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Post Operative Arrhythmias

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1. Introduction

Heart rhythm disturbances are being increasingly recognized during the postoperative period. While many are transient and short lived without altering the recovery phase after cardiac or non-cardiac surgery, they do have the potential to pose a threat to patient's health, prolong hospital stay, and in a minority of patients may even cause death. Continuous monitoring is becoming the standard of care after surgery and therefore rhythm disturbances are being more frequently diagnosed during the postoperative recovery period. While cardiology consultation may be required, surgeons and anesthesiologists are often the first responders and are expected to be able to recognize the rhythm disturbance and treat them appropriately.

2. Normal physiology

Normal sinus rhythm is when the heart beats in an orderly predetermined sequence. The atria contract initially in response to the firing of an impulse by the Sino-Atrial (SA) node located at the junction of the superior vena cava and the right atrium. The SA node contains specialized tissue with 'pacemaker cells', which can initiate repetitive rhythmic action potentials. These potentials then travel via internodal atrial pathways to the AtrioVentricular (AV) node located at the right posterior portion of the interatrial septum. The AV node slows conduction into the bundle of HIS which then leads to its right and left branches. The left bundle branch further divides into anterior and posterior fascicles. The final pathway of conduction is the Purkinje system, which consists of a network of fibers that transmit the electrical impulse to the myocardium near the apex of the heart. (1)

The Electrocardiogram (ECG) is a reliable and practical way to document the underlying cardiac rhythm. It essentially consists of a recording obtained by 12 surface leads which trace the electrical activity of the heart from different directions. The 12 leads include 6 limb and 6 precordial leads. The limb leads include 3 bipolar leads (I, II, III) meaning they have 2 electrodes of opposite polarity. The other limb leads are aVR, aVL, aVF which are the unipolar leads meaning they have only one electrode connecting to a central terminal. The precordial leads are all unipolar and include V1-V6.

The limb leads are the frontal plane leads representing electrical current along the coronal plane of the heart, i.e. right/left and superior/inferior. The precordial leads represent the horizontal plane of the heart measuring transverse currents, i.e. right/left and anterior/posterior. Lead I traces currents from right shoulder to left shoulder, lead II from

right shoulder to left leg, and lead III from left arm to left leg. Lead aVF traces from central terminal, which corresponds to zero potential to the left leg, aVL from centre to left arm and aVR from centre to right arm. The precordial leads work in a similar fashion in that leads V1 – V6 trace their axis center out from right to left respectively, so that V1 represents right of the interventricular septum, V2 and V3 the interventricular septum (anterior wall), V4 the apex (anterolateral), V5 and V6 the lateral ventricular wall. Any current flowing towards the lead causes a positive deflection and current flowing away from the lead causes a negative deflection and vice versa. The strength of the deflection depends on the amount of potential recorded and is affected by cardiac and extracardiac structures. To understand the electrophysiological basis of the 12-lead tracings on an ECG is important, because it gives clues about the origin of an arrhythmia and sometimes guides their therapies.

The first deflection on an ECG is the P wave which represents atrial depolarization. In sinus rhythm without any discernable atrial pathology, P wave is an upright, smooth, rounded wave with relatively low voltage. The PR interval consists of the P wave and the normally isoelectric segment up to the initial deflection of the QRS complex. The PR interval represents the conduction through atria, AV node, bundle branches and Purkinje system. The QRS complex follows the PR segment. The initial negative deflection is the Q wave, a positive deflection which can occur either initially or after the Q is the R wave while any negative deflection which follows the R is the S wave. QRS complex represents intraventricular conduction and depolarization. The J point represents the junction between QRS and the ST segment. The ST segment corresponds with the end of ventricular depolarization and start of the ventricular repolarization. The T wave, which follows the ST segment, represents ventricular repolarization. As such the QT interval represents the complete ventricular depolarization and repolarization period. Occasionally a small hump-like U wave follows the T wave, and is felt to be due to repolarization of the purkinje system. (2,3)

The bedside monitors which are routinely used for continuous cardiac monitoring are typically wireless, i.e. telemetry systems. These can be either 5 lead wire or 3 lead wire systems. The 5 lead wire system allows for monitoring all of the limb leads or the precordial leads while the 3 lead wire system allows monitoring one lead at a time, usually lead II, because the P wave is best visible in this lead. Depending on the monitoring system available it is essential for health care providers to be able to recognize the cardiac rhythm changes based only on a few select leads seen on the monitor as there may be no time to record a 12 lead ECG.

