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ATRIAL FIBRILLATION

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ATRIAL FIBRILLATION

DEFINITION

Atrial fibrillation (AF) is a common arrhythmia defined as a supraventricular tachyarrhythmia with rapid fibrillatory p-waves and an irregular ventricular response.

The ventricular response depends on:
- atrio-ventricular (AV) node properties
- conducting tissues
- vagal versus sympathetic tone
- accessory pathways
- drugs

The heart rate is usually 250-350 beats/min. An irregularly irregular rhythm is seen on electrocardiogram (ECG) with absence of P waves.

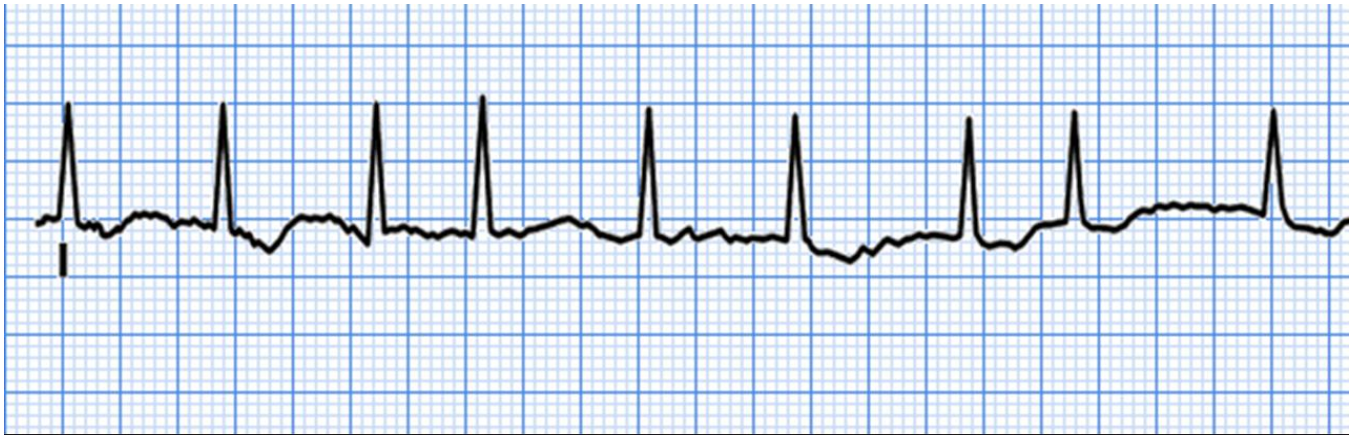


Figure 1

EPIDEMIOLOGY

Affects 5 million people globally [1]
Prevalence increases with increasing age [2, 3]
In those over the age of 80 years, 10% are affected [1]

CLASSIFICATION

Atrial fibrillation is classified according to the duration of episodes [4]

Term	Definition
Paroxysmal AF	Terminates within 7days, spontaneously or with treatment
Persistent AF	Continuous AF sustained > 7 days
Long standing persistent AF	Continuous AF>12 months
Permanent AF	Agreement to stop attempts at restoring sinus rhythm

AETIOLOGY [5]

Can be divided into cardiac and non cardiac causes

Common cardiac causes
- Hypertension
- Ischaemic heart disease
- Rheumatic heart disease
- Valvular heart disease
- Cardiac surgery

Less common cardiac causes
- Cardiomyopathy
- Pericardial disease

Common non cardiac causes
Respiratory
- Chronic obstructive pulmonary disease (COPD)
- Pulmonary hypertension
- Pulmonary embolism
- Lung cancer

Endocrine
- Hyperthyroidism
- Obesity

Immunology
- Acute infections

Other
- Alcohol abuse
- Narcotic abuse

CLINICAL PRESENTATION [4]

History and examination
- Presence and nature of symptoms
- Establish type of AF
- Medical treatment for AF
- Presence of associated underlying disease

Investigations
1. ***ECG***
- Verify AF
- Morphology of p-wave
- Left ventricular hypertrophy
- Bundle branch block
- Previous myocardial infarction

2. ***Transthoracic Echo***
- Left atrial thrombus

- Chamber sizes
- Left ventricular hypertrophy
- Pericardial disease

3. *Blood investigations* looking for organ dysfunction of the:
- Thyroid
- Kidneys
- Liver

4. *Other*
- 6 minute walk test
- Exercise testing
- Holter or event monitoring
- Electrophysiological studies

PATHOPHYSIOLOGY

AF occurs when structural and/or electrophysiological abnormalities alter atrial tissue to promote abnormal impulse formation and/or propagation [4].

