Diagnosis and Management of Perioperative Cardiac Dysrhythmias

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Continuous monitoring techniques can identify transient dysrhythmias in the majority of elderly patients undergoing major surgery. Ventricular dysrhythmias requiring drug treatment are relatively uncommon and are usually the sign of a serious complication such as myocardial ischemia or infarction. Especially in cardiac surgery patients, atrial dysrhythmias in the early postoperative period (ie, first 24 hours) are associated with severe ventricular dysfunction and the requirement for inotropic therapy. These early rhythm disturbances should be viewed separately from the very common atrial fibrillation/flutter which occurs 2-3 days postoperatively in cardiac surgery patients. The latter rhythm occurs in up to 50% of patients and has major implications in terms of length of stay and cost.

Etiology

Cardiac rhythm disturbances are seen in the context of structural heart disease, when there are superimposed "transient imbalances."[1] The importance of transient imbalances is the need for the clinician to correct any abnormalities that can be treated. These include hypoxemia or hyper/hypocarbia, electrolyte deficiency or excess, hypothermia, acid-base derangements, and myocardial ischemia. Many of these are interrelated: hypocarbia causes acute alkalosis and hypokalemia. Less commonly autonomic nervous system imbalance, endocrine disorders (eg hypothyroidism) or mechanical irritants may be contributing.

In cardiac surgery patients, despite the administration of hyperkalemic cardioplegia solutions the majority of patients develop either relative or absolute hypokalemia in the early postoperative period. This may be the result of the very high levels of catecholamines (causing intracellular shift of potassium), hemodilution, and diuretic induced losses. Perioperative hypokalemia is associated with an increased risk of dysrhythmia and other adverse cardiac outcomes.[2] While restoration of a significant potassium deficit may take days, serum levels should be measured and administration of this ion should be considered in all patients with refractory tachydysrhythmias. Similarly, although less well understood, magnesium deficiency is frequently observed after cardiac surgery, and is also associated with tachydysrhythmias.[3] Magnesium is required for the Na+/K+ and Na+/Ca++ exchange pumps and deficiency of this ion appears linked both to hypokalemia and hypocalcemia. Supplementation of magnesium should be considered in most patients, even without verification of serum levels (an exception is the patient with renal failure).

Mechanism of dysrhythmias

The three major mechanisms responsible for cardiac dysrhythmias are *abnormal automaticity* (eg, sinus tachy and bradycardias), *reentry* which also requires abnormal conduction (majority of supraventricular tachydysrhythmias) and *triggered activity*. Each of these mechanisms can occur alone or in combination in areas of myocardium that are structurally abnormal, or rendered abnormal by an acute process such as ischemia or mechanical irritation. Antidysrhythmic drugs have differing actions that may be more or less effective in each type of pathophysiology. In addition, antidysrhythmic drugs may themselves promote dysrhythmias ("proarrhythmia"). The commonest of these is polymorphic ventricular tachycardia or "torsade de pointes," also associated with QT interval prolongation.

As mentioned above, refractory dysrhythmias usually occur in the context of severe ventricular dysfunction (and often atrial distension), often caused by myocardial ischemia. While attempting to control or convert refractory dysrhythmias, attention must be given to identifying correctible causes of ischemia (eg, hypotension, graft occlusion or spasm).

Diagnosis

Specific diagnosis of dysrhythmias requires a high-quality ECG printout, usually of two or more leads. In the cardiac surgical patient, many centers routinely place temporary atrial wires for pacing. These wires can be used as either unipolar (eg, using the V electrode) or bipolar (using the electrodes which comprise standard leads I, II, or III) leads to display the atrial activity, and are very useful in determining the atrial rhythm in tachydysrhythmias. Alternatively, in the intubated and sedated patient, placement of an esophageal electrode is simple and can provide similar information. These leads can be paired with a standard limb lead (eg V5 or lead II) for printout, as the very large atrial spike or p-wave from epicardial or esophageal leads can be confused with a QRS complex. Short of these aids, a multi-lead surface ECGs (ie, in the ICU a standard 12-lead ECG) should be obtained.

