Heartbeat: cardiac resynchronisation therapy pacemaker or defibrillator in patients with heart failure with reduced ejection fraction?

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In patients with heart failure with reduced ejection fraction (HRrEF) who meet criteria for cardiac resynchronisation therapy (CRT), the choice between a CRT pacemaker (CRT-P) alone versus CRT combined with a defibrillator (CRT-D) remains challenging. Maille and colleagues[1] used administrative data from all consecutive patients in France treated with CRT-D implantation between 2010 and 2019 to develop and validate a CRT-D futility model based on 1 year all-cause mortality in these 23 029 patients (mean age 68 years, 21% women). This score included measures of advanced heart failure, predictors of non-response, frailty and other comorbidities (figure 1). A score of 12 or higher was associated with a 1 year mortality of 16.6% suggesting this model can be used to identify patients whom are unlikely to benefit from addition of a defibrillator to CRT alone.

In the accompanying editorial, Straw and colleagues[2] address the question of why a defibrillator might or might not be essential in these patients. On the one hand, death in patients with HFrEF may be due to pump failure with electromechanical dissociation or asystole, rather than a ventricular tachyarrhythmias. In addition, left ventricular remodelling with CRT may reduce the risk of sudden death. On the other hand, previous studies have shown a reduced risk of sudden death with an implanted defibrillator in similar populations. Also, myocardial scar, which serves as an arrhythmia substrate, is unlikely to be affected by medical therapy. Of course, many other factors affect the decision about device therapy, including differences between countries and healthcare systems. They conclude: 'Given patients presenting with symptoms of chronic heart failure have a median age >80 years and a combination of life-limiting comorbidities, should future work fail to demonstrate clear superiority of CRT-D, perhaps the default choice should be CRT-P except where an individual assessment of risk suggests otherwise.'