Traditional thinking attributes the pathophysiology of AF to fibrosis and muscular atrophy of the left atrium from pathological remodeling however emerging evidence points towards inflammatory pathways.

Pro-inflammatory cytokines and hormones such as ATII, TNF alpha, IL 6 and IL 8 trigger myocardial dysfunction by activating local leukocytes and cardiac fibroblasts. The resulting cardiomyocytic apoptosis triggers massive cardiac proliferation and differentiation leading to dysfunction of connexin and ion channels [6].

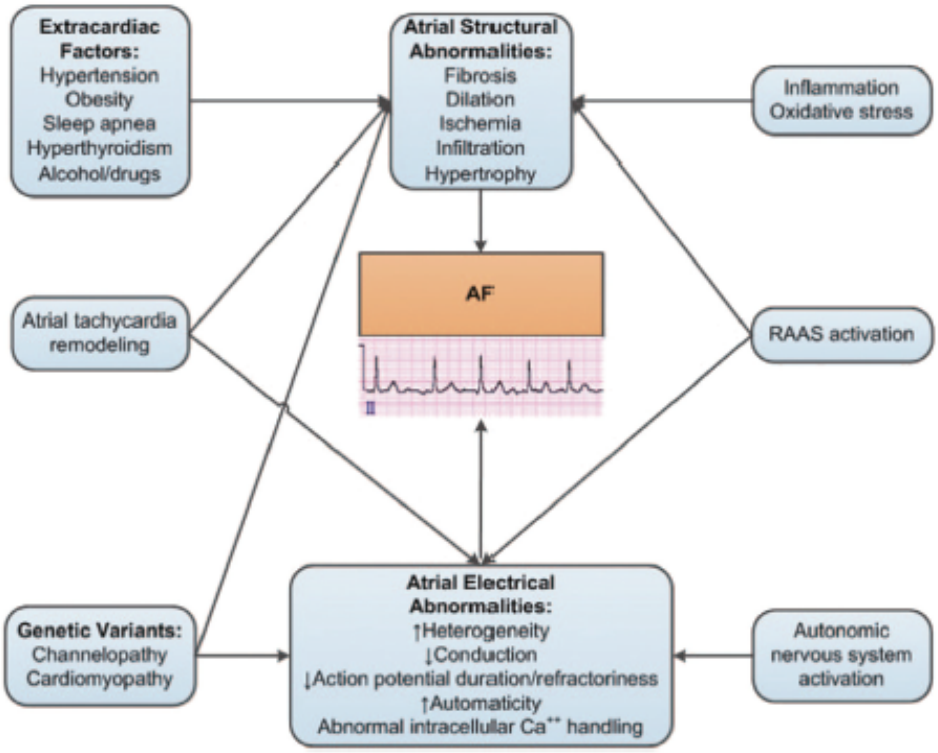


Figure 2

PERIOPERATIVE MANAGEMENT

Aims
1. Severity of symptoms
2. Anticoagulation
3. Establish bleeding risk
4. Anticoagulants
5. Perioperative bridging
6. Rate or rhythm control strategy

Severity of symptoms [7]

The Canadian Cardiovascular Society (CCS) Severity of Atrial Fibrillation (SAF) Scale is used to assess symptom severity.

The severity of atrial fibrillation is assessed→
Firstly by identifying the presence of the following symptoms:
- Palpitations
- Dyspnoea
- Dizziness
- Syncope
- Chest pain
- Fatigue

Secondly by determining if AF is associated with symptoms and if this impacts on the patient's function.

The CCS-SAF SCALE:

Class	Definition
0	Asymptomatic
1	Minimal effect on quality of life • Minimal/infrequent symptoms • Single AF episode
2	Minor effect on quality of life • Awareness of symptoms • Rare episodes
3	Moderate effect on quality of life • Moderate awareness of symptoms on most days • More frequent episodes
4	Severe impact on quality of life • Unpleasant symptoms • Frequent/symptomatic episodes • Congestive cardiac failure

Anticoagulation

Thrombotic risk
The need for anticoagulation is based on ischaemic stroke risk using the following scoring systems: $CHADS_2$ and CHA_2D_2VASc

Comparison of $CHAD_2$ and CHA_2DS_2-VASc Scores [4]

