

https://dspace.org/

Perioperative dysrhymias

Brian Gardner

https://hdl.handle.net/10539/35352
SCOPUS

Downloaded from a DSpace repository. https://dspace.org/

Southern African Journal of Anaesthesia and Analgesia. 2021;27(6 Suppl 1):S136-140
https://doi.org/10.36303/SAJAA.2021.27.6.S1.2699
Open Access article distributed under the terms of the
Creative Commons License [CC BY-NC 3.0]
http://creativecommons.org/licenses/by-nc/3.0

South Afr J Anaesth Analg
ISSN 2220-1181 EISSN 2220-1173
© 2021 The Author(s)
FCA REFRESHER COURSE

Perioperative dysrhythmias

B Gardner

Department of Anaesthesia, Helen Joseph Hospital, University of the Witwatersrand, South Africa
Corresponding author, email: *brian.gardner@wits.ac.za*

Keywords: perioperative dysrhythmias, perioperative cardiovascular complication

Introduction

Dysrhythmias are the most frequently occurring perioperative cardiovascular complication.[1] The fear is that these dysrhythmias could deteriorate into haemodynamic instability or death. The incidence of sudden perioperative cardiac death in non-cardiac surgery patients is reported to be approximately 0.03–0.05% which is, thankfully, very low.[2,3] A multitude of dysrhythmias may present in the perioperative period. It is important to be able to predict, prevent, diagnose and manage these as far as possible. In the event of a malignant dysrhythmia occurring, it is vital to have an approach to rescue the patient. Advanced Cardiac Life Support (ACLS) and Paediatric Advanced Life Support (PALS) training remain crucial for perioperative physicians. This overview will focus on the following aspects:

1. Normal rhythm generation in pacemaker cells and myocytes
2. Sources of abnormal rhythms
3. Classification of anti-arrhythmic agents
4. Changes to the 2020 American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiovascular care (ECC)
5. Highlights of the 2020 AHA scientific statement on drug-induced arrhythmias
6. The most common perioperative dysrhythmia (perioperative atrial fibrillation)
7. The dysrhythmia which can be worsened by anaesthesia agents (long QT syndrome)

Normal rhythm generation

Understanding normal and abnormal rhythm generation is necessary to inform the clinical management of dysrhythmias.

Pacemaker cells

Impulse generation is mainly stimulated by calcium influx.[4] Cardiac pacemaker cells are myocytes which have lost their ability to contract and are instead responsible for impulse formation. They have an unstable resting potential and can depolarise spontaneously. The main site is in the sinoatrial node (SA node). Normally the SA node fires the fastest and overrides the impulse generation of other secondary sites, such as the atrioventricular node (AV node) or areas within the Purkinje system. With SA node failure, these secondary sites can take over the impulse generation function, although at a slower rate.

At resting state, sodium and calcium are mostly extracellular while potassium is intracellular, and the transmembrane electrical potential is approximately -60 mV.

The pacemaker action potential cycle starts with the slow influx of sodium and calcium into the cell until the transmembrane potential reaches -40 mV. (This phase is called Phase 4.)

As soon as the potential reaches -40 mV, voltage-gated calcium channels are opened, leading to the rapid influx of calcium and rapid transmembrane depolarisation to approximately +10 mV. (This is called Phase 0.) Once depolarisation is complete, the potassium channels open and an efflux of potassium causes repolarisation to a membrane potential of approximately -60 mV again. (This is called Phase 3.) There is no Phase 1 or Phase 2 in pacemaker cell action potential generation. Sodium–potassium and sodium–calcium exchange pumps then restore the original electrolyte balance.[1]

Myocytes

Impulse generation is stimulated by sodium influx.[4] Myocytes do not normally depolarise unless these are stimulated by an outside action potential. The normal resting transmembrane electrical potential is approximately -90 mV. When an action potential stimulates the myocyte, the transmembrane potential reduces to a critical level of -60 mV. At this stage, the fast sodium channels open and sodium rushes into the cell causing rapid depolarisation to a potential of approximately +30 mV. (This is Phase 0.) This is followed by a phase of rapid repolarisation when potassium channels open to allow a rapid potassium efflux at the same time as the fast sodium channels closing. This rapid repolarisation phase only reaches a transmembrane potential of approximately +10 mV. (This is Phase 1.) Voltage-gated calcium channels then open which balance the continued potassium efflux. This results in a stable plateau phase at approximately +10 mV. (This is Phase 2.) The calcium channels then close, once more leaving unopposed potassium efflux, which causes further rapid repolarisation of the membrane back to approximately -90 mV. (This is Phase 3.) During Phase 4, the sodium–potassium

and sodium–calcium exchangers reset the system back to the original status, awaiting the next action potential. During Phases 1, 2 and 3, the myocyte remains refractory to stimulation from another action potential.[1]