3. Recognition of arrhythmias

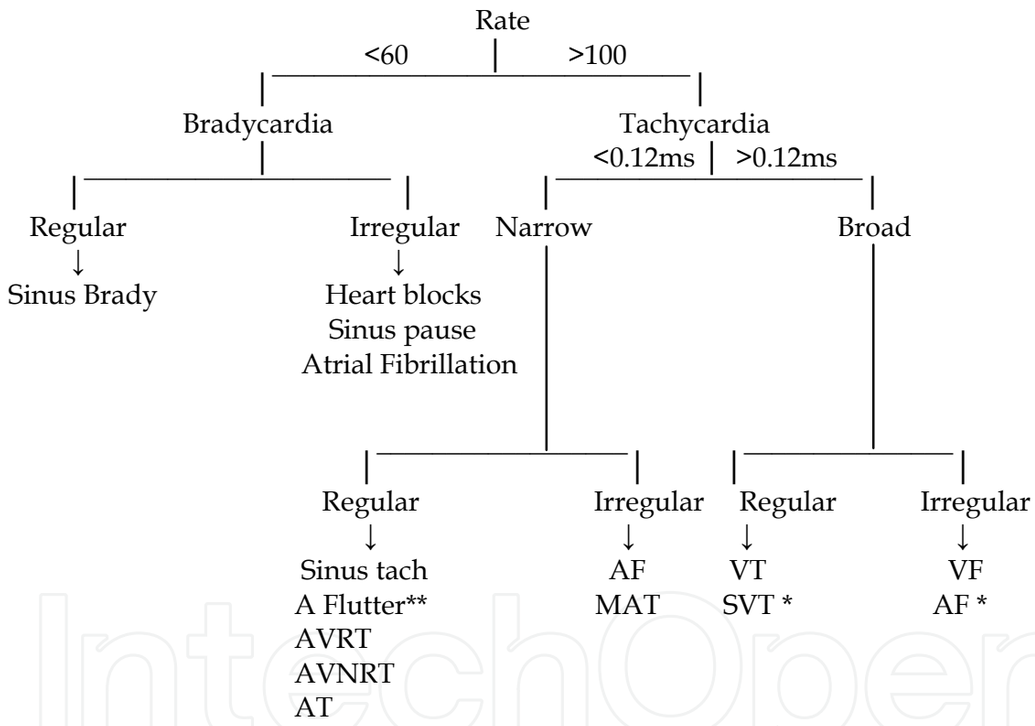
Postoperative arrhythmias though transient are usually sudden in onset. It is essential to recognize a rhythm disturbance and institute treatment as quickly as possible in most cases. A 12 lead ECG is recommended but may be impractical if the rhythm disturbance is an immediate threat to the patient's life. The wave forms visible on the telemonitor or a rhythm strip in one lead tracing may be the only available clue.

It is worthwhile to note some salient points early. Is the patient stable as assessed by the blood pressure, oxygen saturation or mental status? If deemed unstable then more aggressive steps are warranted.

A rapid and accurate interpretation of the ECG can be tricky and readers are advised to develop a personal strategy to identify any given cardiac tracing so that a quick diagnosis can be made. One approach is to identify and describe 5 basic features of the electrocardiogram (2):

Step 1. Determine the ventricular rate – tachycardia is >100 / Bradycardia is < 60
Step 2. Measure the QRS complex – Narrow is < 0.12ms / Broad is > 0.12 ms
Step 3. Determine the regularity of the QRS complex – Regular/irregular
Step 4. Identify the P waves – upright in lead II and III and negative in aVR usually identifies sinus rhythm, P waves are absent in atrial fibrillation, saw tooth appearance at an atrial rate of 300 bpm may indicate atrial flutter
Step 5: Measure the PR interval – helps identify AV delay

The above steps should help one to identify the salient features of any rhythm and place it in one of the following mentioned categories. (Figure 1) We would like to point out that this scheme is only one of many and sometimes more than one arrhythmia can be present in a patient. This scheme also, at times over simplifies natural heart rhythms. For example, a heart rate of 40 beats per minute (bpm) during sleep or in an athletic patient can be normal, while patients with an abnormal conduction system can have supraventricular arrhythmias with heart rates less than 100 bpm.