Definition and Scores for $CHADS_2$ and CHA_2DS_2-VASc		Stroke Risk Stratification With the $CHADS_2$ and CHA_2DS_2-VASc Scores	
	Score		Adjusted Stroke Rate (% per y)
$CHADS_2$		$CHADS_2$*	
Congestive HF	1	0	1.9
Hypertension	1	1	2.8
Age ≥75 y	1	2	4.0
Diabetes mellitus	1	3	5.9
Stroke/TIA/TE	2	4	8.5
Maximum score	6	5	12.5
		6	18.2
CHA_2DS_2-VASc		CHA_2DS_2-VASc†	
Congestive HF	1	0	0
Hypertension	1	1	1.3
Age ≥75 y	2	2	2.2
Diabetes mellitus	1	3	3.2
Stroke/TIA/TE	2	4	4.0
Vascular disease (prior MI, PAD, or aortic plaque)	1	5	6.7
Age 65–74 y	1	6	9.8
Sex category (i.e., female sex)	1	7	9.6
Maximum score	9	8	6.7
		9	15.20

AF is an independent risk factor for stroke [8]
AF is responsible for ¼ of all strokes [8]

$CHADS_2$ score

- Most widely used
- Low risk= 0, intermediate risk= 1-2, high risk= ≥3
- Guidelines:
  - Low risk : aspirin
  - Intermediate risk : aspirin/anticoagulation
  - High risk : anticoagulation
- 65% characterised as intermediate risk with no clear guideline
- Evidence suggested that aspirin does not reduce stroke risk in low risk group [9] and that warfarin is superior to aspirin in the intermediate risk group [10]
- Undertreatment of patients at significant risk of thromboembolism

CHA_2D_2VASc

- Low risk= 0, intermediate risk= 1, high risk= ≥2
- Better identifies low risk group
- Fewer patients in the intermediate risk group
- Well validated
- Performs better than $CHADS_2$
- Incorporated into international guidelines

Bleeding risk [11]

Bleeding is the most feared complication of anticoagulation use. The risk for bleeding has to be considered in conjunction with the anticoagulants that the patient is on.

HAS-BLED score

- Simple tool used for bleeding risk assessment
- Correctable risk factors for bleeding picked up
- Allows for periodic assessment
- Individual INR control taken into account
- Validated
- Favourable performance in comparison to other such scores

HAS-BLED Score

RISK FACTOR	**SCORE**
Hypertension	1
Abnormal liver/renal function	1 or 2
Stroke	1
Bleeding tendency	1
Labile INR	1
Age > 65 years	1

Low bleeding risk = 0-2
High bleeding risk = ≥3

Anticoagulants

i. Warfarin

Pros

- Strong evidence for stroke prevention in AF
- Superior to aspirin for reduction of ischaemic stroke and reduces all-cause mortality by 26% in AF [12]
- Superior to aspirin in the elderly with no difference in bleeding risk [13]
- Superior to dual antiplatelet therapy with comparable bleeding risk [14]

Cons

- Slow onset and offset
- Long half life
- Narrow therapeutic window
- Unpredictable pharmacokinetics and pharmacodynamics
- Numerous drug and dietary interactions
- Frequent monitoring needing regular clinic visits

ii. Novel oral anticoagulant (NOACs)

Dabigatran

- Oral prodrug
- Direct and reversible inhibitor of thrombin
- Onset of action 0.5-4 hours
- Half-life 17 hours
- Eliminated via kidneys

RE-LY Trial

Dabigatran versus warfarin in patients with atrial fibrillation [15], a randomised controlled trial (RCT).

Population	:	18 000 patients with non valvular AF $CHADS_2$ of ≥1 or >65 years with coronary artery disease
Intervention	:	Dabigatran 110mg or 150mg
Control	:	Dose adjusted warfarin
Outcome	:	Stroke, Systemic embolism and major bleeding

Low dose dabigatran was non-inferior to warfarin for stroke prevention and superior to warfarin for major bleeding with a 20% relative risk reduction. High dose dabigatran was superior to warfarin as there was a 34% relative risk reduction in stroke or systemic embolism with similar bleeding rates. Dabigatran was not as well tolerated as warfarin because of dyspepsia in the intervention group.

