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ECG Monitoring After Cardiac Surgery

Postoperative Atrial Fibrillation and the Atrial Electrogram

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ABSTRACT

Atrial fibrillation is one of the most common complications after cardiac surgery and is associated with adverse outcomes such as increased mortality, neurological problems, longer hospitalizations, and increased cost of care. Major risk factors for the development of postoperative atrial fibrillation include older age and a history of atrial fibrillation. β-Blockers are the most effective preventive therapy, although sotalol and amiodarone can also be used for prophylaxis. In the postoperative period, the nurse plays an important role in the early detection of atrial fibrillation by the recording of an atrial electrogram, which is easily obtained from the bedside monitor. Because an atrial electrogram records larger atrial activity than ventricular activity, it can be invaluable in establishing the diagnosis of postoperative atrial fibrillation. Once atrial fibrillation begins, treatment can be started with either rhythm conversion or rate-controlling medications.
Keywords: atrial electrogram, ECG monitoring, postoperative atrial fibrillation

Atrial fibrillation (AF) is the most common arrhythmic complication of heart surgery, affecting more than one-third of patients, and is accompanied by increased morbidity and mortality, prolonged hospitalization, and increased cost of care.[1] For the past 30 years, postoperative AF has been the focus of intensive investigative efforts, yet there are few indications that this problem is decreasing, and, in fact, it may be increasing as the patients undergoing heart surgery get sicker and older. In the meantime, nurses who identify high-risk individuals and seek preventive measures before, and immediately following, surgery can aid in averting this challenging complication. Accurate diagnosis of AF by nurses is an important contribution that can be facilitated through use of the atrial electrogram (AEG), a simple-to-perform procedure for assessing atrial electrical activity. The nurse also administers and evaluates patients' responses to antiarrhythmic medications and monitors the patient for potentially lethal adverse effects. The purpose of this article is to provide the nurse with the background knowledge necessary to monitor and manage the patient with AF after cardiac surgery. The procedure for performing an AEG is emphasized.

Definition, Pathophysiology, and Etiology of Atrial Fibrillation

Postoperative AF has the same characteristics as AF due to other etiologies. In the 2006

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Guidelines for Management of Patients With Atrial Fibrillation, the American Heart Association, American College of Cardiology, and European Society of Cardiology defined *atrial fibrillation* as "a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. On the electrocardiogram (ECG), AF is characterized by the replacement of consistent P waves by rapid oscillations or fibrillatory waves that vary in amplitude, shape, and timing, associated with an irregular frequently rapid ventricular response when atrioventricular conduction is intact."[2] Physiologically, AF consists of multiple reentrant wavelets simultaneously propagating throughout the right and left atrium. Some of these circuits are circular in nature and some are spiral.[3] The circuit length controls the frequency of the reentrant tachycardia: the shorter the circuit, the faster the firing rate and the greater the control over the atria. Interventions that decrease the wavelength (such as vagal stimulation) reduce the circuit size and permit more reentry circuits to exist. Interventions that increase the wavelength (such as antiarrhythmic drugs) reduce the number of circuits and therefore extinguish AF.[3] The current atrial ablation procedures work in this way by creating a physiological barrier to circuit development and reentry. AF that is intermittent (paroxysmal) in nature is often due to irritable triggers in the area of the entry of the pulmonary veins in the left atrium, whereas permanent AF is most likely due to substrate changes in the atrial tissue and can be from the left or right atria.

In the patient who has undergone heart surgery, functional or physiological changes in the atrial tissue render it susceptible to initiation and maintenance of AF. It appears that in some individuals these substrate changes are present in the preoperative period, whereas for others they occur with the surgical process. The specific causes are still unknown, but the etiologies most supported by research include atrial and/or pericardial inflammation, sympathetic stimulation, myocardial ischemia, and/or dynamic volume changes in the atria.[4] Oral describes the mechanisms of AF as a spectrum with single drivers from within the pulmonary vein musculature on one end to the dynamic interaction of multiple mechanisms interacting at once on the opposite end.[5] The latter is very likely what is happening in the patient having heart surgery and partially explains why postoperative AF responds to so many interventions and varies in incidence with differing surgical procedures.