Sources of abnormal rhythms

Abnormal rhythms can originate due to either abnormal impulse generation (SA node dysfunction or enhanced ectopic automaticity) or abnormal conduction through the cardiac conduction system. Abnormal conduction can be due to a block of the conducting system (bradydysrhythmias) or because of re-entry of impulses via abnormal pathways connecting the atria and ventricles (tachydysrhythmias). Goal directed management requires an initial understanding of the origin of the specific dysrhythmia.[1]

According to the AHA ACLS algorithms:[5]

1. Bradydysrhythmias (HR < 60 bpm) are managed with atropine, dopamine, adrenaline or pacing if haemodynamically significant.
2. Tachydysrhythmias (HR > 100 bpm) are managed with electrical or chemical cardioversion, defibrillation or with one of the antidysrhythmic agents if haemodynamically significant.

Whenever a dysrhythmia is haemodynamically significant, full CPR must accompany the management of the rhythm disorder.[1] Where a tachydysrhythmia is caused by re-entry though an aberrant accessory conduction pathway, it may be necessary to ablate the pathway that is allowing the re-entry to occur.

Understanding the source of an abnormal rhythm is vital to inform our approach to perioperative dysrhythmias.[1,6] The questions to answer when addressing a dysrhythmia are:

1. Is the patient haemodynamically stable?
2. What is the likely origin site? (supraventricular/ventricular)
3. What is the heart rate? (tachycardia or bradycardia)
4. Is the rhythm regular or irregular?
5. Is there one P wave per QRS complex?
6. Is the QRS complex normal or abnormal?
7. How urgently does this need to be reversed or controlled?

Classification of antidysrhythmic agents

The management of tachydysrhythmias is based on the manipulation of sodium, potassium and calcium movement as well as second messaging at adrenergic receptors.[7] The difference between the predominant electrolyte causing the impulse generation in the pacemaker and the myocyte cells is exploited in the choice of antidysrhythmic agent. Sodium channel blockers block conduction in myocytes, prolonging the QRS interval and slowing myocardial conduction. Calcium channel blockers block stimulation in the SA and AV nodes, leading to a slower heart rate, a prolonged PR interval and slower AV conduction.[7] The Vaughan-Williams classification of antidysrhythmic drugs (Table I) divides these into four classes, based on the action site.[1]

Table I: Vaughan-William's classification[1]

Class	Action	Effect
I	Sodium channel blockade	Depresses Phase 0 depolarisation
II	Beta blockade	Decreases Phase 4 and Phase 0 in pacemaker cells
III	Potassium channel blockade	Decreases Phase 3 repolarisation (increases myocyte refractory time)
IV	Calcium channel blockade	Blocks voltage-gated calcium channels in SA and AV nodes

Changes to the AHA CPR and ECC guidelines (2020)[5]

1. Early adrenaline administration has been emphasised, especially in patients with non-shockable rhythms.
2. Invasive blood pressure monitoring and end-tidal CO_2 monitoring may be useful during CPR.
3. Double sequential defibrillation is not recommended for intractable shockable rhythms. Changing the direction of the defibrillator pads may be an equally effective and less harmful alternative.
4. Intravenous access is the preferred route for medication delivery. (Intraosseous is an acceptable second option.)
5. After the return of spontaneous circulation is achieved, the following must be monitored closely: PaO_2, blood pressure, temperature, multimodal neuroprognostication and the possibility to offer percutaneous coronary intervention.
6. Debriefing should be done for the care team post-resuscitation.
7. Cardiac arrest in pregnancy: the focus is on maternal resuscitation.
   - Pregnant patients are prone to hypoxia, so oxygenation and airway management should be prioritised in a cardiac arrest resuscitation in a pregnant patient.
   - Foetal monitoring must not be done during a cardiac arrest resuscitation in a pregnant patient.
   - Early postmortem caesarean section is advocated to improve the chances of successful resuscitation of the mother. (And secondarily to possibly save the infant).
8. Recovery has been added as a sixth link to the chain of the survival algorithm.

