Review Article

Cardiac tachyarrhythmias and anaesthesia: General principles and focus on atrial fibrillation

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ABSTRACT

Cardiac tachyarrhythmias are encountered commonly during the perioperative period and need to be promptly identified and appropriately managed by the anaesthesiologist. This review intends to highlight important aspects of these tachyarrhythmias and explore a temporal relationship between common medications employed in the perioperative period and their causation. Mechanisms of initiation of tachyarrhythmias, drugs that can trigger those, as well as their diagnosis and management, are also parts of the current review. Cardiac tachyarrhythmias may not always require treatment, and sometimes, aggressive management can trigger more serious types of arrhythmias. A thorough understanding of these tachyarrhythmias and their pathogenesis enables adopting a more objective approach, eschewing risks of inappropriate or unnecessary management strategies. We performed a MEDLINE search using combinations of MeSH terms such as 'cardiac', 'arrhythmias', 'anaesthesia', 'perioperative', 'tachyarrhythmias' and 'anaesthetic implications'. We reviewed the relevant publications with regard to cardiac tachyarrhythmias occurring in the perioperative period.

Key words: Anaesthesia, anaesthetic implications, arrhythmias, cardiac, perioperative, tachyarrhythmias

INTRODUCTION

Cardiac arrhythmias are one of the most perturbing complications in the perioperative period.[1] Several pharmacological agents and non-pharmacological stimuli[2] during anaesthesia could result in cardiac arrhythmias. It is important for an anaesthesiologist to understand the pathophysiology and management of common cardiac arrhythmias. This review would focus on perioperative tachyarrhythmias during non-cardiac surgery and their management, with a special focus on atrial fibrillation (AF).

CLASSIFICATION OF TACHYARRHYTHMIAS

The cardiac impulse is generated from sinus node; it traverses through atrioventricular (AV) node, His bundle and Purkinje fibres and reaches the ventricles. Tachyarrhythmias occur due to abnormal impulse generation from the sinoatrial (SA) node, AV node or ventricles. In addition, abnormal impulse generation may be associated with abnormal conduction. Thus, tachyarrhythmias could be classified based on the rhythm (regular or irregular), site of origin (supraventricular or ventricular) and complexes on electrocardiogram (ECG) (narrow or broad complex). The classification of tachyarrhythmias into narrow or broad complex is based on the duration of the QRS complex and is shown in Figure 1.

GENERAL CAUSES OF TACHYARRHYTHMIAS DURING ANAESTHESIA

Several physical stimuli and many pharmacological agents could result in tachyarrhythmias. These factors may be classified according to patient, pathology, position, pharmacology and procedure [Table 1].

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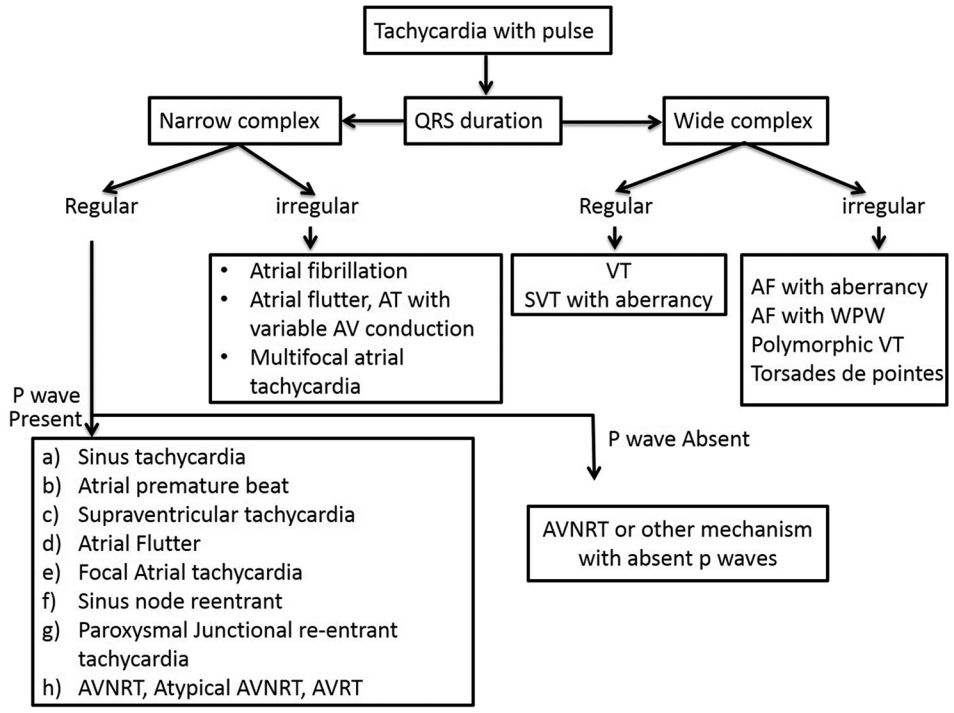