Prevention

All antidysrhythmic drugs have the potential for adverse side effects. The most serious of these is "proarrhythmia" as mentioned above. This is most commonly associated with drugs which predominantly affect sodium channels or potassium channels (Vaughn Williams class I and III agents). Other drugs may have less potential for proarrhythmia, but may cause bradycardia and/or hypotension (eg, beta blockers, calcium channel blockers). Therefore, the prophylactic use of antidysrhythmic drugs is discouraged. The major exception is the use of prophylaxis for postoperative atrial fibrillation.

More than 50 publications have addressed prevention of atrial fibrillation after cardiac surgery. This is because of the very high incidence, the association with adverse outcome (eg, stroke) and the need for additional hospital days to treat or control the rhythm.[4] Rate control is necessary in order to reduce symptoms and reduce myocardial oxygen consumption, and restoration of sinus rhythm improves cardiac output in most patients.

Prolonged atrial fibrillation/flutter is associated with an annual 5% risk of thromboembolism from atrial clot. [5]

A meta-analysis of studies performed before 1990 clearly demonstrates that while beta blockers, digoxin and verapamil (the only intravenous calcium channel blocker with AV nodal blocking action available at that time) all can control the ventricular rate, only beta blockers can reduce the incidence.[6] Recent studies of amiodarone suggest that this drug may also reduce the incidence. A study from the University of Michigan indicated that oral amiodarone for a mean of 12 days preoperatively reduced the incidence of atrial fibrillation lasting at least 5 minutes from more than 50% to about 25%.[7] Problems with this study are the applicability to most American practices (few surgeries are scheduled more than a week in advance), and the very high incidence in the control group. An earlier British study found that intravenous amiodarone given for 24 hours after the aortic cross clamp was removed reduced the incidence of dysrhythmias requiring treatment from 23 to 10%, but many patients developed bradycardia.[8] In the United States the intravenous loading dose of amiodarone (1 gram) costs several hundred dollars to administer.

Oral beta blockers are recommended for post cardiac surgery atrial fibrillation prophylaxis in a consensus publication by the American and European societies of Cardiology and the North American Society of Pacing and Electrophysiology. [8a] Propranolol, metoprolol or atenolol are examples of drugs that have been shown to be effective at reducing the incidence by approximately 50%. The treatment is started as soon after surgery as possible, and continued until the postoperative followup visit 4-6 weeks later. Amiodarone, or the nonselective beta blocking drug sotalol (which has some class III activity) are also mentioned in this recommendation. Intraoperative ablation of the left atrium in the area of the pulmonary venous inflow, and postoperative atrial pacing are nonpharmacologic techniques that have been reported to reduce the incidence of postoperative atrial fibrillation.[8b,c]

Treatment

(a) Supraventricular tachydysrhythmias.

As discussed above, all possible contributing factors should be addressed either before or during acute intervention. Supplementation of potassium and magnesium may be all that is required in some patients. If the patient is receiving catecholamines control of the ventricular rate may be a more realistic goal than conversion to sinus rhythm. If the diagnosis is uncertain (eg paroxysmal atrial tachycardia vs atrial flutter), a dose of adenosine can be administered. A 6-12 mg intravenous dose of this drug causes profound AV nodal blockade for 10-30 seconds in most patients, and will convert reentrant rhythms involving the AV node. Alternatively it may reveal flutter waves. In the case of atrial flutter, if epicardial atrial wires are available overdrive pacing can be effective in restoring sinus rhythm.

In atrial fibrillation/flutter, pharmacologic therapy is designed to reduce the ventricular rate and convert/maintain normal sinus rhythm. Specific drugs may be more or less effective for these two goals. Intravenous esmolol or diltiazem are both effective at controlling the rate, however esmolol may be more effective at conversion.[9] Procainamide may convert the rhythm to sinus but will not control the ventricular response, while amiodarone may do both. Both sotalol and amiodarone are more effective than digoxin for conversion of new onset atrial fibrillation. [10]

Ibutilide is a recent addition to the antidysrhythmic pharmacopaea. It is a Vaughan Williams Class III drug (prolongation of repolarization) with a rapid onset and relatively short duration of action, indicated for the conversion of acute atrial fibrillation/flutter to sinus rhythm. It appears to be somewhat more effective in atrial flutter than fibrillation, converting 35-60% of patients.[11] The drawback to this drug is a significant incidence of proarrhythmia which increases as ventricular function worsens. There are no published studies of the use of ibutilide in anesthetized patients.