Rivaroxiban

- Reversible, direct factor Xa inhibitor
- Half-life 9-12 hours
- Fast onset of action

ROCKET AF [16]

Double blind RCT

Population	:	14 000 patients with non valvular AF History of stroke, TIA or non CNS embolism or Two independent risk factors for stroke
Intervention	:	Rivaroxiban
Control	:	Dose adjusted warfarin
Outcome	:	Stroke, Systemic embolism, Major bleeding

Non-inferior for the prevention of stroke.
Similar bleeding risk (HR 1.03, 95% CI 0.96-1.11) but Rivaroxiban had less fatal bleeding and intracranial haemorrhage.

Apixaban

- Reversible direct factor Xa inhibitor
- Rapid onset of action
- Half-life 12 hours
- Cleared by faeces

ARISTOTLE Trial [17]

Double blind RCT

Population	:	>18 000 patients with non valvular AF
Intervention	:	Apixaban 5mg
Control	:	Dose adjusted warfarin
Outcome	:	All cause stroke, Systemic embolism, Major bleeding

Apixaban

- Superior to warfarin for primary outcomes (HR 0.79, 95% CI 0.66-0.95)
- Benefit in all-cause mortality compared to Warfarin (HR 0.89, 95% CI 0.8-0.99)
- 31% reduction in major bleeding
- Better tolerated than Warfarin

Limitations of NOACs

Patient factors

- Difficult to assess compliance
- Dabigatran and apixaban need to be administered twice daily which may compromise compliance

Drug factors

- Monitoring anticoagulant effects problematic
- Unknown factors include long term safety profile and cost effectiveness

Surgical factors

- Method of anticoagulant bridging therapy not fully established

Approach to NOACs

Continue with warfarin in those patients:

- Well established on warfarin in whom INR control is good
- Suffered a previous MI or with dyspepsia – tolerant of and compliant with warfarin

Change to Dabigatran in those with:

- History of cerebral haemorrhage
- Unable to comply with INR monitoring

Bleeding during NOAC therapy:

- Stop drug
- Supportive measures – transfusion of blood and blood products

If these fail [18]→

- Recombinant FVIIa
- Activated Prothrombin Complex Concentrate (aPCC)
  May have potential but more data required to support this

Dabigatran can be removed by activated charcoal/haemodialysis if rapid reversal required.

Perioperative bridging

Perioperative Bridging Anticoagulation in Patients with Atrial Fibrillation [19]

RCT

Population	:	1884 patients >18 years old with chronic AF or atrial flutter On warfarin therapy for 3 months or longer INR 2-3 $CHADS_2 \geq 1$ Elective surgery
Intervention	:	Dalteparin 100 iu/kg bd
Control	:	No bridging
Primary Outcomes	:	Arterial thromboembolism Major bleeding
Secondary Outcomes	:	Death Acute myocardial infarction Deep vein thrombosis Pulmonary embolism Minor bleeding
Time	:	30 days

Study Outcomes for BRIDGE Trial [19]

Outcome	No Bridging (N=918)	Bridging (N=895)	P Value
	number of patients (percent)		
Primary			
Arterial thromboembolism	4 (0.4)	3 (0.3)	0.01*, 0.73†
Stroke	2 (0.2)	3 (0.3)	
Transient ischemic attack	2 (0.2)	0	
Systemic embolism	0	0	
Major bleeding	12 (1.3)	29 (3.2)	0.005†
Secondary			
Death	5 (0.5)	4 (0.4)	0.88†
Myocardial infarction	7 (0.8)	14 (1.6)	0.10†
Deep-vein thrombosis	0	1 (0.1)	0.25†
Pulmonary embolism	0	1 (0.1)	0.25†
Minor bleeding	110 (12.0)	187 (20.9)	<0.001†

* P value for noninferiority.
† P value for superiority.

Arterial thromboembolism
No bridging group 0.4%
Bridging group 0.3%

Major bleeding
No bridging group 1.3%
Bridging group 3.2%
No fatal bleeds

Secondary outcomes
Minor bleeding increased in the bridging group
No differences in secondary outcomes

Conclusion : Benefit in forgoing bridging
Critique : High risk surgery not represented

Rate or rhythm control

Rate control
Aims to control the ventricular rate without correction to sinus rhythm
Preferred strategy for AF:

- >48 hours/unknown duration until adequate coagulation achieved or intracardiac thrombus ruled out [20]
- initial approach for patients in whom structural remodeling is expected to progress with development to permanent AF

Target rate control

- Symptomatic : HR < 80 beats/min
- Asymptomatic with preserved LV function : HR < 100 beats/min

Drugs of choice

- Beta blocker (BB)
- Non dihydropyridine calcium channel blockers (CCB)
  Contra-indicated in decompensated AF
- In the critically ill patient use amiodarone

Consider AV nodal ablation with permanent ventricular pacing if drug therapy does not suffice and rhythm control cannot be achieved.