Figure 1: Classification of tachyarrhythmias. (AT:Atrial tachcardia; VT: Ventricular tachycardia; SVT:Supraventricular tachycardia; AF: Atrial fibrillation; WPW:Wolff-Parkinson-White; AVNRT:Atrio-ventricular nodal re-entrant tachycardia; AVRT: Atrioventricular re-entrant tachycardia

Table 1: Causes of tachyarrhythmias under anaesthesia

Patient	Pathology	Position	Pharmacology	Procedure
Elderly History of arrhythmias	Cardiac disease Renal disease CNS disorder COPD Electrolyte disturbances	Prone position Lateral position	Premedication Induction agent Muscle relaxant Analgesic Anaphylaxis Vasopressor Inotropic agent Reversal agent Local anaesthetics	Thoracotomy Ophthalmic surgery General anaesthesia Major fluid shift CVP catheter cannulation PA catheter insertion Cardiac surgery ESWL[3]

COPD – Chronic obstructive pulmonary disease; CNS – Central nervous system; CVP – Central venous pressure; ESWL – Extracorporeal shock wave lithotripsy; PA – Pulmonary artery

Most of the inhalational anaesthetics are associated with cardiac arrhythmias. Use of halothane during induction as well as maintenance of anaesthesia is associated with several dysrhythmias including atrial, nodal or ventricular tachycardia.[4] Hence, halothane induction in children has been largely replaced with sevoflurane which is safer.[5] During ophthalmic surgery, many types of arrhythmias are observed due to oculocardiac reflex.

Atropine is one of the commonly used drugs to treat bradycardia during general anaesthesia and also following spinal anaesthesia. It increases the heart rate leading to sinus tachycardia generally, but other arrhythmias including AV junctional tachycardia or ventricular bigeminy may also occur. These rhythms may convert to sinus tachycardia without any treatment.[6]

One of the risk factors for the development of torsades de pointes is QTc prolongation. Duma *et al*. observed that QTc prolongation occurs both after spinal and general anaesthesia which persists post-operatively.[7] Sevoflurane anaesthesia is also associated with prolongation of QTc interval.[8] There was no difference between sevoflurane and desflurane with respect to QTc prolongation.[9] Use of many other pharmacological agents could increase the QTc interval [Table 2]. Anaesthesiologists should be aware of these agents and manage their perioperative consequences accordingly.

Thoracic surgery is associated with an increased incidence of perioperative AF. In high-risk patients, prophylactic administration of amiodarone or magnesium sulphate could reduce the incidence of AF.[10]

MANAGEMENT OF TACHYARRHYTHMIA

The most common causes for sinus tachycardia such as light planes of anaesthesia, lack of adequate analgesia, dehydration or wearing off of muscle relaxation should

be ruled out before further differential diagnoses are considered. The management should include simultaneous assessment of underlying causes for the particular arrhythmia. These could be due to several factors as listed in Table 1.

A schematic means to approach diagnosis of cardiac tachyarrhythmias is shown in Figure 1. Conventionally, cardiologists always require a 12-lead ECG to confirm the exact nature of arrhythmias. However, in the operating room, it is not always possible to get such an ECG done. Anaesthesiologists would have to make the diagnosis by looking at the ECG monitor. Some manufacturers would allow the grid lines and simultaneous ECG from other leads to be displayed on the monitor.

