

Heart failure in patients with atrial fibrillation

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DOI:

[10.1136/heartjnl-2021-319145](https://doi.org/10.1136/heartjnl-2021-319145)

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Document Version

Peer reviewed version

Citation for published version (Harvard):

Chua, WWL & Fabritz, L 2021, 'Heart failure in patients with atrial fibrillation: why it matters now more than ever', *Heart*, vol. 107, no. 16, pp. 1278-1279. <https://doi.org/10.1136/heartjnl-2021-319145>

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This article has been accepted for publication in *Heart*, 2021, following peer review, and the Version of Record can be accessed online at [insert full DOI eg. <http://dx.doi.org/10.1136/heartjnl-2021-319145>].

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COMMISSIONED EDITORIAL

BMJ Heart Journal

Heart failure in patients with atrial fibrillation: Why it matters now more than ever.

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Atrial fibrillation (AF) unabashedly retains the title of most commonly sustained cardiac arrhythmia worldwide¹. It is known as a major cause of stroke by increasing its risk 5-fold. As a result of widespread clinical education and the advent of technologies which avail novel and safer anticoagulants, an increasing number of healthcare professionals and patients alike now have awareness of stroke prevention in AF. This is a huge success. However, as notably stated by Weber et al. in this issue of Heart, heart failure events occur more frequently than strokes after a diagnosis of AF. Heart failure has proven difficult to treat in patients with AF, creating some degree of frustration facing the challenges. Likewise, preventable risk factors like obesity and excessive drinking can be uncomfortable truths to confront patients with and tough to work on.

Weber et al.² introduced their study by presenting a convincing and often repeated ground truth: AF and heart failure commonly occur comorbidly with shared pathophysiology. Both primary and secondary causes of AF and heart failure lead to atrial cardiomyopathy. In patients who have been diagnosed with AF, heart failure hospitalisation is a common event, leading to excess risk of all-cause mortality, with varying rates depending on how long patients were followed-up.

One of the more well-known observations came from the ORBIT-AF registry which followed 6545 patients with AF from 173 participating sites. Heart failure subsequently developed in 236 patients (3.6%) over 2 years. These patients are three times more likely to be hospitalised and have a nearly doubled risk for all-cause mortality³. Given the growing demand for care of patients with AF as the worldwide growth of the population aged 65 and over accelerates at a faster rate than all other age groups, it is urgent that we prevent not only stroke but also heart failure.

Although both AF and heart failure share similar genetic causes, there is a large environmental component in disease development. Preventing heart failure in patients with AF requires identification of risk factors to target. Weber et al.² sought to find these predictors in patients admitted with AF in a large Western Australia (WA) patient registry which contained records for all WA residents. They linked morbidity and mortality data from the Hospital Morbidity Data Collection (HMDC) and WA Death Registry to extract data on incident heart failure hospitalisation and deaths within 3 years. The comorbidities considered as predictors included prior AF, coronary heart disease, myocardial infarction, coronary revascularisation procedures, peripheral vascular disease, stroke, valvular heart disease, chronic kidney disease, cancer, chronic obstructive pulmonary disease, thyroid disease, hypertension, diabetes, obesity, and excessive alcohol use.

In this cohort, in line with other observational AF cohorts, there is a higher proportion of males (58%) compared to females, and on average, females were significantly older (77 years old) compared to males (70 years old). Of the 52447 patients observed, 6153 patients (11.7%) were hospitalised for heart failure for the first time within three years of an admission for AF. Hospitalisation rates and mortality rates were both greater for older females compared to males. Patients who were hospitalised with heart failure had a threefold increase in mortality risk within this follow-up period. This observation,

the first for an Australian cohort, sits firmly in line with previous literature, affirming the increased risk of heart failure and its sequelae in patients with AF. Perhaps the most striking finding is that younger AF patients (<55 years old) who developed heart failure had a 6-fold risk of death compared to those without heart failure.