AF typically appears early after surgery, with 70% cases seen on postoperative days 2 to 4, but can first develop after discharge and is the leading cause of readmission.[6] Although in many cases it stops spontaneously, 43% of the individuals who develop AF have repeated episodes.[1]

AF related to surgery was first identified in 1953 as a complication of thoracic surgeries.[7] It then appeared as a complication of mitral commissurotomy procedures.[8] Even before the use of the cardiopulmonary bypass pump, postoperative AF was documented in heart surgery cases.[9] More recently it has been documented following pediatric cardiac procedures, heart transplant, and all types of valve and coronary artery bypass operations. The incidence of postoperative AF varies depending on a number of factors, including type of procedure, patient demographics, criteria for diagnosis, and methods of ECG monitoring. Rates for postoperative AF range from more than 30% for patients undergoing coronary artery bypass surgery to almost 60% for patients having combined coronary artery bypass and mitral valve surgery.[10] Off-pump surgery, minimally invasive procedures, and robotic-assisted surgery seem to be associated with a lower incidence of AF (Table 1). Although these early results are encouraging, it will be some time before these procedures are widely adapted. In addition, not all patients will be candidates for robotic-assisted operations.

Symptoms, Consequences, and Economic Impact

Atrial fibrillation of all types is primarily an arrhythmia of the elderly. Aranki et al found that the frequency of AF was 18% for patients younger than 60 undergoing heart surgery, but

Table 1: Incidence of Postoperative Atrial Fibrillation in Other Heart Surgeries

Off-pump CABG[37]	17.6%
Minimally invasive, direct CABG[38]	23%
Cardiac transplant[39]	9.5%
Robotic-assisted CABG[40]	8.5%

Abbreviation: CABG, coronary artery bypass graft.

52% for those older than 80.[11] Elderly individuals in general do not tolerate changes in the heart rhythm, and often become symptomatic. Individuals developing AF may exhibit lightheadedness, dizziness, chest pain, fatigue, shortness of breath, and anxiety and may lose consciousness if the heart rate is rapid and blood pressure falls. In addition, urine output can fall and patients may develop heart failure.[10] Funk et al, however, found that 69% of episodes of postoperative AF were not associated with any symptoms.[12]

AF has been linked with several negative consequences (Table 2). The 30-day surgical mortality rate is 6% when the patient has AF, compared with 3% when it is absent.[13] The 6-month mortality rate in patients who experience AF rises to 9.4%, from 4.2% in those who do not.[13] It can result in hemodynamic instability, trigger life-threatening ventricular arrhythmias, increase the risk for stroke, decrease neurocognitive function, require the implant of a permanent pacemaker, and increase length of stay and the cost of care.[14] In addition, the drugs used to treat postoperative AF can have proarrhythmic effects that increase the risk for torsades de pointes.

The costs of treating AF have only recently been addressed in the medical and nursing literature, yet AF can have a profound economic impact. A conservative estimate reveals that 500 000 heart surgery cases per year, with an incidence of AF of 20%, would result in 100 000 episodes of postoperative AF. The average additional cost of treatment and prolonged hospital stay and monitoring has been estimated to be $6000.[2] At this rate, the cost of treating postoperative AF is approximately $600 million annually. This estimate does not account for the postdischarge costs of medications or the impact of medications on the patient's quality of life.

Table 2: Consequences of Postoperative Atrial Fibrillation

- Increased in-hospital, 30-day, and 6-month mortality
- Hemodynamic instability
- Perioperative myocardial infarction
- Persistent heart failure symptoms
- Ventricular arrhythmias
- Stroke (1.2%–5.3%), diminished neurocognitive function, and neurological complications
- Need for permanent pacemakers
- Infectious, respiratory, and renal complications
- Multiorgan system failure
- Increased length of stay and cost of care
- Proarrhythmic effects of drug treatment

Preoperative Risk Assessment

Multiple risk factors have been identified for postoperative AF. Advanced age and a history of AF are the most consistently seen risk factors across multiple studies. Added to these are β-blocker withdrawal prior to surgery, obstructive lung disease, left ventricular hypertrophy, valve surgery, hypertension, and increased intraoperative ischemic times. Advanced age is by far the most consistent predictor of AF. For every 10-year increase in age, there is a 75% increase in the odds of developing postoperative AF; therefore, anyone older than 70 is considered to be at high risk.[1]

The advanced practice nurse is in a key position to identify high-risk individuals prior to surgery, to inform the surgical team of the increased risk, and to monitor for its development in the postoperative period. The nurse can ensure that if a patient has been on β-blocker medications, after surgery the patient's β-blocker medications are continued. If the patient has a history of preoperative AF, the nurse can make sure that prophylactic medications are started either during or immediately after surgery to prevent the development of AF.