The presence of a P-wave will differentiate supraventricular tachycardia (SVT) from junctional tachycardia. In the presence of tachycardia, the P-wave may sometimes be overlapping on the T-wave. On many occasions, changing the sweep speed of ECG (from 50 to 25 mm/sec) may help identify the P-wave. However still, on several occasions, one would find the P-wave merging with the previous T-wave and thus unidentifiable even after altering sweep speeds. If the origin of impulse is above the AV node and below the SA node, the P-wave morphology might be biphasic. However, the absence of P-wave will not reliably rule out SVT. For example, rarely, one might encounter SVTs along with abnormal pathways. Alternatively, several of the vagal manoeuvres could be attempted. A few common cardiac tachyarrhythmias are depicted in Figure 2 to serve as a reference.

Table 2: Drugs prolonging QTc interval

Type of drug	Example
Class Ia anti-arrhythmic	Quinidine, disopyramide, procainamide
Class Ic anti-arrhythmic	Flecainide
Class III anti-arrhythmic	Sotalol, amiodarone
Antipsychotics	Droperidol, haloperidol, phenothiazine, thioridazine, pimozide, quetiapine, risperidone, zotepine
Serotonin reuptake inhibitors	Fluoxetine, paroxetine, sertraline
Macrolide antibiotics	Erythromycin, clarithromycin, azithromycin
5-HT1 agonist	Zolmitriptan, naratriptan
Antimalarial agents	Halofantrine
Antihistamines	Terfenadine
Prokinetic agents	Cisapride

ANTI-ARRHYTHMIC DRUGS CLASSIFICATION AND MECHANISMS OF ACTION (VAUGHAN WILLIAMS)

All anti-arrhythmic drugs act by modifying the action potential, which results from alteration of ion channels [Table 3]. The normal action potential has five phases. The most commonly used classification of anti-arrhythmics is the one proposed by Vaughan Williams [Table 3]. They are classified depending upon their action on the four phases of the cardiac cycle.

TREATMENT

Patients with haemodynamic instability or those with signs of poor tissue perfusion would be treated straight away with electrical therapy. In patients who

Table 3: Mechanisms of action and Vaughan Williams classification of anti-arrhythmic drugs

Class	Mechanism of action	Drugs	Side-effects
Class Ia	Blocks fast sodium channel, depresses phase 0 depolarisation, prolongs action potential	Quinidine Procainamide Disopyramide	Cinchonism, cramping and nausea, enhancement of digitalis toxicity lupus-like syndrome Negative inotropic effect
Class Ib	Sodium channel blockers	Lidocaine Phenytoin Mexiletine	
Class Ic	Most potent sodium channel blockers, markedly depress phase 0 depolarisation	Flecainide Propafenone Moricizine	Life-threatening VT β-blocking and Ca^{++}- channel blocking activity can worsen heart failure
Class II	Beta-blockers	Propranolol, esmolol, timolol, metoprolol, atenolol	Bradycardia, reduced exercise capacity, heart failure, hypotension, and AV nodal conduction block, bronchoconstriction
Class III	Block potassium efflux	Amiodarone, sotalol, ibutilide, dofetilide	Proarrhythmic, can cause bradycardia and atrioventricular block
Class IV	Slow calcium channel blocker	Verapamil, diltiazem	Excessive bradycardia, impaired electrical conduction, depressed contractility
Class V	Variable mechanism	Adenosine Digoxin Magnesium sulphate	Flushing, hypotension, chest pain, sense of impending doom, bronchospasm Toxicity Hypotension

