Management of Arrhythmias in Heart Failure

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Keywords: Cardiology; Heart Failure; Guidelines; Arrhythmias

Citation: Spoletini I and Coats AJS. Management of Arrhythmias in Heart Failure. International Cardiovascular Forum Journal 2017;10:45-49, DOI: 10.17987/icfj.v10i0.446

Introduction

Patients with heart failure (HF) often develop ventricular and supraventricular arrhythmias,[1] due in large part to electrical conduction abnormalities of the heart in this syndrome.[2] Cardiac remodeling and neurohumoral activation typical of HF create a substrate that increases the risk of developing arrhythmias and/or worsening pre-existing arrhythmias. [3] Advances in our understanding of the underlying pathophysiological mechanisms of HF have reinforced the importance of neurohumoral, mechanical and inflammatory processes as progressively more severe pump dysfunction occurs.[4] This combination increases the likelihood of arrhythmias, both atrial and ventricular, such that ventricular arrhythmias are found in up to 80% of patients with severe HF,[5], conferring additional risk of mortality and morbidity [3], in particular via an increased risk of sudden cardiac death.[6–8] Arrhythmias are also responsible for an increased risk of rehospitalisation in one-third of HF patients.[9]

Arrhythmias include tachyarrhythmias (i.e. atrial, ventricular arrhythmias) and bradyarrhythmias (i.e. sinus node dysfunction, conduction disorders). Common HF-related supraventricular tachyarrhythmias include atrial fibrillation, atrial flutter, and paroxysmal supraventricular tachycardia. Ventricular arrhythmias include ventricular fibillation and ventricular tachycardia.

The initial management of arrhythmias in HF requires identification and an accurate diagnosis. In this phase, ambulatory electrocardiographic examination may be required to detect and manage silent atrial fibrillation and monitor patients with suspected arrhythmia-related symptoms. Subsequent risk stratification can facilitate the assessment of the risk of sudden cardiac death or arterial embolism. Bleeding risk scores such as CHA²DS²-VASc and HAS-BLED scores should be administered as recommended in the ESC guidelines for atrial fibrillation (in press). Subsequently, a rate control strategy should be set, with a rhythm control strategy being reserved for patients with persistent arrhythmia-specific symptoms or if haemodynamically compromised by the arrhythmia (see below).

Atrial fibrillation in HF

The most common arrhythmia is atrial fibrillation, which is characterised by uncoordinated atrial activation with resultant impairment of atrial mechanical function.[10] Atrial fibrillation may be associated with other arrhythmias, most commonly atrial tachycardia or atrial flutter. Atrial tachycardia is a supraventricular tachycardia that originates from a “focus” or spot in the left or right atrium with a heart rate of > 100 beats per minute. Atrial flutter is characterised by atrial rates of 240–400 beats/min and some degree of atrio-ventricular node conduction block, with “flutter” waves on the ECG (i.e. a saw-tooth pattern of regular atrial activation). Atrial flutter may develop into atrial fibrillation and atrial fibrillation may reverse to atrial flutter.[10]

HF is a recognised risk factor for atrial fibrillation[16] and the occurrence of atrial fibrillation is associated with increased mortality in HF.[17] A recent meta-analysis has shown that all-cause mortality due to incidental atrial fibrillation is significantly higher in HF patients with reduced ejection fraction (HFrEF) compared to those with preserved ejection fraction (HFpEF).[18] Finally, even in the absence of overt clinical symptoms, atrial fibrillation predicts stroke and worsening of HF symptoms.[19,3]
Nearly 40% of patients with atrial fibrillation go on to develop HF,[13] and the incident HF is associated with worse symptomatology[20] and increased mortality.[17] However, atrial fibrillation in isolation has not been consistently found to be a risk factor for HF independent of other possibly shared risk factors.[21–24] Thus, HF and atrial fibrillation often co-exist, and this may be explained considering common risk factors and intertwining, self-perpetuating processes.[3] In the clinical management of HF it is important, although challenging, to identify whether HF is a trigger or a consequence of atrial fibrillation.