If pre-excitation is present in conjunction with AF then the following drugs should not be administered:

- Digoxin
- Non dihydropyridine CCB
- Amiodarone

Medication Dosages [4]

	Intravenous Administration	Usual Oral Maintenance Dose
Beta blockers		
Metoprolol tartrate	2.5-5.0 mg IV bolus over 2 min; up to 3 doses	25–100 mg BID
Metoprolol XL (succinate)	N/A	50–400 mg QD
Atenolol	N/A	25–100 mg QD
Esmolol	500 mcg/kg IV bolus over 1 min, then 50-300 mcg/kg/min IV	N/A
Propranolol	1 mg IV over 1 min, up to 3 doses at 2-min intervals	10–40 mg TID or QID
Nadolol	N/A	10–240 mg QD
Carvedilol	N/A	3.125–25 mg BID
Bisoprolol	N/A	2.5–10 mg QD
Nondihydropyridine calcium channel antagonists		
Verapamil	0.075–0.15 mg/kg IV bolus over 2 min; may give an additional 10.0 mg after 30 min if no response, then 0.005 mg/kg/min infusion	180–480 mg QD (ER)
Diltiazem	0.25 mg/kg IV bolus over 2 min, then 5–15 mg/h	120–360 mg QD (ER)
Digitalis glycosides		
Digoxin	0.25 mg IV with repeat dosing to a maximum of 1.5 mg over 24 h	0.125–0.25 mg QD
Others		
Amiodarone*	300 mg IV over 1 h, then 10–50 mg/h over 24 h	100-200 mg QD

Rhythm control

Employed in:

- recent-onset atrial fibrillation not spontaneously reverting to sinus rhythm
- symptomatic despite ventricular rate control

Principles:

- Prevention of thromboembolism
- Direct current cardioversion
- Pharmacological cardioversion

Prevention of thromboembolism

- Anticoagulate with Warfarin or NOACs for 3 weeks before and 4 weeks after cardioversion
- If immediate cardioversion required, anticoagulate as early as possible and continue for 4 weeks
- Following cardioversion, long term anticoagulation is determined by thromboembolic risk

Direct cardioversion

- Recommended for pre-excitation and haemodynamic instability

Pharmacological cardioversion

Commonly used drugs

- Flecainide
- Propafenone
- Amiodarone

Medication Dosages [4]

Drug	Usual Doses	Exclude/Use With Caution	Major Pharmacokinetic Drug Interactions
Vaughan Williams class IA			
Disopyramide	• Immediate release: 100–200 mg once every 6 h • Extended release: 200–400 mg once every 12 h	• HF • Prolonged QT interval • Prostatism, glaucoma • Avoid other QT interval–prolonging drugs	• Metabolized by CYP3A4: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin)
Quinidine	• 324–648 mg every 8 h	• Prolonged QT interval • Diarrhea	• Inhibits CYP2D6: ↑concentrations of tricyclic antidepressants, metoprolol, antipsychotics; ↓efficacy of codeine • Inhibits P-glycoprotein: ↑digoxin concentration
Vaughan Williams class IC			
Flecainide	• 50–200 mg once every 12 h	• Sinus or AV node dysfunction • HF • CAD • Atrial flutter • Infranodal conduction disease • Brugada syndrome • Renal or liver disease	• Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7%–10% of population) and renal excretion (dual impairment can ↑↑plasma concentration)
Propafenone	• Immediate release: 150–300 mg once every 8 h • Extended release: 225–425 mg once every 12 h	• Sinus or AV node dysfunction • HF • CAD • Atrial flutter • Infranodal conduction disease • Brugada syndrome • Liver disease • Asthma	• Metabolized by CYP2D6 (inhibitors include quinidine, fluoxetine, tricyclics; also genetically absent in 7%–10% of population)—poor metabolizers have ↑beta blockade • Inhibits P-glycoprotein: ↑digoxin concentration • Inhibits CYP2C9: ↑warfarin concentration (↑INR 25%)
Vaughan Williams class III			
Amiodarone	• Oral: 400–600 mg daily in divided doses for 2–4 wk; maintenance typically 100–200 mg QD • IV: 150 mg over 10 min; then 1 mg/min for 6 h; then 0.5 mg/min for 18 h or change to oral dosing; after 24 h, consider decreasing dose to 0.25 mg/min	• Sinus or AV node dysfunction • Infranodal conduction disease • Lung disease • Prolonged QT interval	• Inhibits most CYPs to cause drug interaction: ↑concentrations of warfarin (↑INR 0%–200%), statins, many other drugs • Inhibits P-glycoprotein: ↑digoxin concentration
Dofetilide	• 125–500 mcg once every 12 h	• Prolonged QT interval • Renal disease • Hypokalemia • Hypomagnesemia • Diuretic therapy • Avoid other QT interval–prolonging drugs	• Primary renal elimination involving glomerular filtration and active tubular secretion: verapamil, HCTZ, cimetidine, ketoconazole, trimethoprim, prochlorperazine, and megestrol are contraindicated; discontinue amiodarone at least 3 mo before initiation
Dronedarone	• 400 mg once every 12 h	• Bradycardia • HF • Long-standing persistent AF/flutter • Liver disease • Prolonged QT interval	• Metabolized by CYP3A: caution with inhibitors (e.g., verapamil, diltiazem, ketoconazole, macrolide antibiotics, protease inhibitors, grapefruit juice) and inducers (e.g., rifampin, phenobarbital, phenytoin) • Inhibits CYP3A, CYP2D6, P-glycoprotein: ↑concentrations of some statins, sirolimus, tacrolimus, beta blockers, digoxin
Sotalol	• 40–160 mg once every 12 h	• Prolonged QT interval • Renal disease • Hypokalemia • Hypomagnesemia • Diuretic therapy • Avoid other QT interval–prolonging drugs • Sinus or AV nodal dysfunction • HF • Asthma	• None (renal excretion)