The 2020 AHA scientific statement on drug-induced dysrhythmias[8]

The American Heart Association (AHA) released a scientific statement on drug-induced dysrhythmias in 2020. The items pertaining to the perioperative patient are as follows:

1. Beware of bradycardia when using the following drugs:
   - Propofol: causes a 14.7% incidence.
   - Suxamethonium: causes a 36% incidence with the administration of a second dose (causes a mean drop of 23 beats per minute).
   - Others: neostigmine, sugammadex, selective serotonin reuptake inhibitor (SSRI) agents, corticosteroids, magnesium,

beta blockers, calcium channel blockers, alpha-2 agonists and amiodarone may all cause a bradycardia.

2. Supraventricular dysrhythmias: atrial fibrillation (AF).
   - An association has been cited between AF and corticosteroid usage. This is probably not due to the corticosteroids but rather due to the underlying condition for which these were prescribed.
   - There may be an association between AF and analgesic use. Nonsteroidal anti-inflammatory drugs (NSAIDs), specifically Cox-2 inhibitors, have been implicated. This seems to be more of an issue in patients receiving Cox-2 inhibitors long-term rather than short-term perioperative use.
3. Ventricular dysrhythmias: the AHA scientific statement identified three risk factors for the development of malignant ventricular dysrhythmias in the perioperative period, namely long QT syndrome, Brugada syndrome and local anaesthetic systemic toxicity (LAST).
   - Long QT syndrome may be exacerbated by some drugs and may lead to Torsades des Points.
   - Brugada syndrome is a genetic disorder of sodium channels that places patients at risk for sudden cardiac death from ventricular fibrillation. These patients have a normal QT interval and a structurally normal heart.[9] A very weak association with propofol and ketamine has been seen in susceptible patients.
   - LAST has a very low overall incidence (approximately 0.0003%). It is most likely to occur with bupivacaine but is also seen with ropivacaine usage. ACLS protocol changes in the management of LAST are that i) the adrenaline dose has been reduced from 1 mg to 1 µg/kg, ii) lipid emulsion therapy must commence early (100 ml bolus followed by 10–15 ml/min infusion for 15–20 minutes), and iii) the early mechanical circulatory support may be needed.

The most common perioperative dysrhythmia (AF)[10,11]

The incidence of perioperative atrial fibrillation (POAF) varies between 5% and 20% in patients undergoing non-cardiac surgery, with the higher incidence more common as the patient's age advances, making POAF the most common perioperative dysrhythmia. POAF significantly increases morbidity by causing cardiac failure with subsequent organ hypoperfusion, which mainly leads to acute kidney injury and myocardial ischaemia. POAF also increases mortality by increasing the patient's risk for stroke and myocardial ischaemia.[8,10] Cardiac failure develops from a fast ventricular rate with resultant ventricular under-filling as well as the loss of the atrial kick from atrial contraction. The loss of atrial kick also predisposes the patient to the formation of clots within the atria.[1] Once AF has been present for 48 hours or longer, cardioversion carries a significant risk of causing thromboembolism.[1]

POAF may be paroxysmal, persistent (i.e. normal sinus rhythm [NSR] can be achieved following treatment) or permanent. Left untreated, it is likely that POAF will not resolve spontaneously but will become permanent.

Table II: Risk factors for developing perioperative atrial fibrillation

Patient-related risk factors	Surgery-/anaesthesia-related risk factors
Advanced age	Hypoxia
Prior atrial fibrillation	Hypovolaemia
Congestive cardiac failure	Hypervolaemia
Ischaemic heart disease	Hypotension perioperatively
Structural cardiac disorder	Hypercarbia
Hypertension	Hypoglycaemia
Chronic renal failure	Electrolyte imbalance (↑K^+, ↓Mg^{++})
Sepsis	Anaemia
Chronic obstructive airway disease	Acidosis
Obstructive sleep apnoea	Hypothermia
Hyperthyroidism	Catecholamine administration/release
	Pain
	Mechanical irritation (e.g. CVC tip)

A perioperative approach to POAF include the following:[7,10,11]

Preoperative

- Look for causes of new-onset POAF, especially myocardial ischaemia, drug effects, metabolic and endocrine causes.
- Request a cardiology consultation and echocardiogram if onset was within 48 hours.
- Consider delaying surgery if time permits optimisation and in patients who present with a rapid ventricular response rate (for cardioversion or rate control).
- Continue rate control agents until the day of surgery.
- Consider anticoagulation and possible bridging therapy. (The risk of stroke vs the risk of bleeding must be balanced.)

