Modern management of arrhythmias

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Introduction

Arrhythmia is a common condition affecting about 0.8% of the general population[1,2]. The management of arrhythmias has changed tremendously over the past decade. During this time two principal tools of modern arrhythmias management, catheter ablation and device therapy, have evolved to treat many conditions.

Supraventricular Tachycardia

Supraventricular arrhythmias are relatively common, often repetitive, but rarely life threatening. The estimated incidence of paroxysmal supraventricular tachycardia excluding atrial fibrillation is 35 per 10000 person-years[1]. Diagnosis is established by electrogram during attack of arrhythmia, usually in the form of narrow complex regular tachycardia. The mechanisms of SVT include reentry in case of atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular re-entry tachycardia (AVRT); and focal automaticity in atrial tachycardia. Most SVT except in case of atrial tachycardia are free from underlying structural heart diseases. In patient with AVRT there is connection between atrium and ventricle called accessory pathway that can usually conduct from ventricle to atrium, forming the retrograde limb of the reentry circuit. If the accessory pathway can also conduct in antegrade direction (from atrium to ventricle) then surface electrocardiogram will show typical short PR interval and delta waves of Wolff-Parkinson-White syndrome (WPW). Vagal manoeurres or AVN blocking compounds can terminate SVT if the atrioventricular node (AVN) form part of the arrhythmia circuit as in case of AVNRT and AVRT. Intravenous adenosine and verapamil are commonly used AVN blocking compounds that are fast in onset and effective in terminating SVT. However, the use of these compounds should be avoided in patients with wide complex tachycardia even if it is supraventircular in origin. If WPW patients develop AF, AVN blocking compounds may precipitate ventricular fibrillation. The long term management options of SVT include vagal manoeuvres during attack, antiarrhythmic agents (AAA) to prevent onset of SVT and catheter ablation. Beta-blocker, calcium channel blockers and sometimes digoxin and class I and class III AAA are effective AAA for SVT. Catheter ablation is highly effective with low complication and recurrence rate. It is now generally accepted as one of the first line therapy for SVT.

Atrial fibrillation

Atrial fibrillation is even more common than SVT affecting 0.4% of the general population. The prevalence increases with age, up to 3 to 5 percent of the population over 60 years of age are affected[2]. The predisposing conditions apart from age are structural heart diseases especially valvular heart diseases, hypertension, ischaemic heart disease, post operation status, chronic pulmonary disease, thyrotoxicosis and it can be a sign of extra-cardiac physiological disturbance like sepsis. The annual risk of stroke in patients with AF and normal valve function has been reported to be 4.5 percent per year. Anticoagulation reduces the risk by about two thirds. The mortality rate for stroke in patients with AF is approximately twice as high as the rate in patients without this rhythm abnormality.

There are three potential therapeutic goals in treating patients with atrial fibrillation. These include restoration and maintenance of sinus rhythm, rate control during atrial fibrillation, and prevention of thromboembolism. Intuitively, maintaining sinus rhythm (rhythm control) would be preferable over the rate control strategy in patients with AF. The landmark AFFIRM study[3] compares the rhythm control (using mainly AAA) versus rate control only strategy in AF patients with age > 65 or with risk factor for stroke or death shows the contrary. There is no statistical difference in death or composite morbidity; and increase in subsequent hospitalisation rates in the rhythm control group. There is also a trend towards more strokes in the rhythm control arm. The likely explanation of the result is the noncompliance of anticoagulation in the rhythm control arm because of false sense of security and the harmful, pro-arrhythmic effect of the AAA. Despite the result of AFFIRM study, rhythm control still plays an important role in very symptomatic patients with paroxysmal episodes of atrial fibrillation. However the effectiveness of most effective AAA in maintaining AF free is still poor with only less than 50% success over a 3 year period[4]. The long term use of amiodarone is also associated with significant side effects including that of thyroid, liver and pulmonary. The quest for more effective and safer methods in rhythm control results in intense researches in the area. Increasing understanding of the electrophysiology of AF, namely pulmonary vein foci in triggering[5] and multiple wavelet reentry in perpetuation of atrial fibrillation, has allowed left atrium catheter ablation to be the new promise for AF

rhythm control. The intermediate term success rate of AF ablation reaches 70 to 90% in selected centres. There is evidence to suggest that AF ablation may also be effective in patients with chronic atrial fibrillation[6]. Catheter ablation of AF has shown to improve the left ventricular function in patient with heart failure[7]. In patients with permanent AFF and difficult rate control by AVN blocking agents, AVN catheter ablation plus permanent pacemaker improves symptom control and left ventricular function[8].

Stroke risk in AF varies across a range of ages and cardiovascular co-morbidities. A risk stratification guideline is setup to identify patients who need long term anticoagulation therapy. Risk factors are valvular heart disease, age > 75 (definition varies among guidelines), diabetes, congestive heart failure and previous history of stroke. Valvular heart disease itself increases the stroke risk by 16 fold. Long term anticoagulation that maintains INR between 2.0 and 3.0 is recommended for individuals with any one of the risk factors. Aspirin is only recommended in relatively low risk patients and is definitely inferior to warfarin. New agents like direct thrombin inhibitor is a potential replacement of warfarin. Adequate anticoagulation should be given 3 weeks prior to and at least 4 weeks after cardioversion if the AF episode last more than 48 hours. It has been shown that cardioversion is relatively safe if transophageal echocardiogram shows no evidence of thrombus in the atrium even if AF lasts more than 48 hours[9]. The "pill in the pocket" regime of single dose oral propafenone[10] is effective in prompt cardioversion of AF episodes in and out of the hospital setting. Cardioversion can be done electrically and latest defibrillator has option of biphasic impulse that theoretically can increase the success rate of cardioversion.

Atrial flutter

Atrial flutter is caused by macro-reentrant around the tricuspid annulus in the right atrium bounded by a protected isthmus of slow condition between the tricuspid annulus and inferior vena cava (cavotricuspid isthmus). Typical atrial flutter results in a saw-tooth pattern in the inferior leads of the 12 lead ECG and a positive F wave in V1. The atrial rate is often around 300/min with a 2:1 AVN block, therefore the ventricular rate is 150/min. Drawing a line of ablation across the cavotricuspid isthmus effectively blocks the circuit for typical flutter and becomes a very effective cure for atrial flutter. Atrial flutter patients also run a risk of stroke though to a lesser extent than AF and high risk patient should also be put on anticoagulation.

Ventricular arrhythmias

Ventricular fibrillation (VF) and ventricular tachycardia (VT) are most commonly the consequence of coronary heart disease or cardiomyopathies. They differ fundamentally from SVT in that they are a common cause of sudden cardiac death. Prospective randomised study has showed the superiority of implantable cardiovertor defibrillator (ICD) over pharmacological treatment, predominantly amiodarone in primary and secondary prevention[11,12]. Latest guideline has included ICD as recommended treatment in patients with heart failure and left ventricular ejection fraction of less than 30%[13]. Idiopathic VT with left bundle branch block morphology and inferior axis on surface electrogram has an origin from right ventricular outflow tract (RVOT) that is caused by focal automaticity. Another idiopathic VT in the left ventricle is caused by reentry in the Purkinje system. They are usually not associated with underlying structural heart disease and are less likely to cause sudden death. These idiopathic VT are amenable to catheter ablation which result in cure.

Conclusions

Contemporary management of arrhythmias is no longer limited to pharmacological agents. Some arrhythmias can be cured with catheter ablation and sudden cardiac death can now be prevented by ICD. However, there are areas that await further improvement such as finding a cure for AF and better screening and stratification of patients at risk of sudden cardiac death.

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