(AF – Atrial Fibrillation, AT – Atrial tachycardia, MAT – Multifocal atria tachycardia, SVT – Supra ventricular tachycardia, VF – Ventricular Fibrillation, VT – Ventricular tachycardia)

*With Aberrancy
** Flutter can be irregular occasionally when the AV block is variable

Fig. 1. Various cardiac rhythm disturbances noted in clinical practice

The Brugada criteria can be used to identify any broad complex tachycardia (4)
Step 1. Are there RS complexes in any of the chest leads?
Step 2. Is the onset of the R wave to the nadir of S > 100 ms?
Step 3. Is there any AV dissociation?
Step 4. Is there any typical bundle branch morphology in leads V1 or V6?

If the answer to any of questions 1-3 is yes or to question 4 is no, the rhythm is probably ventricular tachycardia. There are several additional ways to distinguish between supra ventricular and ventricular arrhythmias, which exceed the objectives of this review.

4. Types of arrhythmias

The conduction system of the heart has to be intact anatomically and physiologically for synchronized contraction of the heart in a regular and coordinated fashion. Arrhythmias are caused when there is disturbance in the working of the conduction system for any reason. The accepted mechanisms include abnormality of conduction (i.e. blocking or Re-entry of the impulse), abnormality of impulse initiation (i.e. altered automaticity or triggered activity). The underlying causes can include ischemia, electrolyte imbalances, scarring or fibrosis of atrial and ventricular tissue, increased or decreased excitability for various reasons including changes in autonomic nervous system, action of drugs and others.
Disturbances of the normal cardiac rhythm can be of many types. A universal hierarchical classification which can encompass all the salient arrhythmias is difficult to conceive. Arrhythmias are usually classified based on morphology, rate or origin. They can be divided into fast or slow based on the rate- Tachyarrhythmias when heart rate is faster than 100 bpm and bradyarrhythmias when rate is slower than 60 bpm. The QRS duration which defines the ventricular depolarization is usually less than 0.12 seconds (or 3 small boxes on the ECG at 25 mm/sec paper speed). If it is more than 0.12 seconds it represents delayed depolarization and can be used as a feature to divide the tachyarrhythmias into narrow complex and wide complex tachycardia. Another classification of arrhythmias describes regular versus irregular rhythms. Finally arrhythmias can generally be classified based on various anatomical substrates of the heart, which initiate these rhythm changes like atrial (or supraventricular) or ventricular arrhythmias. As mentioned above these classifications are an attempt, far from perfect, to distinguish normal from abnormal and define categories with different underlying pathophysiology and treatment options.
The following chapter will discuss Atrial Fibrillation (AF) after cardiac surgery initially as this seems to be the most common and most extensively studied arrhythmia.

5. Postoperative AF

Incidence:

The incidence of any arrhythmia postoperatively can be up to 85% (5). The Multi center anesthesia outcomes study quotes that postoperative arrhythmias can complicate about 70% of the operations. (6) AF seems to be the most common arrhythmia in the post operative period (7). Goldman concluded as early as 1978 utilizing a prospective registry that the incidence of postoperative AF was nearly 3% after non-cardiac surgeries. (8) While AF is certainly more frequent after cardiac surgery, incidence varies between studies. Reasons for these variations seem to be different ascertainment methods between studies (12 lead ECG vs. continuous telemetry monitoring etc). Mathew et al document an incidence of up to 34% with similar incidences in North America and Europe but lower incidence in Asia at 16 %. (13) The incidence also varies depending on the type of cardiac surgery undertaken. AF seems to occur 60% of the time post mitral valve surgery, 36% of the time post aortic valve surgery, (10) and 25% post cardiac transplants. (11) The combination of coronary artery bypass graft surgery (CABG) with valve surgery seems to increase the risk for AF as well. (12) Often times the

definition for postoperative AF includes the need of medical treatment or electrical cardioversion (14) or confirmation on a 12 lead ECG (13) altering the incidence rates further.
The peak incidence of postoperative AF (POAF) has been consistently described on day 2 and 3 after surgery. (10) It usually is transient with 80% of the patients converting to sinus rhythm within 24 hours. The recurrence rate has been quoted to be as much as 50% but still only 10% of patients are still in AF at 6 weeks post operation. (13) Late Postoperative AF is less frequent but was seen in nearly 5% of the postoperative patients after discharge from the hospital. This was found when patients were being followed for cardiac rehabilitation and documented in the ISYDE and ICAROS registries in Italy. (15) Despite better coordinated postoperative care and advances in cardiothoracic surgical and anesthetic practices, the incidence of AF seems stable with no reduction over the last 2 decades.

Pathophysiology of Postoperative AF:

AF is generally due to reentry of multiple wavelets circling the atria. It is likely that a pre-existing substrate is needed to allow peri-operative triggers to initiate AF. It is thus a specific interaction of preexisting and perioperative risk factors which can lead to AF.