Intraoperative

- With acute-onset intraoperative POAF, assess the haemodynamic status and look for a cause such as one of those listed in Table II. Cardioversion will have limited success if the underlying cause is not addressed.
- Manage any surgery- and anaesthesia-related risk factors as outlined in Table II, including avoiding the use of arrhythmogenic agents.
- Urgent electrical cardioversion is required for haemodynamically unstable POAF with a rapid ventricular response rate.
- Use rate control agents for haemodynamically stable new-onset POAF. Esmolol is short-acting and easy to titrate due to its rapid metabolism by plasma esterases. The disadvantage of esmolol is that the ß1 blockade (negative inotropic effect) is poorly tolerated in patients with left ventricular dysfunction. Diltiazem may be better tolerated in these patients. If the patient cannot tolerate beta blockers or calcium channel blockers, then use amiodarone. (Target a heart rate < 110 bpm).

- Digoxin is not recommended in the perioperative period due to its slow onset of action (6 hours).
- Use phenylephrine if a vasopressor is required.

Postoperative

- Check that the patient is haemodynamically stable.
- Electrical cardioversion is required for haemodynamically unstable POAF with a rapid ventricular response.
- Use rate control agents for haemodynamically stable new-onset POAF.
- Identify the cause of new-onset POAF.
- Point-of-care ultrasound can help to diagnose a cardiac cause such as pulmonary embolism or myocardial infarction, as well as the presence of any intra-atrial thrombus.
- Continue with rate control in patients with permanent atrial fibrillation.
- Postoperative POAF significantly increases the patient's risk of thromboembolism and a cerebrovascular accident.
- The HAS-BLED score is useful to determine the risk of bleeding.
- The CHA_2DS_2-VASc score can aid in determining the risk of stroke. (Intra-atrial clots are a significant threat after 48 hours of AF.)

The dysrhythmia which can be worsened by anaesthesia agents (long QT syndrome)[6,7,12]

Long QT syndrome (LQTS) is caused by a cardiac channelopathy (potassium and sodium channels) which leads to a prolonged ventricular repolarisation time.[1] The danger is that it may progress to Torsades des Pointes which can rapidly deteriorate into ventricular fibrillation.

Two forms occur, namely congenital and acquired.

Congenital

There are two types of the congenital form:

1. Jervell and Lange-Nielson syndrome, which is rare and autosomal recessive.
2. Romano-Ward syndrome, which is autosomal dominant with variable penetrance.

Patients with congenital LQTS will likely be on a beta blocker and may have an implantable cardioverter defibrillator (ICD) in situ.

Acquired

This can be caused by electrolyte imbalances, by a severe bradycardia or by the effect of a drug which prolongs the QT interval time.[6] Several drugs from different classes are implicated.

The diagnosis of LQTS (either congenital or acquired) is made by determining the corrected QT interval using the Bazette formula, which corrects for heart rate.

$$QTc = \frac{QT}{\sqrt{RR}}$$

The normal is 450 ms in men and 460 ms in women.

Perioperative approach to a patient with LQTS[7]

Preoperative

1. Continue with beta blockers in known patients.
2. Review all prescribed medications for any that may cause a prolonged QT interval.
3. Request a 12 lead electrocardiogram (ECG) and a cardiology consult in newly diagnosed patients. Have a high index of suspicion in undiagnosed patients who report palpitations, seizures or syncope episodes.
4. Correct low K^+, Mg^{++} or Ca^{++} if present.
5. Check that the ICD is working and has adequate battery life if one is implanted.
6. Prescribe an anxiolytic premedication.

