Atrial fibrillation: An anesthesiologist’s perspective

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Abstract

Atrial fibrillation (AF) is not only the most common arrhythmia in the global population but also the most frequent one encountered in the operating room. For an anesthesiologist, it is crucial to have the ability to maintain hemodynamics and prevent complications of patients who present AF perioperatively. Here we provide a brief review in the novel concept of the classification, pathophysiology, and management of AF to provide a practical approach for physicians coming across this arrhythmia during the perioperative period.

1. Introduction

Atrial fibrillation (AF), the most common cardiac arrhythmia, is a disorder of electrical activity in the atrial myocytes. AF causes many complications, including thromboembolic events, congestive heart failure, and even death. It affects over 5 million people globally1 as the prevalence increases from 1% in individuals aged > 60 years, 7.2% in individuals aged > 65 years, and to 10% in individuals aged > 75 years2,3 and is responsible for substantial morbidity and mortality.

2. Definition, diagnosis and classification

The published guidelines from an American College of Cardiology/American Heart Association/European Society of Cardiology committee of experts defined AF as rapid oscillation and/or fibrillation of the atria due to irregular electrical activity which varies in amplitude, shape, and timing, whereas the atrioventricular conduction remains intact. Initial diagnosis is based on the surface electrocardiogram (ECG) which shows no distinct P waves, absolutely irregular RR intervals, and a short atrial cycle of 200 ms or less (> 300 beats/min).3−5 AF can be classified as three forms: paroxysmal, persistent, and permanent (Table 1).

3. Risk factors and pathophysiology

Although AF is easily detected by ECG, many patients are asymptomatic and remain undiagnosed until a secondary event occurs (e.g., stroke). The predisposing factors can be cardiovascular and/or noncardiovascular in origin. For instance, myocardial infarction, cardiomyopathy, hypertension, valvular disease, and heart failure are well-known risk factors of cardiovascular origin; whereas diabetes, obesity, alcohol intake (“holiday heart syndrome”), hyperthyroidism, chronic obstructive pulmonary disease, and other metabolic disorders are noncardiovascular causes.2−7 The dogmatic point of view has been that AF results from a pathological remodeling of the left atrium due to fibrosis and muscular atrophy. Nevertheless, emerging evidence suggests a pivotal role of inflammatory pathways via leukocyte signaling in the pathophysiology of AF. Proinflammatory cytokines and hormones, such as angiotensin II, tumor necrosis factor-α, and interleukins-6 and -8, trigger myocardial dysfunction by activating local leukocytes and cardiac fibroblasts. The resulting cardiomyocytic apoptosis in turn triggers massive cardiac proliferation and differentiation, leading to dysfunction of connexin and ion channels.

“Lone AF”, which describes AF in patients aged < 60 years without structural heart diseases, has been strongly associated with a novel loss-of-function mutation in the genes encoding connexin 40 (GJA5) and connexin 43 (GJA1).8,9 In comparison, “familial AF” has been associated common variants on the 4q25.
Table 1
Classification of atrial fibrillation.

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<tr>
<th>Classification</th>
<th>Presented pattern of first detected episode</th>
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<tr>
<td>Paroxysmal</td>
<td>Lasting for ≤ 48 h, usually terminates spontaneously</td>
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<tr>
<td>Persistent</td>
<td>Lasting for &gt; 7 d, cardioversion may be needed</td>
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<tr>
<td></td>
<td>Long-standing persistent: lasting for &gt; 1 y, cardioversion failed or not attempted</td>
</tr>
<tr>
<td>Permanent</td>
<td>Long-term atrial fibrillation, no pursuit of rhythm control</td>
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16q22, and 1q21 chromosomes. The above schema suggests that genetic factors play an important role in the development of AF.

4. Management

Anesthesiologists frequently deal with AF in the operating room. The classification of AF (paroxysmal, persistent, and permanent) can be used as a guide for the clinician to determine perioperatively whether the patient should be further managed. In patients with fresh AF occurring during perioperative period, their hemodynamic status must be first evaluated. With a life-threatening condition caused by AF, the arrhythmia must be terminated promptly. A rapid ventricular rate that induces hemodynamic instability is a medical emergency requiring electrical cardioversion. Managing patients with symptomatic and life-threatening AF is to restore sinus rhythm and to stabilize hemodynamics. Asynchronized mode must be used to avoid ventricular fibrillation in case a monophasic orbiphasic defibrillator is employed. Direct transthoracic cardioversion is a well-established treatment for restoring sinus rhythm. Application of high-energy shocks intrathoracic cardioversion may cause patient discomfort and pain, and thus anesthesia is required. If AF occurs during an open heart surgery, epicardial atrial defibrillation which needs very low shock energy (<9 J) would offer a very good help in maintaining sinus rhythm postoperatively without sedation and analgesia. In patients with persistent or permanent AF coupled with stable hemodynamics, a strategy of rate control or rhythm control could be chosen.