ANAESTHETIC CONSIDERATIONS

Patient Factors-Recommendations for Specific Treatment Groups [4]

Cardiac Disease

Acute coronary syndromes

Urgent cardioversion if new onset AF present with:

- Haemodynamic compromise
- Ongoing ischaemia
- Inadequate rate control

No heart failure or haemodynamic instability treat with:

- BB
- CCB

Treat with amiodarone if the patient has the following:

- Severe LV dysfunction
- Heart failure
- Haemodynamically unstable

Heart failure

BB or CCB recommended as treatment
Acute rate control with no pre-excitation use:

- Digoxin
- Amiodarone

AV nodal ablation with ventricular pacing when drug measures have failed

Wolf Parkinson White and Pre-Excitation Syndromes

Cardiovert if haemodynamically unstable with procainamide IV or ibutilide
Catheter ablation of accessory pathway if symptomatic

Respiratory Disease

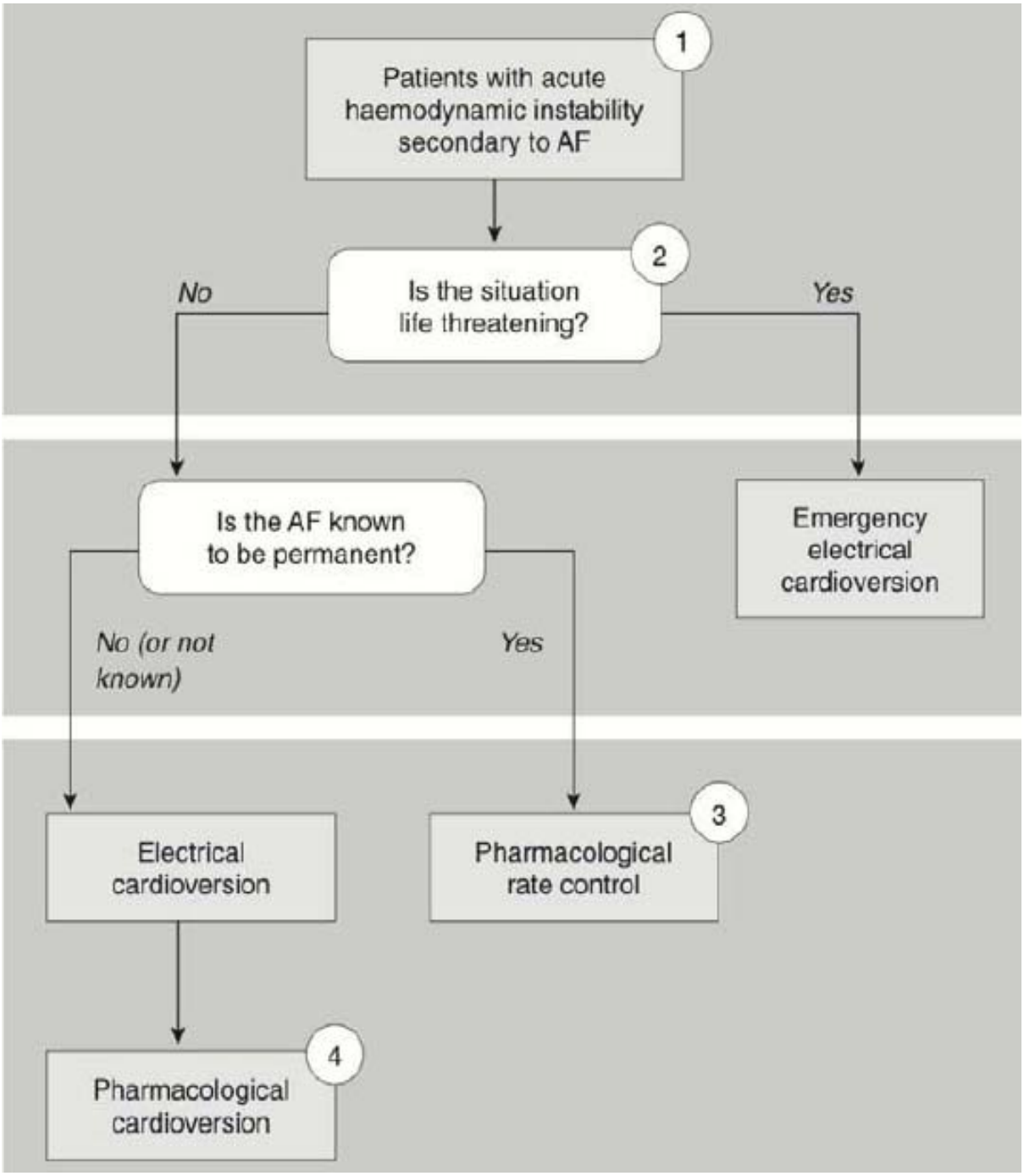
In COPD, CCB recommended for ventricular rate control

Endocrine

Hyperthyroidism:
First line: BB
CCB if BB contra-indicated

Anaesthetic Factors

Haemodynamically Unstable AF



Clinical Guideline for the Management of Post-operative Atrial Fibrillation, Royal Cornwall Hospitals NHS Trust, Clinical Guideline Template [21]

Highest risk:

- Ventricular rate > 150 bpm
- Ongoing chest pain

Aim:

Unacceptably high ventricular rate-control rate
New onset AF secondary to cardiac abnormalities-rhythm control

Management:

Electrical cardioversion
Direct current cardioversion has the highest overall success rate [22, 23].
Patient must be fasted and under general anaesthetic. Cardioversion must be synchronized with the R wave. Biphasic external defibrillators have lower energy requirements and better efficacy than monophasic waveforms [24, 25].

