >3.5 Inexpensive
Dabigatran[b]	Thrombin inhibitor	Kidney		
		CCr >30 mL/min	150 mg bid	Onset of action within hours
		CCr 15–30 mL/min	75 mg bid P-glycoprotein substrate (inducers – rifampin, reduce concentration) (inhibitors – amiodarone, verapamil, dronedarone, quinidine), Proton pump inhibitors may reduce absorption	No reversal agent for bleeding
Rivaroxaban	Xa inhibitor	Kidney	P-glycoprotein substrate	No reversal agent for bleeding
		CCr ≥50 mL/min	20 mg daily	
		CCr 15–50 mL/min	15 mg daily	
Apixaban	Xa inhibitor	Kidney and liver	P-glycoprotein substrate	No reversal agent for bleeding
		Cr >1.5 mg/dL	2.5 mg bid	

16.24 HAS-BLED bleeding risk scoring system for patients receiving oral anticoagulation

	Parameter	Score
H	Hypertension; current systolic blood pressure >160 mmHg	1 point
A	Abnormal liver function (cirrhosis OR bilirubin > twice upper limit of reference range or transaminases > three times upper limit of reference range	1 point
	Abnormal renal function (creatinine >200 µmol/L (2.26 mg/dL)	1 point
S	Stroke history	1 point
B	Bleeding: prior major event	1 point
L	Labile INR on warfarin	1 point
E	Elderly: age ≥65 years	1 point
D	Drugs:	
	Use of antiplatelet drugs	1 point
	High alcohol consumption	1 point
	Maximum total score	9 points

HAS-BLED score of ≥3 points requires close patient monitoring

References:

- Davidson's Principle and Practice of medicine, 23rd edition
- Harrison's Principles of Internal Medicine, 20th edition
- Uptodate

AF Association Global AF Aware Week | 18 - 24 November 2019

THANK YOU