

FI60_64 respectively, by 10% to 12% for FI2-1, FI3-1, FI4-1 and FI4-2, and by 4% and 15% for FIsum and FImean respectively (all $p < 0.05$). Single deficit accumulation had a significant impact on LVmassi and MCFi across all the life-course FIs and overall FIs (all $p < 0.05$). As frailty intensified at each of the 4-time intervals, LVmassi increased by 0.91–1.42%, and MCFi decreased by 0.6–1.02%. As the whole-of-life burden of frailty increased (FIsum and FImean) LVmassi increased by 0.36 and 1.82% and MCFi decreased by 0.33 and 1.04%. One extra deficit accumulated translated into higher multiplicative odds of increasing LV filling pressure by 4.3 for FI60-64, 2.1 for FI4-1, 75.4 for FI4-2 and 78.5 for FI4-3 (all $p < 0.02$).

Conclusion Frailty during childhood, young adulthood, middle age and older age associates with having a weaker (\square EF/MCFi), thicker (\square LVmassi) and stiffer (\square E/e') heart in later-life. The accumulation of new deficits from one age interval to the next also associates with poorer cardiac function in later-life. It appears that multimorbidity and health deficits accumulated over the life-course strain the myocardium resulting in pathological myocardial hypertrophy potentially paving the way to later-life systolic or diastolic dysfunction in susceptible individuals

Conflict of Interest None.

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EFFECT OF FRAILTY ON TREATMENT, CAUSE OF DEATH AND HOSPITALISATION IN PATIENTS WITH CHRONIC HEART FAILURE

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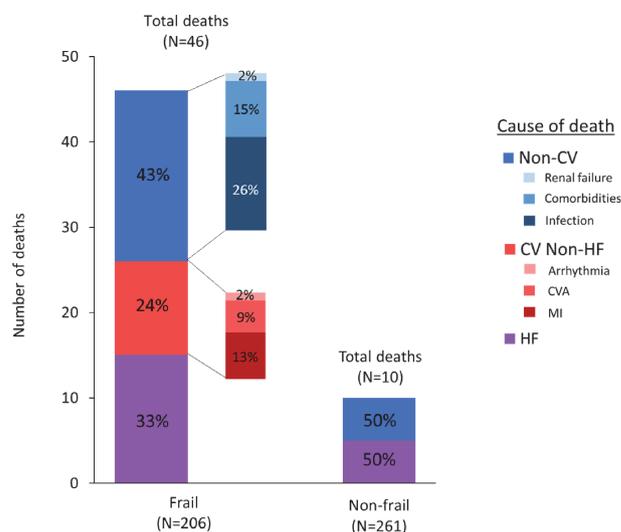
Background Frailty is common in patients with chronic heart failure (CHF) and is associated with poor outcomes. We investigated the relation between frailty and treatments, hospitalisation and death in patients with CHF.

Methods Frailty was assessed using the clinical frailty scale (CFS) in 467 consecutive patients with CHF (67% male, median (IQR) age 76 (69-82) years, median (IQR) NT-proBNP 1156 (469-2463) ng/L) attending a routine follow-up visit. Those with CFS > 4 were classified as frail. We studied the primary cause of death and hospitalisations, ascertained from electronic records, autopsy reports and death certificates, within 1 year of enrolment.

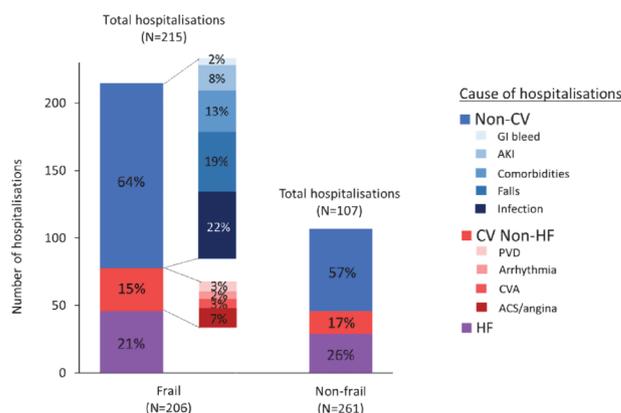
Results 206 patients (44%) were frail. Frail patients with heart failure with reduced ejection fraction (HFrEF) were less likely to receive optimal treatment, with many not receiving an angiotensin-converting enzyme inhibitor or angiotensin receptor blocker (frail: 25% vs non-frail: 4%), a beta-blocker (16% vs 8%) or a mineralocorticoid receptor antagonist (50% vs 41%).

After 1 year, there were 56 deaths and 322 hospitalisations, 46 (82%) and 215 (67%) of which, respectively, occurred in frail patients. 43% of deaths in frail patients were due to non-cardiovascular causes (non-frail: 50%), commonly infections (60%). 58% of cardiovascular deaths in frail patients were due to HF progression (non-frail: 100%). (Figure 1)

64% of hospitalisations in frail patients were due to non-cardiovascular causes (non-frail: 57%), commonly infections (34%) and falls (30%). 59% of cardiovascular hospitalisations



Abstract 83 Figure 1 Cause of death at 1 year in frail vs non-frail patients



Abstract 83 Figure 2 Cause of hospitalisations at 1 year in frail vs non-frail patients

in frail patients were due to decompensated HF (non-frail: 63%). (Figure 2)

Conclusion Frailty in patients with CHF is associated with sub-optimal medical treatment for HFrEF and a high rate of non-cardiovascular events, suggesting that interventions not directed to treating CHF might be appropriate.

Conflict of Interest None

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PARTICIPANTS WITH T2DM HAVE IMPROVED CARDIAC FUNCTION WITH FATTY ACID METABOLISM, DESPITE UNCHANGED CARDIAC ENERGETICS

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Background/Introduction The Phosphocreatine-to-Adenosine Triphosphate ratio (PCr/ATP) is an established indicator of cardiac energetic status. Measurement of the Creatine Kinase pseudo-first order rate constant (CKkf) provides a more sensitive measure of cardiac energetics, and allows calculation of ATP delivery rate through the Creatine Kinase shuttle (CK