Preexisting factors:

Age is the most consistent risk factor seen in past studies. Advancing age increases the risk with each decade. The incidence of POAF is around 6% when less than 40y of age, 18% in less than 60y olds and increasing to as much as 50% in patients older than 80 years. (13,14,16) Other risk factors include male gender, history of prior AF, heart valve disease (especially if the mitral valve is affected), prior cardiac surgery, prior cardiac structural changes like increased left atrial size and left ventricular hypertrophy. Preexisting medical conditions like obesity, chronic lung disease, peripheral vascular disease, hypertension, prior stroke are associated with increased incidence of POAF. However certain other morbid factors like preexisting diabetes, chronic kidney disease, hyperlipidemia, smoking have not been shown to be individual risk factors for POAF in some studies. (17) Pericarditis which is usually a consequence of the cardiac surgery itself is mechanistically involved. Other unique factors such as preoperative use of Digoxin or Dopamine, raised Brain natriuretic peptide (BNP) and right-sided coronary artery disease have been associated as well. ECG features like increased P wave duration of more than 140 ms, which is suggestive of atrial conduction delay can increase the susceptibility to AF. (18) Withdrawal of preoperative Angiotensin converting enzyme inhibitor (ACE I) or Beta blocker therapy is also contributory if not immediately initiated after the surgery. (13)

Intraoperative factors:

Certain operative features like aortic cross clamping, pulmonary venting, bicaval venous cannulation, increased length of cardiopulmonary bypass time and mitral valve surgery can increase the propensity for POAF. It has also been noted that at times cardioplegia via coronary sinus does not stun the atria completely and may be associated with occurrence of POAF(19) Direct cardiac injury due to operative techniques causing inflammation is perceived as plausible cause as well.

Postoperative features:

The postoperative period is a critical stage as the body is yet to recover from the operative stress completely. Many proarrhythmic features such as pericardial inflammation, acute blood pressure or volume changes, acute cardiac ischemia, electrolyte imbalances,

hypothyroidism are present at this juncture. (20, 21, 22) Increased sympathetic activation causing exaggerated adrenergic responses could be a factor as well. (23)
The assumption is that non-uniform disruption of the electric conduction properties leads to changes in the resistance between adjacent cells in the atria. This causes decreased atrial conduction and creation of micro reentry loops causing AF. (24) Various factors mentioned above change the atrial refractoriness/transmembrane potentials causing increased local reentry and subsequent AF. (25) The suggestion that expression of connexin 40, a gap junction protein in the atria is altered during the postoperative period lends credence to the theory that the gap junction function in the atria is altered. (26)

Clinical Significance:

POAF is usually transient as the underlying mechanical and metabolic changes are usually reversible and not long lasting. However it is associated with significant morbidity and mortality even when it occurs briefly. POAF can increase the risk of stroke by 3-4 folds. (27) Cresswell et al noted that the occurrence of stroke postoperatively with AF was at 3.3 vs.1.2% without AF. (12) However, other features such as increased age, prior stroke, length of cardiac bypass time seem to be playing additional role in the additive risk of stroke postoperatively.
AF has been shown to increase overall health care cost. The hospitalization time is increased by an average of 2 to 5 days. (13, 14) The costs were higher by as much as $10,000 per patient if AF occurred postoperatively. The chances that the patient will suffer infection, renal failure, and mechanical ventilation also seem to be higher when AF is present. There has been suspicion that cognition of the patients can be affected as evidenced by a fall in the Mini Mental Score postoperatively when AF occurred. (10, 17, 28)
AF remains the leading cause for readmission after hospital discharge following cardiac surgery. It is estimated that AF contributed to nearly 23% of readmissions in one series. (9) So it would seem AF is a problem even after discharge and it would argue for continued monitoring of the patient as an outpatient preferably in a cardiac rehabilitation program.
AF seems to be associated with increased mortality both early and late after operation even after correction for many important confounding variables. It is estimated that the mortality associated with postoperative AF is around 5% compared to around 2% without AF. (13)

Management:

Given the morbidity and mortality associated with POAF it has long been a target for preventive as well as suppressive therapy. A variety of interventions have been studied and validated. We would like to clarify that our list does not claim to be complete, but only gives an overview of some of the most important therapies available. In most cases consultation with a Cardiologist is recommended. Multiple studies (29) have shown that AF can be suppressed in the postoperative period and various meta analyses confirmed their findings. Incidence of AF was reduced by as much as 50%. (30, 31) We categorize preventative measures into preoperative, operative and postoperative measures.

