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Cardiac Arrhythmias: Management of Atrial Fibrillation in the Critically Ill Patient

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Atrial fibrillation (AF) is a common arrhythmia in the ICU, second only to ventricular tachycardia. A study of prevalence of arrhythmias showed that AF may occur in up to 31% of patients in medical, cardiac, and surgical ICUs [1]. AF is associated with a significantly longer ICU stay. In-hospital mortality associated with acute myocardial infarction (MI) is higher in patients who have AF (25% versus 16%) [2]. AF is associated with a twofold increase in mortality in the community, and it is influenced by the severity of underlying heart disease [3,4].

It is estimated that 2.2 million people in the United States have AF. The prevalence of this disease increases with age. The burden of AF is expected to rise as population ages, and it will continue to be associated with significant health care costs [5].

Etiology and associated conditions

AF is associated with various cardiac or extracardiac conditions, some of which are chronic, while others are short lived. In the setting of acute illness, AF may present de novo, or it may recur in patients who have a history of AF. Surgery, especially cardiac or thoracic, pulmonary embolism or other pulmonary conditions, myocarditis, electrocution, alcohol consumption, thyroid disorders, and other metabolic conditions may contribute to the development of AF.

AF also often is associated with history of hypertension and coronary artery disease (CAD). Eleven percent of patients presenting with acute MI

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develop AF during their hospitalization [6]. Left ventricular (LV) hypertrophy and associated diastolic dysfunction likely play a role in the genesis of AF because of an increase in stretch in the left atrium or the pulmonary veins [7]. Valvular heart disease, especially mitral stenosis and mitral regurgitation resulting in left atrial (LA) dilatation, commonly are associated with atrial arrhythmias. AF also may be related to hypertrophic cardiomyopathy (HCM) or dilated cardiomyopathy, and various forms of congenital heart disease in adults, especially atrial septal defect. Less common causes of AF include restrictive cardiomyopathies (such as amyloidosis, hemochromatosis, and endomyocardial fibrosis), pericarditis, and cardiac tumors. The known causes of AF are listed in Box 1.

Among noncardiac chronic conditions, obesity is an important, recently identified risk factor for developing AF [8,9]. There is a direct relationship between LA size and body mass index (BMI). An association between obstructive sleep apnea (OSA) and AF also has been reported [10].

The term lone AF is used to describe AF in individuals younger than 60 years of age who have no clinical or echocardiographic evidence of cardiac disease. In approximately 45% of patients who have paroxysmal AF and 25% of patients who have chronic AF, no cardiac disease can be identified [11]. Fluctuations in autonomic tone appear to play a role in the initiation of AF, especially in structurally normal hearts. Both vagal and sympathetic tone may surge in the minutes that precede initiation of AF [12,13].

Definitions

Various terms have been used in the literature to describe patterns of AF occurrence. The North American Society of Pacing and Electrophysiology and the European Society of Cardiology recently endorsed a new nomenclature [14]. Episodes are classified as first-detected, recurrent, paroxysmal, persistent, or permanent. When a patient has had two or more episodes of AF, AF is considered recurrent. If it terminates spontaneously, AF is designated as paroxysmal; when it is sustained beyond 7 days, it is called persistent. If cardioversion has failed or had not been attempted, and the patient has been in AF for longer than 1 year, AF is referred to as permanent.

Pathophysiology

The mechanisms responsible for AF are multifactorial [15]. Multiple wavelet reentry [16], anisotropic reentry with high-frequency focal sources and centrifugal fibrillatory conduction [17], and perturbations in the autonomic innervation of the atrium [18] have been proposed as potential mechanisms of AF. Pulmonary veins have arrhythmogenic activity and are implicated in the initiation [19] and perpetuation of paroxysmal AF [20]. In patients who have persistent and permanent AF, fibrosis is thought to

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