AV – Atrioventricular; VT – Ventricular tachycardia

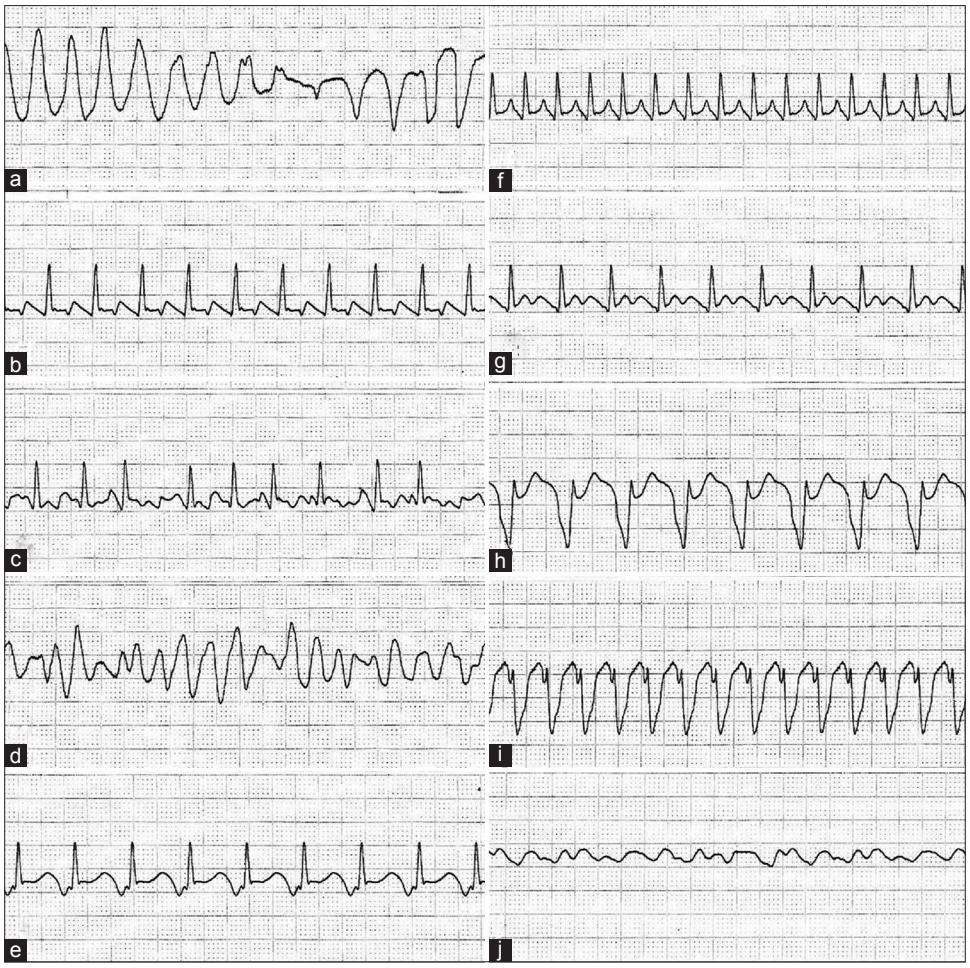

Figure 2: Common arrhythmia strips (a) torsades, (b) atrial flutter, (c) atrial fibrillation, (d) coarse ventricular fibrillation, (e) junctional tachycardia, (f) atrial tachycardia, (g) sinus tachycardia, (h) slow ventricular tachycardia, (i) fast ventricular tachycardia, (j) fine ventricular fibrillation

are otherwise stable, the clinical benefits of prompt cardioversion in the emergency department are less clear. The case in favour of emergency cardioversion in these patients hinges on the presumption that a regular sinus rhythm is preferable to the chaotic activity instituted by a tachyarrhythmia like AF.[11] However the basis for such observations is principally from anecdotal patient experiences and a single non-randomised trial.[12] Patients with structural heart diseases or refractory ventricular arrhythmias in spite of standard anti-arrhythmia management may need catheter ablation. Alternatively, implantable cardioverter defibrillator could be used preoperatively to manage these conditions. Thoracic epidural anaesthesia might reduce the incidence of ventricular arrhythmias by blocking cardiac sympathetic fibres.[13] Stellate ganglionectomy is another option to treat sympathetically mediated VT.[14] In patients undergoing thoracic surgeries, thoracic epidural infusion is believed to reduce the episodes of tachycardia.[15] Whenever there is a need for temporary pacing, the cardiologist may be called for intervention.

VAGAL MANOEUVRES

The first line of treatment for SVTs is to attempt vagal manoeuvres such as Valsalva and carotid sinus massage (CSM). These manoeuvres will be of help only if the arrhythmia is dependent on the AV node. Valsalva manoeuvre is done by increasing the intrathoracic pressure to 30–40 mmHg. The Valsalva is generally divided into four separate phases: Phase 1, onset of straining and the beginning of an increase in intrathoracic pressure with glottic closure; Phase 2, persistent straining and maintenance of the increased intrathoracic pressure; Phase 3, release of breath-holding and glottic pressure with a sudden drop in the intrathoracic pressure and Phase 4, sudden increase in cardiac output and aortic pressure.