Preoperative measures:

The main thrust has been to reduce the sympathetic drive and Beta blockers seem to be the mainstay of this preventive approach. The 2004 ACC/AHA guidelines give a class I recommendation for preoperative and early postoperative beta blockade to prevent POAF. (32)
Amiodarone which blocks Potassium and Calcium ion channels, and has both alpha and beta blockade properties has been assessed in various trials such as AFIST, ARCH, AFIST 2, GAP and PapaBear for prevention of POAF and has been summarized in a meta analysis

(33). It can reduce postoperative AF by 50% - 70% and evidence suggests that ventricular arrhythmias are also reduced. However, there is concern about possible complications including proarrhythmia, sudden respiratory distress or bradycardia requiring pacing following Amiodarone prophylaxis or treatment. Therefore, Amiodarone therapy needs to be closely monitored.

Digoxin has also been studied but seems to be better only when used along with beta blockers and is currently not recommended. (34)

Magnesium has been studied as a preventive strategy and while hypomagnesaemia does definitely portend arrhythmias (20) supplementation does not seem to be helpful in reducing rhythm disturbances. One meta analysis (35) has shown a positive outcome but another study (36) has cast a doubt on the utility of Magnesium supplementation in preventing POAF.

Sotalol which has beta blocker as well as potassium channel blocking properties has been shown in certain studies (37) to be useful in preventing AF with relative risk reduction of up to 90%. However, studies generally involved small sample sizes. (29) There may not be an incremental effect of Sotalol along with Beta blocker therapy to prevent POAF.

Angiotensin Converting Enzyme inhibitor (ACE I) therapy did not consistently reduce POAF, but incidence may be increased if the ACE inhibitor therapy is withdrawn in patients who were receiving it before surgery.

Statins appear to have a beneficial effect in preventing AF. The ARMYDA 3 trial showed that taking a statin two weeks prior to surgery significantly reduces the incidence of POAF, although there were concerns about the relatively high incidence of AF in the control group. (38)

Various other agents like Non steroidal anti inflammatory agents (NSAID), Ascorbic acid, N-acetyl cysteine, Nitroprusside, Glucocorticoids, Fish oil have been tried on the premise that they reduce the oxidative stress and help modify the inflammatory process that seems to be present postoperatively and thereby contributing to lower the risk of POAF. However despite positive results in small trials, larger randomized controlled trials are necessary to ascertain any true benefit. (39)

Pacing via epicardial wires introduced at the time of the surgery has been recognized as an effective method in controlling AF in as much as 63% of the cases. (40) Pacing is done either at the sinus rate or faster with overdrive pacing. Studies have showed that bi atrial pacing (BAP) seems to be better than pacing in only one atrium. The American College of Chest Physicians (ACCP) guide recommends BAP over either right or left single atrial pacing. (41)

Beta blockers seem to provide additive benefits along with pacing in preventing POAF.

The odds ratios of various agents used in POAF prevention are given below: (21)

Beta blockers – 0.35
Sotalol – 0.36
Amiodarone – 0.54
Pacing – 0.57
Potassium – 0.53
NSAID – 0.49
ACE inhibition – 0.62

The ACC/AHA/ESC 2006 guidelines recommend using a beta blocker routinely to prevent POAF and using Amiodarone or pacing only if the patient is intolerant of beta blockers or in high risk cases such as when the patient is undergoing mitral valve surgery or if they have had prior history of AF. (32)

Intraoperative measures:

Certain operative practices and techniques have shown to be of some benefit in reducing the incidence of POAF. Off pump surgery may decrease occurrence of AF, even when taking age into account. (42, 43) The anterior fat pad present in the mediastinum is considered to have parasympathetic nerves, which may play a role in initiating POAF. One study showed that preservation of the fat pad was protective but it could not be replicated in other studies. (44,45) Other factors include inducing hypothermia during Cardio Pulmonary Bypass (CPB), using posterior pericardiotomy and Heparin coated CPB circuit etc. (27)

Postoperative measures:

The only postoperative preventative measure may be early reinitiation of beta blockers and ACE-inhibitors. (13) There was a suggestion in a recent study that early statin use post operatively may be beneficial in preventing POAF after cardiac surgery as well. (46)

If AF does occur and is persistent despite the prophylactic measures, treatment should be initiated. There are two general approaches to AF treatment, Rate control or Rhythm control with both being acceptable as to preferred outcomes. (47) Whatever approach is taken, initial efforts need to be made to try and correct any obvious precipitating or co-existent mitigating factors. Meticulous attention needs to be paid to pain control, volume status, electrolyte balance, correcting anemia and hypoxia. Anticoagulation needs to be initiated as well if the AF is persistent for more than 48 hours.