Can we slow the progression of heart failure to prevent deaths in patients with AF? Identifying the first overtly presenting condition may allow stratification of AF patients with primary and secondary causes of heart failure which can enable selection of different therapies targeting the index condition. For heart failure with preserved ejection fraction, this identification can be achieved by observing if patients respond well to diuretics (heart failure dominant) or rate control and cardioversion (AF dominant). Treatment of underlying factors would likely benefit heart failure dominant patients while AF dominant patients would benefit from antiarrhythmic therapies⁴. This approach is one of the promising avenues to personalise therapies in patients with concomitant AF and heart failure for better outcomes.

How to prevent heart failure in patients with AF? As obesity was a strong modifiable predictor for developing heart failure, Weber et al. reiterated the call for lifestyle interventions. Weight management has been shown to be an effective target for improving overall cardiovascular health. Despite the significant benefits, losing and maintaining weight loss is easier said than done. Compounds such as GLP-1 analogues and SGLT2 inhibitors show promise as viable interventions and warrant further mechanistic interrogation. Lifestyle interventions, in tandem with early rhythm control, can reduce complications from AF including heart failure⁵. Mobile apps can gently nudge patients to increase compliance⁶. Taking the broader view, how about the psychological and environmental aspects which promote healthy lifestyles? Public health measures, urban planning to create safe outdoor spaces, incentives for adopting cycling and walking as an alternative to driving, a societal appreciation of balanced health and well-being, all play a role to reverse the obesity epidemic driving many cardiovascular diseases.

Aside from the valuable clinical insights, this manuscript by Weber et al.² is an exemplar for big data analysis. Data linkage provides the opportunity for deeper interrogation to provide a new granularity to answer the same research question, revealing a more integrated view of the people behind the numbers. The infrastructure to support the linkage enabling this research should be emulated and made more widely accessible to all researchers. This effort is not trivial. Researchers who handle data wrestle with ever tightening data protection requirements and lengthy procedures. Though necessary, they are barriers which render data access as a prize for the forbearing. Let us endeavour to simplify data access not just between national healthcare systems and academic institutions but internationally as well to accelerate research.

Inequalities in healthcare have been and remain a challenge to overcome. Specifically, in AF, we know that there are disparities in providing treatment options, for example, catheter ablation is more often offered to males even though evidence demonstrates there is no difference in benefit for females⁷. Furthermore, in a large US patient registry, selection of rhythm control therapies was demonstrated to be driven by race, type of health insurance, and whether or not the patient was seen by an electrophysiologist – factors unrelated to patient risk factors⁸. So, when we start focusing on lifestyle interventions as targets for preventing heart failure, we need to strive to diminish such disparities. As a medical and scientific community, let us root for patient empowerment and conditions that ensure everyone has access to green spaces for exercise, options for healthy foods, and knowledge to make informed choices.

This is our call to action. While we count the increasing number of adequately anticoagulated patients, let's urgently ramp up our effort to prevent and manage heart failure in patients with AF.

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Footnotes

Contributors. WC drafted the editorial. LF reviewed the editorial.

Funding. The authors acknowledge funding from MAESTRIA Grant agreement ID: 965286. In addition, the Institute of Cardiovascular Sciences, University of Birmingham, has received an Accelerator Award by the British Heart Foundation AA/18/2/34218.

Competing interests. LF has received institutional research grants and non-financial support from European Union, British Heart Foundation, Medical Research Council (UK), several biomedical companies and DFG. LF is listed as inventor of two patents held by University of Birmingham (Atrial Fibrillation Therapy WO 2015140571, Markers for Atrial Fibrillation WO 2016012783).

Patient and public involvement. Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Provenance and peer review. Commissioned; internally peer reviewed.

Linked article.

Incidence, predictors and mortality risk of new heart failure in patients hospitalised with atrial fibrillation. Weber C, Hung J, Hickling S, et al. *BMJ Heart* 2021; - Published Online First: 11 Mar 2021. doi: 10.1136/heartjnl-2020-318648