



## CARDIAC TACHYARRHYTHMIAS AND ANAESTHESIA

### Anaesthesiology

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### ABSTRACT

Cardiac tachyarrhythmias are recurrent or chronic and in some cases life-threatening conditions. Cardiac tachyarrhythmias are encountered commonly during the perioperative period and need to be promptly identified. 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.

### KEYWORDS

#### INTRODUCTION

Cardiac arrhythmias are one of the most perturbing complications in the perioperative period.[1] It is important to acknowledge and understand the impact of cardiac tachyarrhythmias on the patient.

Several pharmacological agents and non-pharmacological stimuli [2] during anaesthesia could result in cardiac arrhythmias.

#### Causes

Several physical stimuli and many pharmacological agents could result in tachyarrhythmias. 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.[4,5]

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.[6, 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. 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

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.

#### Treatment

Patients with haemodynamic instability or those with signs of poor tissue perfusion would be treated straight away with electrical therapy. In patients who 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.[15,16] Whenever there is a need for temporary pacing, the cardiologist may be called for intervention.

#### 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.

#### 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.

### 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<sub>2</sub> 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<sub>2</sub> 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<sub>2</sub>DS<sub>2</sub>-VASc score has shown better predictive value in stroke-risk assessment. The CHA<sub>2</sub>DS<sub>2</sub>-VASc score has been developed in an attempt to increase the predictive value for risk of stroke, especially in low-risk patients.[26] CHA<sub>2</sub>DS<sub>2</sub>-VASc is now preferred over CHADS<sub>2</sub> in the latest European 2012 and American 2014 guidelines.[27,28] CHA<sub>2</sub>DS<sub>2</sub>-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<sub>2</sub> and CHA<sub>2</sub>DS<sub>2</sub>-VASc have also been laid down.[26,27,30]

### 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<sub>2</sub> 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] 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.

### 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

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.

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