The cardioversion rate with this is less as is the requirement for other pharmacological management. Hence, a postural modification of the Valsalva manoeuvre has been evaluated. This included the

conventional semi-recumbent position during the initiation of the Valsalva manoeuvre, followed by supine position and leg raise immediately after its release.[16]

CAROTID SINUS MASSAGE

This is one of the vagal manoeuvres used in the treatment of SVTs. A recent review concluded that CSM should be considered first in the treatment of SVT.[17] CSM is performed by applying a steady pressure over right or left carotid sinus for 5–10 s. The complications of CSM include stroke. Hence, one has to rule out the presence of carotid bruit clinically before applying CSM. Alternatively, this could be ruled out by an ultrasound examination. Recently, a prospective crossover trial concluded that ultrasound-guided CSM is a suitable alternative to classical CSM.[18] An algorithmic approach to the acute treatment of regular SVT of unknown origin is shown in Figure 3.

PERIOPERATIVE MANAGEMENT OF ATRIAL FIBRILLATION

AF is one of the most common arrhythmias in non-cardiac surgeries, especially in the elderly. AF occurs when there is an abnormal impulse formation or propagation from atrium due to structural or electrophysiological abnormalities. This could be classified as acute or chronic from the perspective of decision-making for management. Many modalities have been tried to prevent the incidence of AF including prophylactic beta-blockers, amiodarone, corticosteroids and magnesium. AF of acute onset is usually treated. An ECG with typical varying RR interval and absence of P-waves is diagnostic of AF.

CLASSIFICATION OF ATRIAL FIBRILLATION

AF is classified according to the onset and duration. Paroxysmal AF is defined as AF which terminates spontaneously or with any intervention within 7 days of onset. If it persists for >7 days, it is defined as persistent AF. Long-standing persistent AF continues for >12 months.

TREATMENT OF ATRIAL FIBRILLATION

The treatment of AF is focussed on five domains.[19] These include (a) presence of hemodynamic instability, (b) presence of precipitating factors, (c) anticoagulation, (d) rate control and (d) rhythm control. All these domains may not be applicable as such for perioperative AF that occurs for the first time. First onset of AF should be treated aiming for the restoration of haemodynamic stability while focussing on optimising the precipitating factors, rate control and rhythm control. Anticoagulation is not the initial focus intraoperatively.

RATE VERSUS RHYTHM CONTROL

Pharmacological rate control is achieved with beta-blockers, digoxin, verapamil, diltiazem or a combination of drugs. Few antiarrhythmic agents such as amiodarone, dronedarone and sotalol have both rate control as well as rhythm control properties. The suggested algorithm for rate control is given in Figure 4. Even though the target for rate control is not very clear, it would generally be recommended to achieve a rate of <110 beats/min. The frequently used drugs include metoprolol and esmolol, followed by diltiazem and verapamil. These drugs are preferred over digoxin due to their rapid onset of action and also

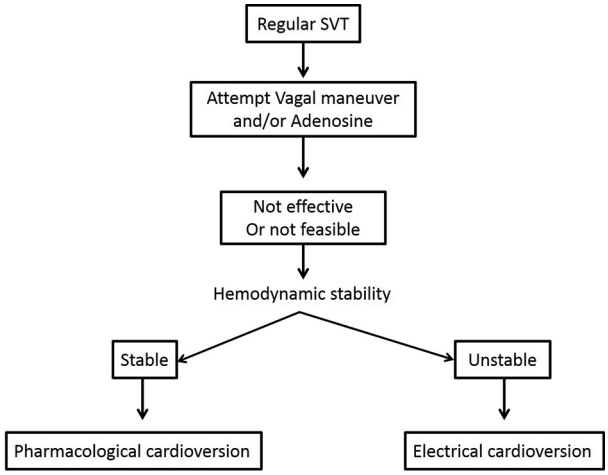

Figure 3: Acute treatment of regular supraventricular tachycardia (SVT) of unknown origin