The amount of energy needed for initial attempts are controversial.
Initial attempts→
- Monophasic : start at 200J
- Biphasic : 100-200J

Regarding biphasic energy, the Best AF Trial [26] showed that biphasic energy of 200J is more successful in the overweight and obese patients in comparison to 100J for first shock success however there was no difference in first shock success regardless of whether 100 or 200J were used in the patients with a normal body mass index.

If cardioversion is unsuccessful after shock delivery then incremental higher energy repeat direct cardioversion is attempted until
- Arrhythmia aborts
- Decision is made to abandon direct current cardioversion

ACLS Defibrillation Protocol with Zoll Rectilinear Biphasic Waveform-AHA/ERC Guidelines 2005 recommends the following energy requirements for synchronized cardioversion:

Monophasic			**ZOLL Biphasic**		
200J	300J	360J	100J	150J	200J

Patients with implantable devices:
- Place electrode paddle approximately 8cm from battery
- Use anteroposterior paddle position
- Use low energy to avoid damage to device
- Interrogate pacemaker following cardioversion

Pharmacological cardioversion
Drug of choice:
Structural heart disease – amiodarone
No structural heart disease – Flecainide or Propafenone

Amiodarone
Loading dose : 5mg/kg over 20 minutes
Infusion : 900mg over 24 hours via a central line
Maximum dose : 300mg bolus

Flecainide
Loading dose : 2mg/kg over 10-30 minutes
Infusion : 1.5mg/kg/hour for 1 hour followed by
100-250ug/kg/hour for up to 24 hours, if required
Maximum dose : 600mg over 24 hours