In some cases, individuals who have a history of AF may undergo additional procedures during their heart operation to eliminate or reduce the occurrence of AF. These procedures include the Cox maze procedure, microwave ablation, radiofrequency-wave ablation, and cryoablation. The Cox maze procedure involves surgical incisions into the atrial tissue to create a scar barrier around excitable tissue and irritable foci. Microwave and radiofrequency-wave ablation involve the use of heated energy to create a similar type of scar barrier around areas of the atria where AF is believed to be developing. Cryoablation uses cooling energy in place of heated energy to perform the ablation. These procedures may accompany coronary artery bypass or valve procedures. In this situation, the nurse will be

preparing the patient for the additional surgical procedures. In all cases, patients require careful postoperative monitoring for AF. It is important to note that AF can occur even after a successful Cox maze procedure, microwave ablation, radiofrequency-wave ablation, or cryoablation procedure, because it takes months for the refractory period to lengthen and the scar tissue to mature fully. Early postoperative AF does not mean that the corrective procedure will not eventually work to eradicate or reduce the patient's chronic AF.

In most institutions, ECG monitoring is performed up to the time of discharge. When the patient develops any type of rapid heart rhythm, and particularly if its interpretation is in question, then the recording of an AEG may be beneficial.

Diagnosing Atrial Fibrillation: The Atrial Electrogram

The AEG is an electrocardiographic recording of atrial electrical activity taken directly from the surface of the right atrium via temporary atrial pacing wires that are routinely placed at the conclusion of most cardiac surgical procedures. Standard ECG monitoring records electrical events from the heart using electrodes on the surface of the patient's body, which is at a distance from the myocardium. One limitation of standard ECG monitoring is its inability to detect P waves effectively. AEGs detect electrical events directly from the atria, thus providing a greatly enhanced tracing of atrial activity. This enhanced tracing permits comparison of atrial events with ventricular events and determination of the relationship between the two.[15]

Furthermore, the AEG can be useful in diagnosing rapid atrial arrhythmias, junctional tachycardias, sinus tachycardias, and aberrancy versus ventricular tachycardias. It can also aid in distinguishing supraventricular from ventricular arrhythmias. During tachycardias, atrial activity is often not evident on the ECG tracing because P waves are buried within the larger QRS-T waveforms. Because an AEG records larger atrial activity (P, flutter, or fibrillatory waves) than ventricular activity (QRS complexes), it can be used to establish the mechanism of the tachycardia.[16,17]

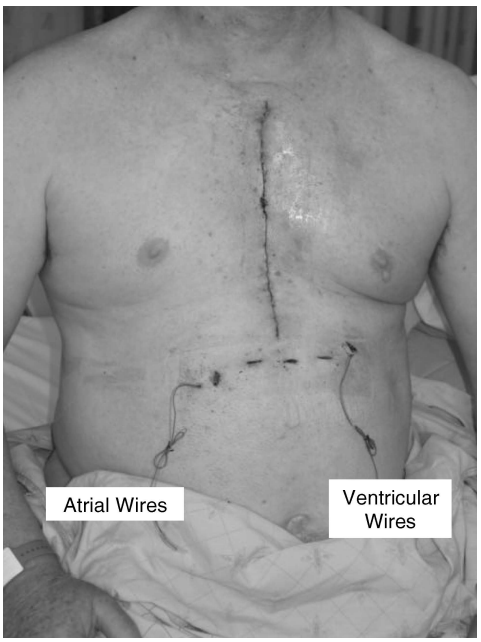
One of the earliest studies on the use of the AEG for the diagnosis of postoperative AF was published in 1978. Waldo and colleagues[18] retrospectively assessed the frequency of use of the AEG and found that in 70 consecutive cardiac surgical patients, AEGs were recorded 63 times in 34 patients. Forty-one times, AEGs were used to establish an arrhythmia diagnosis and 22 times to confirm the arrhythmia diagnosis originally suspected from the surface ECG. It was used to diagnose or confirm premature atrial and ventricular contractions, atrial fibrillation, atrial flutter, atrial tachycardias, aberrant atrioventricular conduction, junctional rhythm, ventricular tachycardia, atrioventricular block and dissociation, and sinus rhythm. In a study of pediatric patients who underwent congenital heart defect repair, atrioventricular conduction disturbances were found to be incorrectly diagnosed 23% of the time and narrow QRS tachycardias were incorrectly identified 86% of the time.[19] The errors in identification were detected by an AEG. Since this time, numerous anecdotal accounts of its benefits have been published.[20–23]