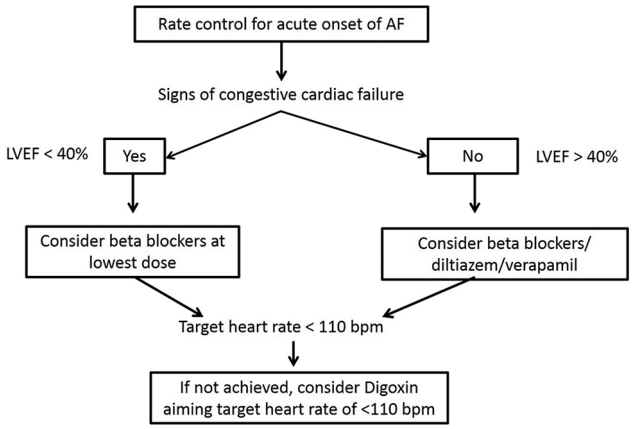

Figure 4: Acute heart rate control of atrial fibrillation. (AF:Atrial Fibrillation; LVEF:Left ventricular ejection fraction)

the effectiveness at high sympathetic tone. In addition, anaesthesiologists are more familiar with these drugs than digoxin when it comes to intravenous use.

RHYTHM CONTROL

Maintaining the sinus rhythm in AF is essential to preventing long-term complications of thrombus formation and stroke. It is believed that maintaining the sinus rhythm might improve the outcome. Many trials have compared the rate control only versus both rate and rhythm control and found no significant differences between the two approaches.[20-24] Hence, rhythm control may be attempted if rate control does not improve clinical symptoms. Amiodarone is the commonly used agent for restoring sinus rhythm. Before achieving rhythm control, anticoagulation of the patient should be considered. A basic algorithm for rhythm control of acute AF is shown in Figure 5. Table 4 shows drugs which may be used for rate or rhythm control of AF.

RISK STRATIFICATION FOR EMBOLIC COMPLICATIONS IN ATRIAL FIBRILLATION

Risk stratification scores for individuals with non-valvular AF have been described to help clinicians estimate the adjusted stroke risk in these individuals and guide recommendations for antithrombotic therapy. $CHADS_2$ is a simple, well-validated points-based risk assessment tool widely used to assess individual patient risk for stroke. The risk factors included in the $CHADS_2$ score include congestive heart failure (1 point), hypertension (1 point), age >75 years (1 point), diabetes mellitus (1 point) and prior stroke/transient ischemic attack (TIA)/thromboembolism (2 points).[25]

The more recently introduced CHA_2DS_2-VASc score has shown better predictive value in stroke-risk assessment. The CHA_2DS_2-VASc score has been developed in an attempt to increase the predictive value for risk of stroke, especially in low-risk patients.[26] CHA_2DS_2-VASc is now preferred over $CHADS_2$ in the latest European 2012 and American 2014 guidelines.[27,28] CHA_2DS_2-VASc identifies 'major' risk factors, comprising stroke/TIA/thromboembolism and age ≥75 years (2 points each), and 'clinically relevant non-major' risk factors, comprising congestive heart failure, hypertension, diabetes mellitus, age 65–74 years, female gender and vascular disease (1 point each).[29] Recommendations to initiate treatment in patients based on the $CHADS_2$ and CHA_2DS_2-VASc have also been laid down.[26,27,30]