**Management of atrial fibrillation in HF**

Atrial fibrillation may be treated with drugs to slow the heart rate (“rate control”) or to convert the rhythm to normal sinus rhythm (“rhythm control”).
at rest is 60–100 bpm according to the ESC/HFA guidelines[28], should be documented by ECG and the preferred ventricular rate in Figure 10.1. Notably, ACEI, ARB, beta-blockers and MRA for the management of atrial fibrillation in HF are reproduced guidelines on HF[28]. The recent ESC/HFA recommendations of atrial fibrillation in HF have been developed by the 2016 ESC Updated evidence-based recommendations for the management of atrial fibrillation in patients without atrial fibrillation.[26,27]

Due to this worse clinical status, HFpEF patients with atrial fibrillation have worse pulmonary artery pressure, higher biomarker levels and worse complicated phenotype[25] characterised by increased potassium/magnesium, ongoing ischaemia) should be considered to be sought and corrected in patients with ventricular arrhythmias.

Potential aggravating/precipitating factors (e.g. low serum potassium/magnesium, ongoing ischaemia) should be considered to be sought and corrected in patients with ventricular arrhythmias.

Treatment with an ACEI (or ARB), beta-blocker, and MRA in optimal doses reduces risk for SCD and is recommended for patients with HFrEF and ventricular arrhythmias (as for other patients).

Implantation of an ICD or CRT-D device is recommended for selected patients with HFrEF.

Several strategies should be considered to reduce recurrent symptomatic arrhythmias in patients with an ICD (or in those who are not eligible for ICD), including attention to risk factors and optimal pharmacological treatment of HF, amiodarone, catheter ablation and CRT.

Routine use of antiarrhythmic agents is not recommended in patients with HF and asymptomatic ventricular arrhythmias because of safety concerns (worsening HF, proarrhythmia, and death).

Ventricular arrhythmias in HF
Ventricular arrhythmias include several different clinical pictures, spanning from isolated and asymptomatic ventricular ectopy at ECG to fatal ventricular fibrillation. Ventricular arrhythmias are common in chronic HF, particularly in patients with HFrEF. The risk of ventricular arrhythmias is higher in HF patients with an ischaemic aetiology[32] and in those with concurrent co-morbidities.[3] According to the 2016 ESC/HFA guidelines, the management of asymptomatic ventricular arrhythmias starts with prevention of precipitants such as electrolyte abnormalities (low serum potassium and magnesium), interruption of potentially arrhythmogenic agents and optimisation of pharmacological therapy. In particular, since ventricular arrhythmias are associated with an increased risk of sudden death, ACEIs, beta-blockers, MRA and LCZ696 may be used for prevention in patients with HFrEF.[28] Other antiarrhythmic drugs (e.g. amiodarone in the setting severe HF) should be avoided as they may affect adversely the prognosis.[6,33–34] Implantable cardioverter defibrillators should also be used to reduce the risk of sudden death in selected patients (see Chapter 9). Updated evidence-based recommendations for the management of ventricular tachyarrhythmias in HF developed by the 2016 ESC guidelines are listed in Table 1.

Symptomatic bradycardia, pauses and atrio-ventricular block in HF
Patients with HF are often characterised by bradyarrhythmias and pauses, occurring in particular during the night (due to the drop of sympathetic activity and the rise of parasympathetic activity), and may be triggered by sleep apnoea.[35–37] Pauses and bradycardia may lead to a worse prognosis in HF. In particular, intermittent high-degree atrioventricular block[38] and bradycardia[39] are associated with a high risk of cardiac death in HF patients. Pacing devices as recommended for prevention by current ESC/ European Heart Rhythm Association (EHRA) guidelines on cardiac pacing and cardiac resynchronization therapy,[40] ICDs with pacemaker function (see Chapter 9) have been demonstrated to be effective for the prevention of bradycardia, beyond the correction of ventricular arrhythmias. In particular, ESC/EHRA guidelines recommend intervention for pauses over 6 seconds in the absence of myocardial dysfunction. On the other hand, in patients with HFrEF also shorter pauses require intervention.[38] For the treatment of bradycardic pauses >3 seconds or symptomatic bradycardia of rate <50 bpm in sinus rhythm or <60 in AF the need for and dose of bradycardic medication should be reviewed. In HF patients with symptomatic, prolonged or frequent pauses despite adjustment of rate limiting medication, the next steps to be considered include either beta-blocker withdrawal or back-up pacing, although pacing solely to permit initiation or up-titration of beta-blockers is not recommended. In HFrEF patients who do have a compelling