Rhythm control where in AF is converted to sinus rhythm is preferred when the patient is deemed unstable such as if there is hypotension, ongoing ischemia, co-existing heart failure, if pre excitation is suspected or if the patient is very symptomatic. It is also preferred if anticoagulation is not an option for any reason. Rhythm control can be achieved either with pharmacological cardioversion or electrical cardioversion. Various anti arrhythmic agents can be used to convert AF, Amiodarone is typically preferred, because it can be transitioned to oral route, has comparatively lower proarrhythmic potential and may be better at ventricular rate control. Also as most patients have some underlying left ventricular dysfunction or coronary artery disease, Amiodarone is a safer choice in such patients. It is usually given as an initial bolus at 5 mg/Kg body weight over 30 minutes and then continued as an infusion at a dose of 25 mg /Hr. Various other pharmacological rhythm control agents used include Disopyramide, Procainamide, Flecainide, Ibutilide and Dofetilide. (10)

Direct current (DC) cardioversion is a quick and safe way to attempt rhythm control. Initial shock is attempted at 100 - 200 joules with synchronization when monophasic waveforms are used and 50 – 100 joules when biphasic waveforms are used. As usually the POAF has been present only for a short time DC cardioversion can successfully convert the AF to sinus in up to 95% of the cases. If it is not successful, intra venous Ibutilide can be given before repeat electrical cardioversion. However, significant pauses and risk for Torsades make Ibutilide less attractive for most practitioners. The transvenous electrodes or epicardial wires placed during surgery can be used for cardioversion or patient can be shocked by two pairs of external patch electrodes.

Rate control can be achieved with a variety of agents such as beta blockers including Metoprolol, Esmolol, Atenolol or Calcium channel antagonists like Diltiazem. Digoxin, Amiodarone or the newer agent Dronedarone are also popular choices at rate control. (48) Anticoagulation with warfarin is recommended if the AF is persisting for more than 48 hours. (32, 48) Heparin bridging is not recommended unless high risk features are present such as Mitral valve disease, prior stroke. (49) The criteria for anti coagulation per ESC are based on the CHADS2 – VASc score. Risk factors including increased Age > 75y and prior

Stroke, transient ischemic attack (TIA) or thrombo embolism are given 2 points each. Factors including Hypertension, Congestive heart failure, Diabetes, Ages 65-74y, female Sex and co existent Vascular disease are scored 1 point each. Anticoagulation is indicated if the combined score is $\geq$ 2. (48) Newer agents like Dabigatran are available on the market but studies will need to be done to assess its value specifically in the postoperative period.
Not much significant data is available as to the management of patients after discharge. They are usually reassessed 4-6 weeks after discharge and often times Holter monitoring is employed. Most of the patients can stop their anti arrhythmic medications and anti coagulation if they are deemed to be in sinus rhythm without intermittent AF, 3-6 months after hospital discharge.
In spite of all studies and evidence regarding preventing and treating POAF, doubts still exist whether any real benefit is obtained. Some evidence suggests that AF prevention does not or only minimally reduces the length of stay or the overall cost. (50) It is also noted that there is no actual decrease in the stroke incidence post operatively even if the AF is suppressed. It is unclear if the mortality and morbidity are improved if the AF is indeed suppressed. (51) It seems that stroke may be an epiphenomenon and not directly related to the occurrence of POAF. However a large Meta analysis does seem to suggest some overall benefit with prophylaxis measures and prevention of POAF. (52)

6. Post cardiac surgery ventricular arrhythmias

These include the more common benign isolated ectopic beats or Non sustained ventricular tachycardia (NSVT) and the more dangerous ventricular tachycardia or ventricular fibrillation (VT/VF) which fortunately are less common. The incidence of sustained ventricular arrhythmias has been quoted at around 0.4 – 1.4% (53) to 0.7 – 3% (54). The benign rhythm changes including ectopic ventricular beats and NSVT can occur in up to 60% of patients (55) but are not known to portend the more malignant rhythms like VT/VF (56) nor do they portend any rise in mortality risk (55, 57) if no underlying structural heart disease is suspected. The mortality of sustained VT is high at around 50% in hospital and a further 10% die within 2 years. (53)
The risk factors for the occurrence of VT/VF seem to correlate with factors associated in general cardiology practice. Any underlying structural heart disease, prior myocardial infarction, reduced left ventricular ejection fraction or congestive heart failure increase the risk of life threatening ventricular arrhythmias. Immediate postoperative features which set off the rhythm disturbance include any hemodynamic instability, electrolyte or acid base disturbances, hypoxia, anemia, new onset ischemia etc. An occasional cause can be acute graft closure after bypass grafting. Any inotropes used in the postoperative phase can also be pro arrhythmic.