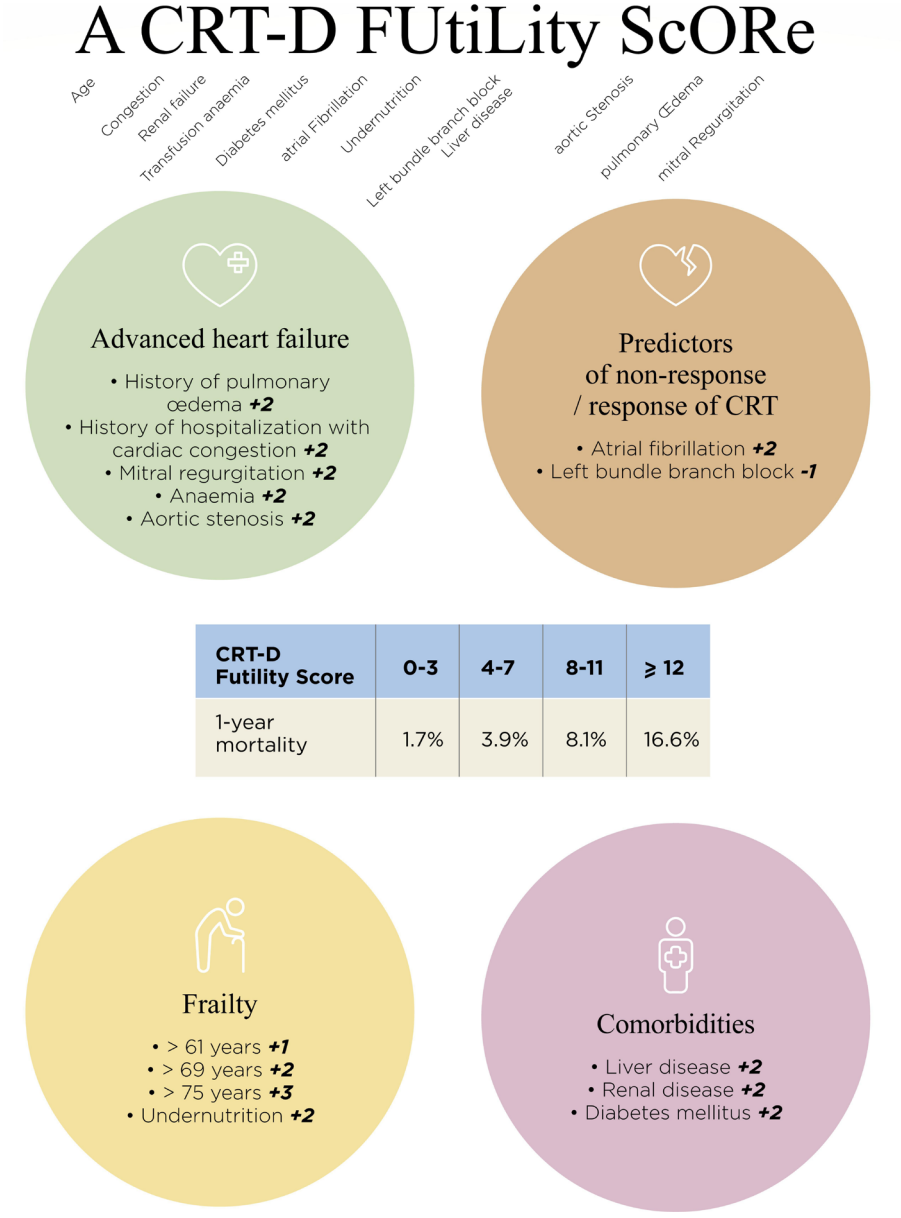

Figure 1 A CRT-D (cardiac resynchronisation therapy defibrillator) futility score. Predictors of futility and risk of all-cause death according to risk level.

Another study in this issue of *Heart* used a large administrative database to address the question of whether the risk of atrioventricular block (AVB) is hereditary. Data were merged from a nationwide cohort of parental-linked individuals with a registry of all pacemaker implantations in Denmark from 1982 to 2019.[3] In 26 880 consecutive individuals, first degree relatives of a patients with a pacemaker had a relative

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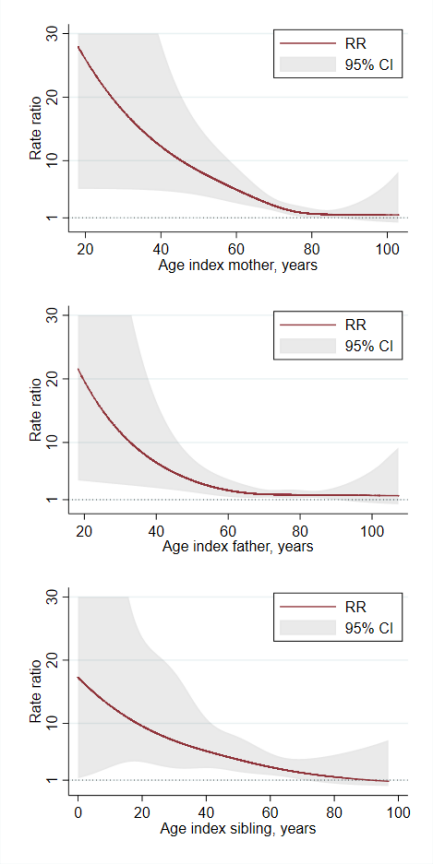

Figure 2 Adjusted rate ratio (RR) of atrioventricular block for first-degree relatives as function of age of the index relative.

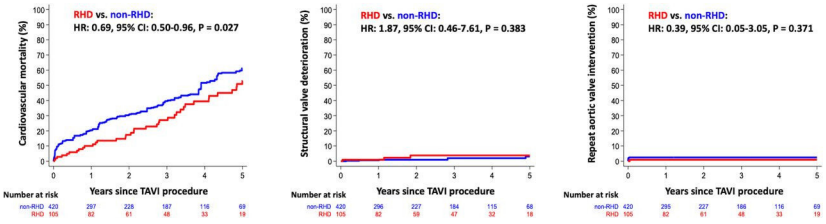

Figure 3 Kaplan-Meier curves for cardiovascular death, structural valve deterioration and unplanned repeat aortic valve intervention up to 5 years in the propensity score-matched cohort. Structural valve deterioration was defined according to the Valve Academic Research Consortium-2 criteria.[17] Unplanned repeat aortic valve intervention was defined as a composite endpoint including valve-in-valve procedure, balloon valvuloplasty, surgical revision or paravalvular leak closure. HR and p values were calculated using Cox proportional hazard models. RHD, rheumatic heart disease; TAVI, transcatheter aortic valve implantation.

risk ratio of 2.1 (95% CI 1.8 to 2.5) for development of AVB compared with the general population. Risk of AVB was inversely proportional to the age of the index case at time of pacemaker implantation with an adjusted risk ratio of 15.8 (4.8–52.3) if a mother [or 10.0 (3.3–30.4) if a father] had pacemaker implantation before age 50 years (figure 2) This finding suggests the possibility of pathogenic genetic variants accounting for AVB in some families.