### Table 1

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>Class</th>
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<tr>
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<td>IIA</td>
</tr>
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<td>III</td>
</tr>
</tbody>
</table>

Class of recommendation: I= evidence and/or general agreement that a given treatment or procedure is beneficial, useful, effective; II= conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure (IIa: weight of evidence/opinion is in favor of usefulness/efficacy; IIb: usefulness/efficacy is less well established by evidence/opinion); III: evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; CRT = cardiac resynchronization therapy; CRT-D = defibrillator with cardiac resynchronization therapy; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; ICD = implantable cardioverter defibrillator; MRA = mineralocorticoid receptor antagonist.

Control”). Electrical cardioversion may be used for rhythm control especially when the clinical status is not stable.[10] Ablation may be a strategy for relapse prevention in patients with HF and HFrEF, chronic atrial fibrillation and controlled ventricular rate.[3] In patients with HFrEF and atrial fibrillation there is a more complicated phenotype[25] characterised by increased pulmonary artery pressure, higher biomarker levels and worse systolic function than HFrEF patients without atrial fibrillation.

[19] Due to this worse clinical status, HFrEF patients with atrial fibrillation are at higher risk of mortality and hospitalisation than those without atrial fibrillation.[26,27] Updated evidence-based recommendations for the management of atrial fibrillation in HF have been developed by the 2016 ESC guidelines on HF.[28] The recent ESC/HFA recommendations for the management of atrial fibrillation in HF are reproduced in Figure 10.1. Notably, ACEI, ARB, beta-blockers and MRA may be used to prevent atrial fibrillation in HF, but ibabradine is not indicated as it may increase its incidence.[29] Rate control should be documented by ECG and the preferred ventricular rate at rest is 60–100 bpm according to the ESC/HFA guidelines[28], although more studies are probably needed for accurate targeting of preferred AF rates. Despite the fact that the optimal strategy to control ventricular rate remains elusive, beta-blockers, digoxin and the combination may be used[30] with beta-blockers the preferred first-line agent.[28] A rhythm control strategy (including pharmacological or electrical cardioversion) has not been shown to be superior to a rate control strategy in reducing mortality or morbidity in chronic HF,[31] and is indicated only if atrial fibrillation is life threatening or specifically related to disabling symptoms.[39]
indication for pacing in the presence of high degree AV block, CRT rather than RV pacing is recommended, and in the absence of high degree AV block, pacing modes that avoid inducing or exacerbating ventricular dysynchrony should be considered.

Arrhythmias in acute HF

Acute HF may present with severe rhythm disturbances that should be corrected immediately by pharmacotherapy, electrical cardioversion and/or temporary pacing[28,40,41] especially if (whether atrial or ventricular) is thought to be contributing to the patient’s haemodynamic distress. Rate control should be preferred in acute HF, followed by a rhythm control strategy after the acute phase of HF has resolved.[3] Some patients with acute HF may have persistent ventricular arrhythmias; the latter often perpetuates haemodynamic instability and vice versa. In this case, immediate angiography and electrophysiological testing with radiofrequency ablation may be considered.[41]

Conclusions

The high risk of arrhythmias should always be considered during the clinical management of all HF patients, due their association with worse prognosis and increased mortality. In particular, HF and atrial fibrillation mutually worsen the impact of each other. Treatment of atrial fibrillation in the setting of HF includes a variety of approaches such as drugs, devices, and ablation. Restoration of sinus rhythm is not superior to optimal rate control, and the deleterious effects of antiarrhythmic drugs should be considered. Finally, cardiac function, symptoms, and quality of life may improve with catheter-based ablative therapies in appropriately selected patients with HF.[42]

Declaration of Interest

The author declares no conflicts of interest.

Acknowledgements

The authors have abided by requirements for ethical publishing in biomedical journals [43].

References