Procedure for Performing an Atrial Electrogram

An AEG can be recorded with a standard 12-lead ECG machine or with the bedside monitor. The simplest way to record an immediate AEG at the bedside is to unsnap the chest (V) lead wire from the patient's chest, hold it against the tip of the epicardial pacemaker lead wire so that metal is touching metal, and print a 15- to 30-second rhythm strip. Dual-channel ECG rhythm strips will display a selected limb lead on one channel and the AEG on the V channel. Gloves should be worn when handling epicardial pacemaker leads because even a small amount of current traveling through the wire directly to the heart can induce serious arrhythmias.[16,17]

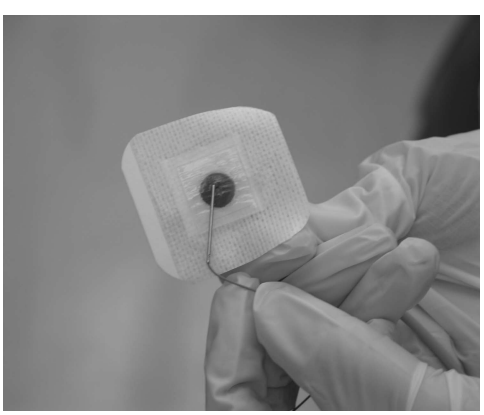
An alternative procedure for obtaining an AEG is shown in Figure 1. This procedure does not depend on the nurse to hold the atrial wire to the V lead wire, but rather employs the adhesiveness of the monitor electrode pad to maintain the contact. This procedure is preferable if longer-term monitoring is indicated to diagnose an unknown intermittent rhythm. A lead wire with alligator clips at both ends or an AEG-modified patient lead wire with an alligator clip at one end and the other end plugged into the V port can also be used to secure the connection to the atrial lead wire.[15]

Figure 2 shows AF as recorded on the surface ECG (upper tracing) and the corresponding AEG (lower tracing). The R waves are marked on the AEG tracing and the atrial



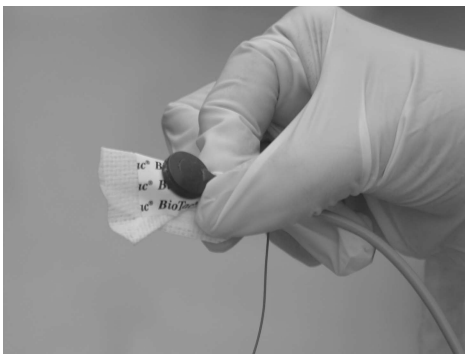
1A. Wearing gloves, carefully remove tape and dressing securing the atrial wires. Atrial wires typically exit the chest to the right of the patient's sternum and ventricular wires exit to the left of the patient's sternum.

1B. Prepare to attach atrial wire to V lead monitor electrode pad.

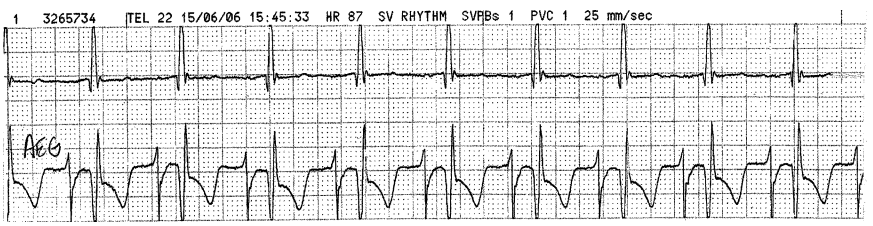


1C. Place the atrial wire onto the sticky side of the monitor electrode pad, ensuring that the wire is in contact with the conductive gel of the pad.