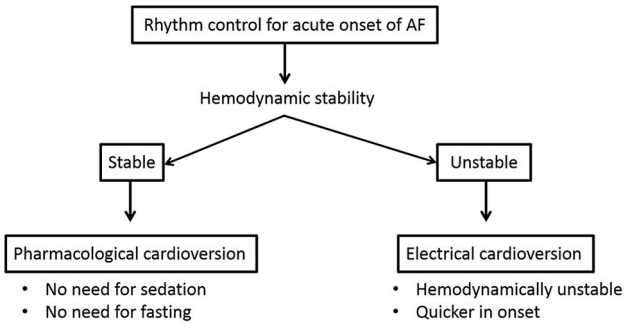

Figure 5: Rhythm control for acute onset of atrial fibrillation

Table 4: Drugs used for rate and rhythm control of acute atrial fibrillation

Drug	Bolus	Infusion
Esmolol	0.5 mg/kg over 1 min	0.05-0.25 mg/kg/min
Diltiazem	0.25 mg/kg over 2 min	5-15 mg/h
Verapamil	0.075-0.15 mg/kg If no response after 30 min, give 10 mg	0.005 mg/kg/min infusion
Digoxin	0.5 mg	0.75-1.5 mg over 24 h in divided doses
Amiodarone	300 mg diluted in 250 ml of 5% dextrose infused over 30-60 min	10-50 mg/h over 24 h

ANTICOAGULATION

Patients with acute onset of AF, who are haemodynamically unstable, can undergo electrical cardioversion without anticoagulation. In patients with AF for >48 h, it is recommended to start oral anticoagulation for 3 weeks before cardioversion.[31-33] If patient needs early cardioversion, transoesophageal echocardiography should be done to rule out left atrial thrombus before attempting cardioversion. Vitamin K antagonists were the first and commonly used anticoagulants in AF. However, they need frequent monitoring of international normalised ratio to titrate the dose to achieve the therapeutic range. Once this is achieved, they are effective in preventing stroke. The use of newer non-Vitamin K antagonists such as direct thrombin inhibitors (dabigatran) and factor Xa inhibitors (apixaban, edoxaban and rivaroxaban) are increasing as they have a more predictable effect.

Newer non-Vitamin K antagonists inhibit the action of certain specific coagulation factors and are named accordingly as rivaroxaban, apixaban and edoxaban, which oppose the action of factor Xa, whereas dabigatran etixalate is a thrombin inhibitor [Figure 6].[34] These drugs score over warfarin in having comparatively

shorter half-lives, predictable pharmacokinetics and pharmacodynamics, with less requirement for rigorous monitoring [Table 5].[35] The current evidence seems to suggest that the newer non-Vitamin K antagonists are identical to warfarin in prophylaxis of stroke or systemic embolisation in AF without escalated rates of intracranial bleed or mortality.[35-40] These drugs have also demonstrated a better safety profile and efficacy in comparison to low molecular weight heparin for the prevention of deep venous thrombosis, following major orthopaedic surgery.[40-44] However, perioperative management of patients on these newer non-Vitamin K antagonists may be challenging in the settings of emergency surgery, trauma, bleeding or drug overdose.

In patients receiving warfarin, bridging therapy with heparin may be recommended when the risk of thrombosis during withdrawal for elective surgery is high, but no clear guidelines state how to proceed in those at moderate risk of thrombosis. Bridging may be reserved for patients with mechanical heart valves and high risk for thromboembolism, history of recent stroke, TIA or $CHADS_2$ score 5 or 6.[45] However, a recent randomised trial seems to suggest that bridging therapy may not provide added benefit and on the contrary may increase perioperative blood loss.[46] In patients on oral non-Vitamin K antagonists and a high risk of thrombosis, bridging may be initiated 12 h after the last dose.[47]

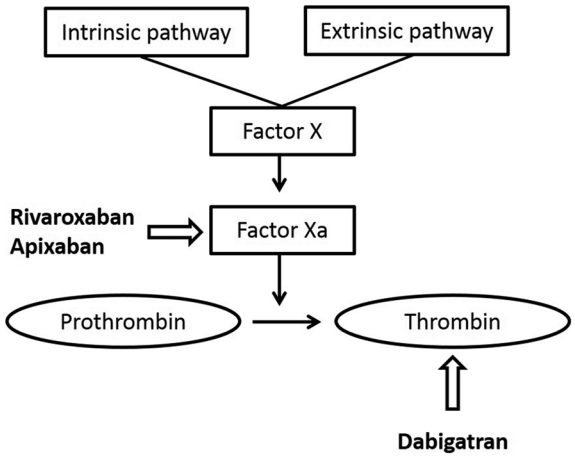

Figure 6: Sites of action of new oral Vitamin K antagonists

Reversal of warfarin effects can be achieved by Vitamin K as it can result in switching on of endogenous synthesis of Vitamin K-dependent clotting factors. If rapid reversal of warfarin effects is desired, as for impending surgery or for the management of major warfarin-induced bleeding, the options are between prothrombin complex concentrates (PCCs) and frozen plasma. PCC can reverse warfarin effects in no >6 h. Frozen plasma may be used as an alternative if PCC is either unavailable or contraindicated. Recommendations for warfarin reversal are shown in Table 6.[48]

