Dyssynchrony predicts better survival in low EF groups were non-prognostic (p > 0.05). Taking a different approach to define poor ventricular function—using low S-wave velocity—EF had prognostic significance (p < 0.05) and dyssynchrony markers were non-prognostic (p > 0.05).

Conclusion Dyssynchrony predicts better survival in low EF groups because dyssynchrony acturally lowers EF without damaging survival. The effect is independent of CRT. Replacement of EF with dysynchrony-neutral measures of LV function, for example, peak S-wave velocity would avoid the appearance that dysynchrony is favourable.

Background Single-centre studies have shown that a low serum albumin at baseline forecasts enhanced mortality in chronic heart failure (CHF) possibly because it reflects aberrations (eg, inflammation, impaired nutrition, plasma volume expansion) that can exacerbate disease. We hypothesised that attenuations in serum albumin over time would be prognostically more ominous than baseline values, and would be so even in a multicentre setting.

Methods We analysed the survival implications of baseline albumin and Δalbumin in a derivation cohort of 246 CHF outpatients (mean [±SD] age 65±12 years, LVEF 29±8%, 48% NYHA class >2) from University College London Hospital and then in a validation cohort of 148 CHF outpatients (age 69±12 years, LVEF 28±10%, 41% NYHA class >2) from Imperial Healthcare (St Marys Hospital and Hammersmith Hospital, London).

Results In the derivation cohort, 51 (21%) patients died over 13 months. Baseline albumin independently predicted mortality (HR 0.89, 95% CI 0.84 to 0.94, χ²:18, p < 0.0001). However, Δalbumin (unadjusted HR 0.89, 95% CI 0.84 to 0.92, χ²:53, p < 0.0001) was even more predictive (Difference in ROC AUC for baseline vs Δalbumin 0.16, p < 0.0001) and did so independently of all covariates including baseline albumin. A reduction in albumin > 6 g/l optimally predicted death (ROC AUC 0.82, p < 0.0001) and conferred a sixfold escalated risk of mortality (HR 6.42, 95% CI 3.67 to 11.22, p < 0.0001). In incremental prognostic analyses, the addition of Δalbumin to the strongest four variable model (baseline albumin, NYHA class, Δurea, Δhaemoglobin) dramatically augmented the χ² value (45 vs 84, p < 0.0001). In the validation cohort, 43 (30%) patients died. Δalbumin (unadjusted HR 0.89, 95% CI 0.86 to 0.92, χ²: 44, p < 0.0001) was again prognostically superior to baseline albumin with a fall >6 g/l predicting an ~sixfold increased risk (HR 5.64, 95% CI 3.08 to 10.31, χ²: 35, p < 0.0001). Addition of Δalbumin to the strongest three variable model (baseline red cell distribution width, Ared cell distribution width, Δurea) also augmented the χ² value (51 vs 65, p < 0.001).

Conclusions A fall in serum albumin over time consistently predicts an amplified risk of death in systolic CHF and enables simple and cheap risk stratification.

Background Controversy exists regarding the importance of glycaemic control in patients with type 2 diabetes mellitus (T2DM) and chronic heart failure (CHF) based on conflicting reports that had used a single baseline HbA1C.

Abstract 009 Table 1

<table>
<thead>
<tr>
<th></th>
<th>HFNEF with LVH</th>
<th>HFNEF without LVH</th>
<th>Controls</th>
<th>p-value (ANOVA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>SD systolic motions (ms) at Rest</td>
<td>53.3±32.7</td>
<td>45.5±33.2</td>
<td>44.8±25.7</td>
<td>0.456</td>
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<tr>
<td>SD systolic motions (ms) on exercise</td>
<td>48.0±28.3*</td>
<td>28.7±18.7</td>
<td>25.7±15.7</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

*p < 0.05 compared to controls.  †p < 0.05 compared to HFNEF patients without LVH.

Abstract 009 Figure 1