Response to: ‘Disease activity and left ventricular systolic function in rheumatoid arthritis’ by Giollo et al

We thank Giollo et al1 for their interest and comments to our paper. In this paper, we demonstrated that rheumatoid arthritis (RA) disease activity was associated with lower left ventricular (LV) myocardial systolic function when assessed by stress-corrected midwall shortening (scMWS) or global longitudinal strain (GLS).2 Further, we found that this association was independent of the presence of a normal ejection fraction and traditional cardiovascular risk factors like hypertension and smoking. In a letter to the editor, Giollo et al1 present data from a series of 235 patients with RA.2 In contrast to our findings, Giollo et al did not find an association between RA disease activity and LV systolic myocardial function when assessed by scMWS or tissue Doppler mitral annular systolic velocity (S’), despite a much higher median RA disease activity in their population.3 The higher RA disease activity probably also explains the much higher prevalence of abnormal LV geometry in the study by Giollo et al,1 since prevalences of hypertension, diabetes and obesity, all factors associated with LV hypertrophy, did not differ between the study populations.2,3 Of note, the higher RA disease activity was found despite a higher use of immunomodulation therapy in their study. Giollo et al suggest that the association between RA disease activity and LV systolic function should have been evaluated in relation to LV geometry, and the results are shown in table 1 below. As demonstrated, LV systolic function was lower among patients with active RA (Simplified Disease Activity Index ≥3.3) compared with patients with RA in remission when LV geometry was normal. Also when LV geometry was abnormal, patients with active RA had numerically lower LV systolic function although this was not statistically significant, due to the low number of patients with RA in remission who actually had abnormal LV geometry in our study (table 1).

The link between RA disease activity and cardiovascular disease has been well established over the recent years.4 Treatment with immunomodulation therapy has been shown to improve GLS in patients with RA,5 and disease activity has been correlated to myocardial fibrosis and impaired strain on cardiac MRI.6 Our finding of lower LV systolic function in patients with active RA is in line with these previous publications. It is likely that the higher RA disease activity in the Giollo study contributed to the differential findings between the studies. Furthermore, GLS may capture longitudinal function better than the simpler S’ used by Giollo et al. We, therefore, do not agree that the reported association between RA disease activity and lower LV systolic function solely reflects changes in LV geometry.

<table>
<thead>
<tr>
<th>Table 1 Systolic left ventricular function in relation to left ventricular geometry and disease activity in patients with RA</th>
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<tbody>
<tr>
<td>Normal geometry</td>
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<tr>
<td>Active RA, SDAI &gt;3.3</td>
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<tr>
<td>(n=55)</td>
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<tr>
<td>GLS (%)</td>
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<td>scMWS (%)</td>
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</table>

GLS, global longitudinal strain; RA, rheumatoid arthritis, scMWS, stress-corrected midwall shortening; SDAI, Simplified Disease Activity Index.

Received 22 September 2016
Accepted 26 September 2016
Published Online First 14 October 2016

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Competing interests None.

Provenance and peer review Commissioned; internally peer reviewed.

http://dx.doi.org/10.1136/annrheumdis-2016-210482