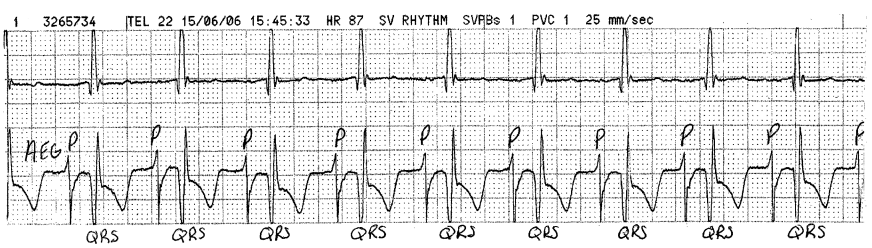
Figure 1: (*Continues*)



1D. Wrap the monitor electrode pad around the atrial wire. (If the electrode pads used are foam and too stiff to wrap around the wire, then a wire with alligator clips at both ends or an AEG-modified patient lead wire with an alligator clip at one end and the other end plugged into the V port can be used to secure the connection to the atrial lead wire).



1E. Set up your printer to print a simultaneous limb lead and the V lead that is attached to the atrial wire. Print a long strip of the rhythm. If the rate is rapid, it may be helpful to set the recorder to run at 50 mm/sec rather than the standard 25 mm/sec. This spreads out the complexes and makes them easier to see on the rhythm strip. You may leave the AEG displayed on the monitor (especially helpful if the patient is in and out of an undetermined rhythm). Be sure to indicate on the monitor that the "V" lead is an AEG.



1F. Label the printed rhythm strip with "AEG" next to the AEG recording. Determine the ventricular complexes on the AEG by aligning the QRS complexes with the deflections on the AEG. If there are spikes between the QRS complexes, these are atrial spikes. There will be one spike per P wave, flutter wave, or fibrillation wave. Once you have completed the recording, while wearing gloves, carefully detach the atrial wire from the monitor electrode pad. Re-insulate and redress the atrial wire according to your hospital policy. Place the labeled recording in the patient's medical record.

Figure 1: (*Continued*) Procedure for performing an atrial electrogram from a bedside electrocardiogram monitor. Abbreviation: AEG, atrial electrogram.

activity (fibrillatory waves) can be seen as the varying spikes between each R wave.

Preventing Atrial Fibrillation

The American College of Chest Physicians (ACCP) published evidence-based guidelines on the prevention and management of postoperative AF in 2005.[2] For the ACCP guideline section on prophylaxis, data from 91 studies were reviewed and summarized. The clinical recommendations for prophylaxis can be seen in Table 3.

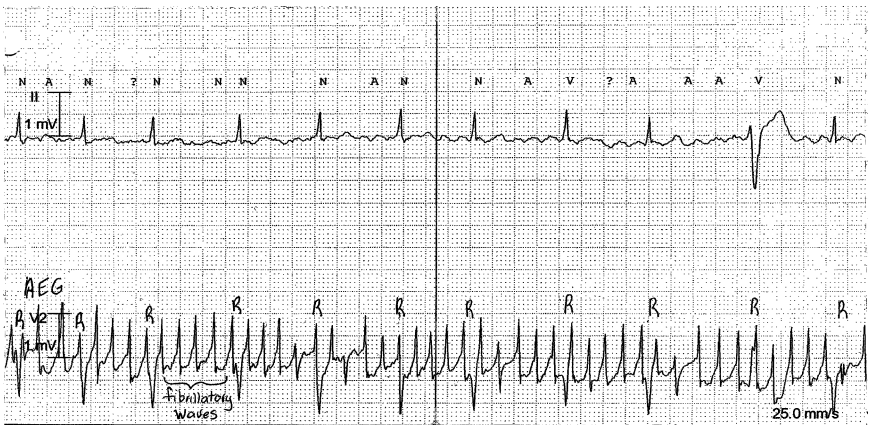


Figure 2: Atrial fibrillation as recorded on the surface electrocardiogram (upper tracing) and the corresponding atrial electrocardiogram (AEG) (lower tracing). The R waves are marked on the AEG tracing and the atrial activity (fibrillatory waves) can be seen as the varying spikes between R waves.