ATRIAL FIBRILLATION IN PREGNANCY

Pregnant patients with pre-existing cardiac disease are more prone for AF. Among the options for rate control, placental transfer to foetus should be kept in mind. The safety of beta-blockers and calcium channel blockers are not well documented. Atenolol may cause growth retardation; hence, it is avoided. The first-line therapy for rate control in AF would be digoxin, followed by other drugs. For rhythm control, there are no randomised controlled trials. As rhythm control agents are associated with severe adverse foetal side effects, they are used only in emergency conditions. Electrical cardioversion is attempted where facilities are available to monitor foetal heart rate and also to intervene to manage foetal distress.

Table 5: Comparison of warfarin and newer non-Vitamin K antagonists

Variables	Warfarin	Dabigatran	Rivaroxaban	Apixaban
Mechanism of action	Vitamin K-dependent clotting factor inhibitor	Thrombin inhibitor	Factor Xa inhibitor	Factor Xa inhibitor
Bioavailability	80%-100%	3%-7%	60%-100%	60%-70%
Plasma protein binding	99%	35%	90%	87%
Plasma half-life	20-60 h	12-16 h	5-13 h	8-15 h
Peak level after ingestion	4 h	1.25-3 h	2-4 h	3-4 h
Renal clearance	Minimal	85%	30%	30%
Hepatic degradation	Almost entirely	15%	70%	70%
Permeability-glycoprotein transporter interaction	-	++	+	+
CYP3A4 interaction	++	-	+	+
Stop before regional anaesthesia or elective surgery	5 days	5 days	3 days	3 days
Check before block	PT/INR	Thrombin time, aPTT	Anti Xa, PT/INR	Anti Xa

PT – Prothrombin time; INR – International normalised ratio; aPTT – Activated partial thromboplastin time

Table 6: Recommendations for warfarin reversal

Urgency of procedure	Recommendations
Emergency (0-6 h)	PCC based on weight and INR/ FFPs based on weight and INR Vitamin K1 slow IV
Urgency (6-12 h)	Vitamin K1 slow IV
Non-urgent (24-36 h)	Vitamin K1 slow IV

PCC – Prothrombin complex concentrate; FFPs – Fresh frozen plasmas; INR –International normalised ratio; IV-intravenous

FUTURE PERSPECTIVES

Future pharmacological therapy of AF might include atrial selective modulation of ionic currents to avoid ventricular side effects and targeting other mechanisms involved in AF generation and maintenance with multifunctional compounds, with particular attention paid to avoid adverse effects in patients with left ventricular dysfunction. These include late or persistent sodium current inhibition, potassium channel activation, ultra-rapidly activating potassium current blockade, acetylcholine-regulated potassium current inhibition, sodium-calcium exchanger inhibition and hyperpolarisation-activated, cyclic nucleotide-gated pacemaker 'funny current' blockade, as provided by ivabradine. Owing to the heterogeneous mechanisms involved in tachyarrhythmia development, compounds with multiple sites of action including dronedarone, vernakalant and resveratrol could demonstrate better efficacy in their management.

CONCLUSIONS

The differentiation between a benign dysrhythmia and one that could lead to sudden haemodynamic decompensation is absolutely vital. Choice of anaesthetic agents might be of relevance in mitigating incidences of tachydysrhythmias. In patients who are haemodynamically unstable, emergent cardioversion or defibrillation is the management modality of choice, with anti-arrhythmic medications as indicated, to prevent recurrence of the particular tachyarrhythmia. In other patients who are haemodynamically stable, medications would be used first up. However, it must be remembered that management of cardiac tachyarrhythmias in the perioperative period does not always involve use of anti-arrhythmics, although their use should not be delayed when indicated.

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Conflicts of interest
There are no conflicts of interest.

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