Treatment:

Even though frequent ectopics and NSVT are considered benign it would be prudent to look for any reversible factors mentioned before in the acute phase. Lidocaine and pacing have been studied to suppress these rhythm disturbances but no actual benefit was observed. (53, 58) Sustained Ventricular arrhythmia is invariably quite unstable and quick remedial measures need to be instituted to treat the patient. Electrical cardioversion with 200 – 360 Joules is usually the first line option to convert the arrhythmia. If Direct Current cardioversion is not an option or if medications are preferred as per the clinical situation, various drugs like Lidocaine, Amiodarone, Procainamide can be considered. Emergency

pacing via epicardial leads placed during surgery can be used sometimes to provide overdrive pacing to get the heart out of the arrhythmia. Emergency bypass surgery can be considered in some situations. (59) Readers are also referred to the American Heart Association (AHA) 2010 guidelines on advanced cardiovascular life support (ACLS) for dealing with unstable tachycardia. (75)

If the patient does survive and is back in sinus it is prudent to initiate them on long term beta blocker and ACE inhibitor therapy according to current ACC guidelines. For those who sustained VT/VF and have recovered, if there are no underlying risk factors mentioned prior, a cardiac electrophysiological study can be considered and an implantable cardiac defibrillator (ICD) is advised if there is any inducible VT or VF. If the patient is deemed to have an underlying heart disease that is unlikely to respond to medical therapy, an ICD may be indicated without electrophysiological study.

7. Bradyarrhythmias after cardiac surgery

Bradyarrhythmias include sinus pauses, sinus bradycardia and various blocks depending on the site of abnormal conduction including SA node, AV node or parts of HIS bundle. Bundle branch blocks are common and are not only transient but also harmless in most cases. Various bundle branch blocks can occur in up to 50 – 60% of cases after CABG but are usually transient. (60, 61, 62, 63) Symptomatic blocks needing permanent pacemaker (PPM) insertion complicate 0.8 – 3.4% of CABG operations and up to 2 – 4% of valve surgeries. (64, 65) The incidence of symptomatic bradyarrhythmias is higher after aortic or tricuspid valve surgeries. Repeat surgeries are complicated by blocks needing pacing more often. (65) Heart transplantation is complicated by sinus node dysfunction needing a pacemaker in 21% of cases while AV node blocks needing pacemaker can happen in 4-5% of cases. (53)

Risk factors include increased age, prior Left bundle branch block (LBBB), valve calcification, left main coronary blockage, longer cardiopulmonary bypass time, higher number of bypassed arteries during surgery, associated Left Ventricular aneurysmectomy etc. Valve surgeries seem to be more of a risk than CABG. Increased vagal tone due to surgery, the type of anesthesia used or occurrence of postoperative pain seem to be important underlying factor as well.

Specific factors involved in increasing the risk of bradyarrhythmias after heart transplantation include Biatrial rather than bicaval transplant, older donor age, longer donor ischemic time, longer aortic cross clamp time.

Treatment:

It is prudent to stop all unnecessary medications that can cause increased AV block like beta blockers or calcium channel blockers. Atropine can reverse symptomatic bradycardia. Aminophylline and Theophylline can be used to increase the heart rate during sinus node dysfunction or high grade AV blocks. (66, 67) Readers are also referred to the AHA 2010 guidelines on advanced ACLS for dealing with unstable bradycardia. (75)

Patients with complete heart block, symptomatic AV block or sinus node dysfunction need to have a temporary pacer inserted. It is advisable to wait for 5 – 7 days post op so that any possible edema of the conduction system of the heart resolves before a permanent pacemaker is inserted if still indicated. (68)

Patients who already have a permanent pacemaker or ICD prior to surgery pose a challenge for the surgeons and anesthetists. Electrocautery-induced electromagnetic interference can

cause problems during the surgery. The cautery can inhibit the pacer and may cause inappropriate discharge of the ICD if the sensing function is not disabled. A comprehensive evaluation of the patient prior to surgery by an electrophysiologist is indicated. A magnet can be placed on top to disable the devices during the surgery so as to not cause any interference. Another option is to switch the pacer/ICD to asynchronous mode so that the cautery does not influence its function. However, patients need to be continuously monitored while the devices are in asynchronous mode as any malignant arrhythmias need to be treated via external defibrillator. (76)