β-Blockers can significantly reduce the incidence of post–cardiac surgery AF, particularly in individuals who received long-term β-blocker therapy prior to surgery.[24] The rationale for the use of these drugs is the reduction in sympathetic tone induced by surgery. They reduce the proarrhythmic effects of catecholamines and inhibit the vagal withdrawal that accompanies ischemia. Sotalol, a β-blocker and potassium channel blocker, has also demonstrated the ability to decrease AF significantly. It has some advantages over pure β-blockers in its rapid onset of action (serum levels peak in 2–4 hours following oral administration). Seven of 8 trials reviewed found sotalol to be beneficial, although the reviewing panel notes

Table 3: Prophylaxis for Postoperative Atrial Fibrillation[24]

Drug	No. of Patients Analyzed	Reduction in Postoperative Atrial Fibrillation vs Control?	Strength of Recommendation[a]
β-Blockers	2901	Yes	A
Sotalol	1279	Yes	B
Amiodarone	1699	Yes	B
Verapamil	541	Inconclusive	D
Diltiazem	60	Inconclusive	D
Magnesium	1853	Inconclusive	D
Digoxin	1401	Inconclusive	I
Digoxin + propranolol	292	Yes	C
Dexamethasone	216	Yes	I
Glucose, insulin, potassium solution	102	Inconclusive	D
Insulin	501	Inconclusive	D
Triiodothyronine	301	Inconclusive	D
Procainamide	146	Inconclusive	D
Alinidine	32	Inconclusive	D
Quinidine	100	Inconclusive	D

[a]A = strong recommendation; B = moderate recommendation; C = weak recommendation; D = negative recommendation; I = no recommendation possible, or inconclusive.

that there were several limitations to these trials. The adverse effects of sotalol, including hypotension and/or bradycardia, are no more frequent than with other β-blockers, but there is a risk for QT-interval prolongation and torsades de pointes; therefore, patients receiving sotalol should have the QT interval measured every 8 hours. It is also important to note that the studies on sotalol and class II β-receptor antagonists were limited to patients who did *not* have low left ventricular ejection fraction, bradycardia, emphysema, or renal insufficiency. Therefore, these results cannot be generalized to these patient groups. Of note, sotalol may be the preferred drug for individuals with implantable defibrillators because it does not elevate the defibrillation threshold.[25]

Amiodarone has been evaluated in 10 randomized controlled trials. In reviewing these trials, the ACCP panel found that in only 4 of the 10 trials was amiodarone associated with a statistically significant reduction in AF after cardiac surgery.[2] Although amiodarone can reduce the incidence of AF by 50%, this requires preoperative loading for 7 days, which may be impractical given same-day surgery admissions. Intravenous loading of amiodarone within 3 hours after surgery can reduce the risk for AF by 26%, but there is a higher incidence of hypotension associated with this route.[26] Kerstein et al tested a combination of intravenous and oral amiodarone and found that the combination resulted in a statistically significant decrease in the development of AF: 5.88% in the amiodarone group versus 26.08% in the placebo group.[27] They also found that length of stay was decreased with amiodarone. In the Reduction in Postoperative Cardiovascular Arrhythmic Events (REDUCE) trial, Mooss et al compared amiodarone to sotalol.[28] This was also a combination of intravenous amiodarone (15 mg/kg over 24 hours followed by 200 mg orally 3 times per day), which was compared to sotalol 80 mg twice daily. Patients were randomized to 1 of these 2 groups and both drugs were started prior to surgery, with sotalol being started 2 hours before surgery and amiodarone initiated at the start of surgery. The incidence of AF was 17% in the amiodarone group and 25% in the sotalol group ($P = .21$).[28] The duration of AF was significantly shorter in the amiodarone group ($P = .04$). Amiodarone use was more likely to be accompanied by hypotension, lowered pulmonary artery pressures, and lowered systemic vascular resistance within the first 24 hours after surgery compared with sotalol. The sotalol group required increased inotropic support, increased vasopressor support, and an increased need for pacing, with a higher overall morbidity and mortality; however, none of these proved to be statistically significant differences, possibly because of the sample sizes. Although this study had many positive aspects, the investigators suggest that it may have been underpowered.[28] In addition, the doses used were not fundamentally equivalent; low-dose sotalol was compared with high-dose amiodarone. Clearly a larger study with drug doses of similar efficacy would be very beneficial.