Propofenone
Loading dose : 150mg 8hrly-300mg 8hrly

Pharmacological rate control (60-80 beats/min)
Drugs of choice:
- Nondihydropyridine calcium channel blockers (CCB)
- B-Blockers

CCBs
- Verapamil 5-10mg iv over 2 minutes, can repeat very 5-10min

B-Blockers
- Esmolol 50-200ug/kg/min IV
- Metoprolol 5mg IV repeated after 2-5min with maximum dose of 15mg
- more effective than CCBs for controlling ventricular response during AF [27]
- faster conversion to sinus rhythm [28]

Surgical Factors

Carotid Endarterectomy (CEA)
Incidence of perioperative stroke 0.7-1.5% and subsequent death is 0.5% [29].
Patients with AF are at higher risk for having perioperative stroke and death [30].
Atrial fibrillation is associated with increased risk of perioperative stroke and death from carotid endarterectomy [22].

Retrospective cohort study:
Population : 20 022 patients with occlusion and stenosis of precerebral arteries requiring CEA
Outcome : Stroke and death
Time : In hospital stay

Results

Stroke 0.94%
Death 0.29%
Stroke and death 1.15%
7.2% with AF
17.8% of combined outcome had AF

Conclusion : Patients with AF had over twice the odds of death/stroke compared to patients without AF
Critique : AF patients may have had higher stroke rate and death with medical Management

Postoperative AF [PAF]

Most frequent complication following cardiovascular surgery [31].
Commonly develops on the second postoperative day.
High incidence in the elderly [32].

Risk Factors:

Pre-operative Factors
- Enlarged left atrium
- Left ventricular hypertrophy
- Hypertension
- Diabetes
- Obesity
- Metabolic syndrome

Intra-operative Factors
- Myocardial ischaemia
- Damage to atrium
- Acute volume change
- Venous cannulation
- Endotracheal tube

Postoperative Factors
- Increase in afterload
- Increase in preload
- Hypotension
- Inflammation
- Electrolyte abnormalities

Preventing PAF:

The following drugs have been shown to prevent PAF
- B-Blockers
- Amiodarone
- Statins
- Corticosteroids

Controversial in PAF prevention
- Magnesium
- Off-pump coronary artery bypass grafting
- Atrial pacing

NOVEL THERAPIES

Left atrial appendage exclusion

This serves as an option for the management of devastating bleeds whereby a Nitinol framework is delivered percutaneously via a trans-septal puncture to the left atrial appendage under echocardiographic and fluoroscopic guidance [33].
Full endothelialisation occurs after a few months.
Adverse effects include
- Pericardial effusion
- Stroke

For paroxysmal AF:

1. Radiofrequency ablation versus cryoablation

An electrical barrier between pulmonary vein and left atrium created via

- Radiofrequency or
- Cryoenergy

General anaesthesia is required
No difference in efficacy or safety outcomes [34] shown
Single procedure : 65% of patients are AF free for 6 months
Additional procedure : 74% of patients are AF free for 12 months

2. Visually guided laser balloon catheter ablation

The pulmonary vein ostium visualised through an endoscopic transplant balloon.
Laser therapy is then used for ablation.
61% of patients are AF free at 12 months [35].
It is not approved for use in the U.S.

For paroxysmal and persistent AF:

Pulmonary vein isolation versus electrogram-guided ablation strategy [36]

Pulmonary vein isolation (PVI) versus high frequency ablation showed no difference between the groups**.**
In the PVI group with paroxysmal AF-69% of patients were AF free at 6 months
In the high frequency ablation source group – 65% of patients were AF free at 6 months
In the persistent AF group-59% of patients were free of recurrent AF after a single procedure. Fewer complications were seen in the high frequency source ablation group

For persistent atrial fibrillation

Surgical ablation approaches [37]

The source of AF isolated by atriotomy scar.
There is better freedom from AF compared to no ablation.

CONCLUSION

AF is a common arrhythmia affecting a wide range of patients.
Treatment requires a multi-disciplinary approach.

Principles of management include

- Establishing the severity of symptoms
- Establishing the need for and adequacy of anticoagulation
- Deciding between a rate or rhythm control management strategy

Perioperative considerations include

- Establishing stroke and bleeding risk
- Consideration of the need for bridging
- Re-establishing anticoagulation

There are many new advances being made to optimise AF treatment in an endeavour to improve mortality and morbidity outcomes.

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