In an editorial, Roseboom and Maass[4] agree that genetic testing may be appropriate in younger patients with AVB because some cases may be related to known pathogenic variants for channelopathies or cardiomyopathies which have implications both for patient management and cascade screening of family members. However, evaluation for acquired causes of AVB also is important, such as ischaemic heart disease, sarcoidosis, giant cell myocarditis, Lyme myocarditis, Chagas disease or a metabolic disorder. They conclude: 'In young patients with AVB, it is critical to identify underlying heart disease to determine who is at risk for sudden cardiac death and might benefit from implanted cardiac defibrillator therapy, and to define proper treatment in reversible AVB to prevent unnecessary device implantations and unnecessary right ventricular pacing with the risk of dyssynchronous heart failure.'

Transcatheter aortic valve implantation (TAVI) is an established therapy for symptomatic older adults with severe calcific stenosis of a trileaflet aortic valve. Although rheumatic heart disease (RHD) is the most common cause of valve disease worldwide, few of these patients were included in randomised trials of TAVI for severe aortic stenosis (AS). In a prospective Swiss registry of 2329 patients undergoing TAVI over a 12 year period, of whom 4.5% had rheumatic AS, technical success was similar regardless of aetiology despite the theoretical concern that commissural fusion might impair optimal valve deployment in patients with rheumatic AS.[5] In addition, rheumatic AS patients, compared with matched patients with calcific AS, had a lower 30-day (1.9% vs 8.9%) and 1 year cardiovascular mortality (10.0% vs 20.3%) suggesting that TAVI may be appropriate for treatment of severe symptomatic AS in older adults even when due to rheumatic valve disease (figure 3).

The anatomic differences between calcific and rheumatic AS are elegantly illustrated by Saji and Nanasato[6] using CT imaging (figure 4). As they point out, the relative amount of valve calcification may be relevant in ensuring the TAVI valve is anchored securely in the annulus. If future studies confirm that TAVI is safe and effective in patients with severe rheumatic AS, as reported by Okuno and colleagues,[5] more widespread availability of TAVI may be instrumental in reducing global disparities in treatment of severe AS.

The *Education in Heart* article[7] in this issue 'provides information on the importance of diet for cardiovascular disease prevention. It gives insight to elements of the diet that are harmful or

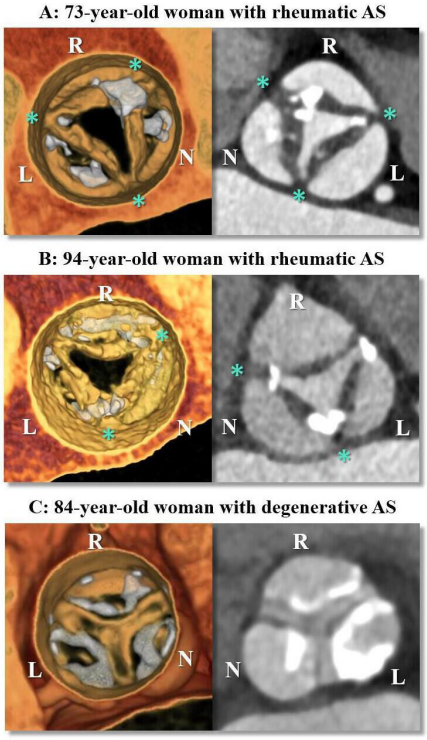

Figure 4 CT images in patients with rheumatic and degenerative AS (A) A central triangular-shaped systolic orifice, thickened leaflets, commissural fusion with calcification in rheumatic AS in a 73-year-old woman. (B) A central triangular-shaped systolic orifice, thickened leaflets, commissural fusion with calcification and slightly more diffuse basal calcification in degenerative calcified rheumatic AS in a 94-year-old woman. (C) A stellate-shaped orifice (no commissural fusion) with severe basal calcification shown in degenerative AS in an 84-year-old woman. commissural fusion. AS, aortic stenosis.

protective and stresses the importance of the totality of the diet. In addition, dietary choices are discussed in relation to environmental sustainability of food production.'

The *Cardiology in Focus* article in this issue[8] discusses how social media can be used to improve medical education including interactive learning, higher retention rate, ability to use learning materials anytime and anyplace, collaboration in medical education rather than each institution working in isolation and ensuring global equity in education.

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