While awaiting further trial results, the recommendation of the ACCP panel remains relevant, that the preferred antiarrhythmic drugs for prevention of post–cardiac surgery AF are the class II β-receptor antagonists. Sotalol may be considered, but it is associated with an increased risk for toxicity. When β-blockers are contraindicated (eg, in obstructive lung disease or asthma), then amiodarone should be considered. The guidelines go on to state that calcium channel antagonists and magnesium are not beneficial and are not recommended. Digitalis, when used alone, is also nonbeneficial. The other agents listed in Table 3 have not (at the time of this writing) undergone sufficient clinical study to merit recommendations.

Rhythm Control After Surgery

At this time there has been no large, randomized, controlled, blinded trial to compare rhythm control with rate control in the patient who develops postoperative AF. In the setting of heart surgery, conversion to sinus rhythm is desirable because of the concerns with anticoagulation, the risk for hemodynamic instability, and undesirable symptoms related to AF. It is important to note, however, that rhythm conversion should not be attempted in the patient with chronic AF that is not being addressed by surgical ablation.

The authors of the ACCP guidelines were able to find only 19 studies that met their inclusion criteria for review that addressed rhythm control.[29] The strength of the recommendations for antiarrhythmic therapy for conversion was generally weak with negative recommendation (D rating) or inconclusive results with no

Table 4: Pharmacologic Rhythm Control of Postoperative Atrial Fibrillation or Atrial Flutter[29]

Drug	Strength of Recommendation[a]	Evidence Grade	Net Benefit
Amiodarone	E/C	Low	Intermediate
Sotalol	C	Low	Intermediate
Class IA antiarrhythmic	C	Low	Small and weak
Ibutilide	C	Low	Small and weak
β-Blockers	I	Low	None
Calcium channel blockers	I	Low	None
Digoxin	I	Low	None
Class IC	D	Low	Negative
Dofetilide	D	Low	Negative

[a]C = weak recommendation; D = negative recommendation; E/C = weak recommendation based on expert opinion only; I = no recommendation possible, or inconclusive.

recommendation (I rating). The results of their review and synthesis are shown in Table 4. In the presence of depressed left ventricular function, the guidelines recommend use of amiodarone. Patients with normal left ventricular function may receive amiodarone, sotalol, ibutilide, or class 1A agents (procainamide, quinidine, or disopyramide) for conversion of AF.[29] Intravenous procainamide often causes hypotension and should not be used in the presence of renal dysfunction. Four to 6 weeks of antiarrhythmic therapy is recommended. Drugs that were not recommended for conversion of AF included flecainide, digoxin, and calcium channel blockers. The panel recommended against the use of dofetilide for the conversion of AF to sinus rhythm as it is suspected to be associated with an increased risk for toxicity and ventricular arrhythmias, and, at this time, the evidence for its safety and efficacy is weak.[29]

Control of Ventricular Rate in Postoperative AF

Guideline developers found only 9 studies addressing rate control in the patient with postoperative AF.[30] They recommend β-blocker therapy as the first choice for rate control, followed by calcium channel blockers (diltiazem and verapamil) as second choices. Although amiodarone may be used, it is not one of the top choices because of the bradycardia and/or respiratory dysfunction that may develop. Digoxin is also rejected for rate control as it does not have any effect on adrenergic tone. Finally, the guidelines panel recommends against the use of any drugs that "may be, or have been shown to be, pro-arrhythmic."[30] They specifically point out that propafenone has a potential for bradycardia in this population and dofetilide, again, is not efficacious and is associated with proarrhythmic adverse effects.[30] Anticoagulation is necessary in the setting of AF unless the risks of anticoagulation outweigh the risks of stroke.[31]