Direct current synchronized cardioversion is the treatment of choice in patients with tachydysrhythmias associated with hypotension or significant "hemodynamic compromise." During or after major surgery this treatment is likely to be ineffective or only temporarily effective until the underlying cause for the dysrhythmia is addressed (eg volume overload, diuretic therapy, or the presence of catecholamine infusions).

The pathophysiology and treatment of atrial fibrillation after cardiac surgery have recently been reviewed by Hill et al,[12,13] and surgical options including intraoperative ablation of atria are reviewed by Gillinov et al.[14]

(b) Ventricular tachydysrhythmias

The first line treatment for hemodynamically unstable ventricular tachydysrhythmias is direct current cardioversion. The first line antidysrhythmic drug this rhythm with stable hemodynamics, or to prevent recurrence has historically been lidocaine, administered as a load (1.5 - 2 mg/kg) followed by an infusion (2 mg/min) at the time of the cardioversion. As mentioned above, unless Mg++ levels have been documented as elevated or a dose has been given recently, in the cardiac surgical patient a dose of 1-2 grams of Mg++ should be given and the serum level of K+ measured. Any metabolic abnormalites should be treated, and if catecholamine infusions are running consideration should be given to using the least arrhythmogenic combination of drugs (eg, use of norepinephrine and milrinone appears less likely to exacerbate dysrhythmias than epinephrine or dobutamine).

Second line drug treatment for refractory ventricular dysrhythmias has traditionally been another class 1 drug (eg procainamide) or the class III drug, bretylium. Recent publication of revised guidelines for the treatment of life threatening cardiac dysrhythmias has moved amiodarone from a 3rd line drug to either first line, or second after lidocaine. [15] Amiodarone compares favorably to bretylium in medical patients with refractory ventricular dysrhythmias, being equally effective and less likely to cause hypotension.[16] In patients suffering cardiac arrest out-of-hospital, amiodarone improves early survival vs placebo [17] and vs lidocaine. [18] There is no such supporting evidence for any other antidysrhythmic drug. While some concerns exist about the use of amiodarone in the cardiac surgical patient (early reports of refractory hypotension and bradyarrhythmias, and case reports of postoperative ARDS[19]), these concerns are outweighed by the effectivenss of amiodarone and the hemodynamic profile and lack of proarrhythmia effects in comparison with alternative drugs.

The foregoing discussion does not distinguish between monomorphic vs polymorphic ventricular tachycardia ("torsade des pointes"), or ventricular fibrillation/pulseless ventricular tachycardia. For the latter, clearly defibrillation is the treatment of choice and should never be delayed in favor of pharmacotherapy. Hemodynamically stable monomorphic ventricular tachycardia can be converted with procainamide, sotalol, amiodarone or beta blockers more effectively than with lidocaine. Polymorphic ventricular tachycardia (“torsade de pointes”) is often drug or electrolyte related, and pharmacotherapy may or may not be effective.

Summary

Refractory postoperative dysrhythmias should be viewed as the expression of a serious underlying disorder such as ischemia. Many treatable factors can exacerbate or initiate abnormal rhythms; hypomagnesemia and hypokalemia are common in this setting. Many drugs can control the ventricular rate in atrial tachydysrhythmias, but fewer drugs facilitate conversion to sinus rhythm. Prophylaxis of postoperative atrial fibrillation/flutter can be achieved with oral beta blocking drugs given after surgery and oral or IV amiodarone. Oral amiodarone must be given for at least a week before surgery and IV amiodarone is very expensive, leaving (postoperative) beta blockers as the most practical choice for prophylaxis. For refractory ventricular dysrhythmias, amiodarone is gaining favor as first-line therapy.

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