8. Postoperative arrhythmias after non cardiac surgery

Arrhythmias complicate postoperative period after non cardiac surgery in up to 5 -20 % of the times. (69) Again, AF seems to be the most common arrhythmia making up about 68% of the documented arrhythmias. (8) Benign ventricular rhythms like ectopics or NSVT occur in up to 5 -25% of the patients and sustained VT is rather rare occurring in less than 1% of the cases. (70)

The rate of incidence after non-cardiac surgery also seems to depend on the type of surgery. Non vascular abdominal surgery, especially colorectal surgery seems more prone with rates of around 20%. The incidence seems increased after any instance of thoracotomy (10%) as well. In other instances the rate is around 0.01% after ophthalmologic surgery and 4% after orthopedic surgery.

The risk factors seem to be similar to those implicated in post cardiac surgery including male sex, increased age > 70y, heart valve disease, prior history of arrhythmia, co existing asthma, congestive heart failure, and hypertension. (71) Post operative causes include electrolyte imbalances, hypoxia, and hypercarbia. (72) Sepsis seems to be a recurring factor implicated as a causative factor of arrhythmias. In fact all kinds of stress inducing causes like stroke, Gastrointestinal bleed, Pulmonary Embolism, Myocardial Infarction, pulmonary edema and others have been implicated. Some specific factors noted to cause postoperative arrhythmias also include anastomotic leak (77) or acute alcohol withdrawal. (69) Increased vagal tone due to anesthetic practices like laryngoscopy is also a risk factor for any bradyarrhythmia.

Apart from associated morbidity similar to post cardiac surgery arrhythmias, post non-cardiac surgery arrhythmias can also cause mortality of around 12 – 50%. (72, 73, 74)

Management:

No large scale randomized trials validating the treatment of post non-cardiac surgery arrhythmias are available. However the management can be closely extrapolated from both post cardiac surgery treatment and non-surgical related general cardiology treatment protocols. Initial priority is to assess the physiological impact and stabilize the patient hemodynamically while searching for the specific causes that initiated the rhythm disturbance. One needs to rectify these issues while simultaneously initiating specific therapy to halt the arrhythmia. Specific treatment methods for individual rhythms are similar to the approach already explained for post cardiac surgery arrhythmias.

In conclusion, postoperative arrhythmias, especially AF are common and are associated with significant morbidity and mortality but can be prevented to some extent. Further research is required to completely understand causes of such arrhythmias and to improve their prevention and treatment.

9. Abbreviations

Postoperative Atrial Fibrillation (POAF), Coronary artery bypass graft (CABG), SinoAtrial (SA) node, AtrioVentricular (AV) node, Electrocardiogram (ECG), Ventricular tachycardia (VT), Ventricular fibrillation (VF), Angiotensin converting enzyme inhibitor (ACE I), Cardio Pulmonary Bypass (CPB), Implantable cardiac defibrillator (ICD), Permanent Pacemaker (PPM), American college of Cardiology (ACC), American Heart Association (AHA), European society of Cardiology (ESC), Advanced cardiovascular life support (ACLS).

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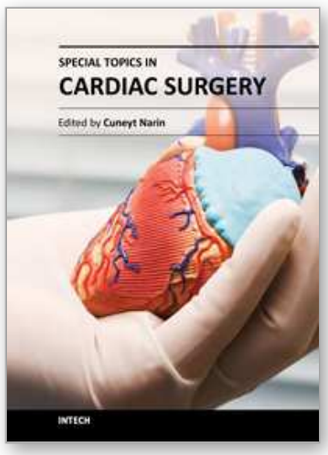

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This book considers mainly the current perioperative care, as well as progresses in new cardiac surgery technologies. Perioperative strategies and new technologies in the field of cardiac surgery will continue to contribute to improvements in postoperative outcomes and enable the cardiac surgical society to optimize surgical procedures. This book should prove to be a useful reference for trainees, senior surgeons and nurses in cardiac surgery, as well as anesthesiologists, perfusionists, and all the related health care workers who are involved in taking care of patients with heart disease which require surgical therapy. I hope these internationally cumulative and diligent efforts will provide patients undergoing cardiac surgery with meticulous perioperative care methods.

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