Seeking New Possibilities: The Role of Anti-inflammatory Agents

Over the years, multiple researchers have theorized about the role of inflammatory changes in the development of AF. Many have noted that the peak incidence of AF is on the second and third postoperative days, which correspond to the pericardial friction rub that is heard on auscultation. Further evidence for an inflammatory link has been the demonstration of elevated levels of C-reactive protein and interleukin-6, increased leukocyte count, and red blood cell transfusions as being associated with an increased risk for new-onset AF.[32–34] A number of studies demonstrated that a high-sensitivity C-reactive protein level was associated with AF and is a predictor of early AF relapse after successful cardioversion.[32]

Very recently, statin drugs have been advocated for the prevention of postoperative AF,

adding more evidence for an inflammatory etiology. The Atorvastatin for Reduction of Myocardial Dysrhythmia After Cardiac Surgery (ARMYDA-3) study was conducted on 200 patients undergoing elective cardiac surgery with cardiopulmonary bypass, who had not previously used statins and had no history of AF prior to surgery.[35] Patients were randomized to receive atorvastatin 40 mg/d, starting 7 days before surgery, or to placebo. The primary end point was incidence of AF, with secondary end points of length of stay, 30-day major adverse cardiac and cerebrovascular events, and postoperative C-reactive protein variations. Atorvastatin was associated with a statistically significant reduction in the incidence of AF, compared with placebo (35% vs 57%, $P = .003$), and a statistically significant reduction in length of stay ($P = .001$). Peak C-reactive protein levels were lower in patients without AF ($P = .01$). The investigators calculated that treatment with atorvastatin resulted in a 61% risk reduction in AF (odds ratio, 0.39; 95% confidence interval, 0.18–0.85) and found that elevated levels of C-reactive protein were associated with an increased risk for AF (odds ratio, 2.0; 95% confidence interval, 1.2–7.0).[35] The investigators suggest that the anti-inflammatory, antioxidant, and membrane ion stabilization effects of atorvastatin may have accounted for its beneficial effects.

In a randomized clinical trial of 241 patients reported in 2007, Halonen et al[36] showed that the administration of 100 mg of intravenous hydrocortisone the evening of the operative day, then 1 dose every 8 hours during the next 3 days, significantly reduced the incidence of AF after cardiac surgery. All patients also received oral metoprolol (50–150 mg/d) titrated to heart rate. The incidence of postoperative AF was significantly lower in the hydrocortisone group (30%) than in the placebo group (48%; adjusted hazard ratio, 0.54; 95% confidence interval, 0.35–0.83; $P = .004$). Compared with patients in the placebo group, patients receiving hydrocortisone did not have higher rates of superficial or deep wound infections, or other major complications. This trial adds further evidence to support the inflammatory etiology of postoperative AF.

According to the 2005 ACCP Guidelines, however, corticosteroid drugs "do not appear to be beneficial and, in fact, may be detrimental."[2] Clearly, the role of inflammation in the development of AF needs further study.

Summary

Postoperative AF has been a major problem after heart surgery for more than 30 years. There are currently no medications that are even 90% effective in preventing AF and most antiarrhythmic drugs influence ventricular electrical properties in a way that may lead to adverse events. Of all the medications that are available, β-blockers remain the most effective preventive therapy. Sotalol and amiodarone can also be used for prophylaxis. New drugs that are currently in development include atrial-selective antiarrhythmic drugs, amiodarone congeners, and stretch-activated channel blockers that offer hope for better management of postoperative AF.

In the early postoperative period, the nurse plays an important role in the prompt detection of AF. Because an AEG records larger atrial activity than ventricular activity, it can be invaluable in establishing the diagnosis of postoperative AF. Once AF begins, treatment can be started with either rhythm conversion or rate-controlling medications. At this time, heart surgery procedures are undergoing significant evolutionary advances with the development and increased use of thoracoscopic and robotic procedures that eliminate the need for cardiopulmonary bypass and, in many cases, the need for a medial sternotomy incision. These modifications of surgical technique decrease surgical time and there is hope that they may alter the pathology that supports the development of postoperative AF.

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