Intraoperative

1. Avoid drugs which prolong the QT interval.[1,7] These include volatile agents, sufentanyl, succinylcholine, anticholinesterase agents, anticholinergic agents, droperidol and serotonin type 3 receptor antagonists.
2. Monitor the QT interval in lead II.
3. Avoid unopposed sympathetic stimulation.
4. Avoid hypothermia.
5. Keep the intrathoracic pressures low.
6. Local anaesthetic agents are all safe but avoid using preparations that contain adrenaline.
7. Consider a prophylactic magnesium infusion in high-risk patients.
8. Monitor and correct any electrolyte abnormality.

Postoperative

1. Continue with beta blockers.
2. Ensure cardiac monitoring for 24 hours. Consider placement in intensive care.
3. If torsade de pointes does develop, then load the patient with magnesium sulphate and start a magnesium infusion. Check the K^+ and replace if low. Do not use amiodarone. Overdrive pacing to a heart rate above 90 bpm may help. If the patient becomes unstable or develops ventricular fibrillation, then standard life support with defibrillation is required.

ORCID

B Gardner https://orcid.org/0000-0003-3893-6974

References

1. Kwon CH, Kim SH. Intraoperative management of critical arrhythmia. Korean J Anesthesiol. 2017;70(2):120-6. https://doi.org/10.4097/kjae.2017.70.2.120.
2. Nunnally ME, O'Connor MF, Kordylewski H, Westlake B, Dutton RP. The incidence and risk factors for perioperative cardiac arrest observed in the national anesthesia clinical outcomes registry. Anesth Analg. 2015;120(2):364-70. https://doi.org/10.1213/ANE.0000000000000527.
3. Kaiser HA, Saied NN, Kokoefer AS, et al. Incidence and prediction of intraoperative and postoperative cardiac arrest requiring cardiopulmonary resuscitation and 30-day mortality in non-cardiac surgical patients. PLoS One. 2020;15(1):e0225939. https://doi.org/10.1371/journal.pone.0225939.

4. Maltsev VA, Lakatta EG. Normal heart rhythm is initiated and regulated by an intracellular calcium clock within pacemaker cells. Heart Lung Circ. 2007;16(5):335-48. https://doi.org/10.1016/j.hlc.2007.07.005.
5. American Heart Association. Highlights of the 2020 American Heart Association Guidelines for CPR and ECC [Internet]. Texas: American Heart Association; 2020. Available from: https://cpr.heart.org/-/media/cpr-files/cpr-guidelines-files/highlights/hghlghts_2020_ecc_guidelines_english.pdf. Accessed 10 Sept 2021.
6. BMJ Best Practice [Internet]. BMJ Best Practice Overview of dysrhythmias (cardiac). London: BMJ Publishing Group. Available from: https://bestpractice.bmj.com/topics/en-us/837/pdf/837/Overview%20of%20dysrhythmias%20%28cardiac%29.pdf. Accessed 9 Sept 2021.
7. Thompson A, Balser JR. Perioperative cardiac arrhythmias. Br J Anaesth. 2004;93(1):86-94. https://doi.org/10.1093/bja/aeh166.
8. Vanneman MW, Madhok J, Weimer JM, et al. Perioperative implications of the 2020 American Heart Association scientific statement on drug-induced arrhythmias - a focused review. J Cardiothorac Vasc Anesth. 2021;11:S1053-0770(21)00381-5. https://doi.org/10.1053/j.jvca.2021.05.008.
9. Harris P, Lysitsas D. Ventricular arrhythmias and sudden cardiac death. BJA Educ. 2016;16(7):221-9. https://doi.org/10.1093/bjaed/mkv056.
10. Karamchandani K, Khanna AK, Bose S, et al. Atrial fibrillation: current evidence and management strategies during the perioperative period. Anesth Analg. 2020;130(1):2-13. https://doi.org/10.1213/ANE.0000000000004474.
11. Stewart AM, Greaves K, Bromilow J. Supraventricular tachyarrhythmias and their management in the perioperative period. BJA Educ. 2015;15(2):90-7. https://doi.org/10.1093/bjaceaccp/mku018.
12. Levy D, Bigham C, Tomlinson D. Anaesthesia for patients with hereditary arrhythmias; part 2: congenital long QT syndrome and arrhythmogenic right ventricular cardiomyopathy. BJA Educ. 2018;18(8):246e253. https://doi.org/10.1016/j.bjae.2018.04.005.