4.1. Rate control

For patients with AF in whom the duration of the AF is unknown or known to be >48 hours, a rate control strategy is preferable until adequate anticoagulation is achieved or intracardiac thrombosis is ruled out through echocardiogram. Furthermore, rate control may be the initial approach in patients in whom structural remodeling is expected to progress well and their arrhythmia has developed to permanent AF. Rate control aims to control the ventricular rate without restoring the arrhythmia to sinus rhythm, which is mainly achieved by administering rate-controlling medications (Table 2). Commonly used rate-controlling drugs have side effects (such as hypotension, bradycardia, heart block, and heart failure), which may deteriorate the hemodynamics under anesthesia. Small initial dose and a continuous infusion of a rate-controlling drug (e.g., esmolol) titrated to an appropriate hemodynamic point may decrease incidence of adverse effects. Even in recent years, the preferred rate-control intervention for AF, whether be it lenient or strict, remains largely debatable. The Rate Control Efficacy in Permanent Atrial Fibrillation: a Comparison between Lenient versus Strict Rate Control II (RACE II) study concluded that lenient rate control (<100 beats/min) is as effective as strict rate control (<80 beats/min) with similar outcomes. There is no significant difference in the prevalence of AF symptoms in patients with a left ventricular ejection fraction >40%. Lenient heart rate control parameters may be easier to achieve for the anesthesiologists, and thereby making lenient rate control as a reasonable strategy.

4.2. Rhythm control

Patients with recent-onset atrial fibrillation could generally have it convert back to sinus rhythm spontaneously. If the patient does not achieve this, pharmacological or electrical cardioversion can be considered. However, the benefits of restoring sinus rhythm must be prudently weighed against the long-term sequelae of antiarrhythmic therapy. Current consensus for rhythm control is to convert sinus rhythm for patients who are still symptomatic (AF causes acute heart failure, hypotension, or worsening of angina pectoris) despite ventricular rate control. In general, electrical cardioversion is faster, more effective, and more efficient than pharmacological cardioversion. After successful electrical cardioversion, long-term pharmacological support may be required to prevent recurrence of AF. Vaughan Williams class I antiarrhythmics, including flecainide, dofetilide, propafenone, and ibutilide are recommended with level of evidence A for pharmacological cardioversion of AF. Furthermore, administration of amiodarone is a reasonable option for pharmacological cardioversion of AF.

Other novel strategies have been developed to treat AF. Catheter ablation of AF yielded the benefits of lower rates of stroke and death. “Ablate and pace” therapy, which is to permanently pace the ventricles with cardiac resynchronisation after atrioventricular nodal ablation, has been shown to improve heart failure and quality of life. Surgical intervention to prevent macroreentrant rhythms of AF with the “maze” procedure during mitral valve surgery could maintain atrial transport function and, when combined with amputation or obliteration of the left atrial appendage (LA), postoperative thromboembolic events are substantially reduced.

5. Echocardiography

The risk of intracardiac thrombus formation is greatest when the arrhythmia has been present for >48 h. Although transesophageal
echocardiogram (TEE) is not part of the standard initial investigation of patients with AF, it is still very helpful in management of patients with AF. The ability to visualize left-side cardiac structures (LAA) with TEE is significantly superior to transthoracic echocardiogram. It is the most sensitive and specific technique to detect sources and potential mechanisms of cardiogenic embolism. The technology has been also used to stratify stroke risk in patients with AF and to guide cardioversion. A study of TEE in patients with AF prior to cardioversion showed that LA or LAA thrombus incidence is 5–15%.

In conclusion, AF is the most common arrhythmia that an anesthesiologist may face and it actually increases morbidity and mortality. The anesthesiologist must be proficient in the management of patients with symptomatic AF and hemodynamic instability during the perioperative period. Immediate intervention with electrical cardioversion and following pharmacological support are always the way of initiate treatment. Also, it is crucial to be aware of the causes, consequences, and therapies of patients with new-onset AF or patients with chronic AF when we come across them in the operating room.

References